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Evaluation of Redox Status, Clinicopathological Parameters and Cytogenetic Changes in Dairy Cattle Exposed to AflatoxinB1 and Zearalenone Contaminated ration

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SUMMARY

The objective of this study was to evaluate the effect of aflatoxin B1 and zearalenone on oxidant / antioxidant balance, liver and kidney functions in addition to chromosomal abnormalities in dairy cows. A total number of 33 dairy muliparous Holstein Friesian lactating cows were under investigation, their body weight ranged from 650 -750 kg. Cows were classified into two groups, control group (n=11) received ration where aflatoxin B1 and zearalenone concentrations not exceed the permissible limits. While the other group (n=22) fed ration contaminated with aflatoxin B1 and zearalenone mycotoxins. Three blood samples were taken from each cow .The first used for serum separation and the obtained sera were used for determination of copper and zinc levels, liver enzymes (ALT- AST), urea and creatinine. The second sample used for plasma separation and estimation of enzymatic antioxidant activities (glutathione peroxidase, superoxide dismutase and catalase) and malondialdehyde. While the third blood sample used for cytogenetic analysis. The obtained results of the second group showed that there were significant imbalance in most of oxidant / antioxidant parameters (significant decrease in glutathione peroxidase, superoxide dismutase and catalase on contrary a significant increase in malondialdehyde). Copper and zinc showed statistically non significant decrease. Additionally, liver enzymes and urea showed significant increase while statistically non significant

decrease recorded in the level of serum creatinine. Concerning the cytogenetic changes, the same group revealed significant increase in numerical aberrations (peridiploidy) and structural aberrations (deletions, fragments) in addition to gonosomal anomalies (59-XO). Generally, the total percent of aberrated cells was significantly increased. In conclusion, aflatoxin B1 and zearalenone contaminated ration cause a harmful effects on redox status, liver function, in addition to their adverse effect on chromosomal abnormalities which reflect negatively on farm production and economy.

INTRODUCTION

In most developing countries, livestock production is an important part of the national economy (Lanyasunya et al., 2005). Mycotoxins are unavoidable contaminants all over the world. Contamination of food and feed with mycotoxins is a global problem causing great economic loss in both live stock industry and agriculture (Bhat and Vasanthi, 2003). Mycotoxins are highly toxic secondary metabolites produced by fungi. The major fungal producing mycotoxins include Asperagillus, Fusarium and Pencillium. The most common mycotoxins are Aflatoxins, Zearalenone, Ochratoxin A, Fumonisin, Deoxynivalenon and T-2 toxin (Lawlor and Lynch, 2005). Among the naturally occurring aflatoxins (B1, B2, G1, G2) aflatoxin B1 is toxic for man and animals, as following ingestion it metabolized in liver resulting in various metabolitis, aflatoxin M1 is the most important one (4-hydroxy metabolite of B1) which excreted in milk (EFSA, 2004). Zearalenone has estrogenic effect on mammals; this negative effect on reproductive systems makes it a concern in animal husbandary (Stopper et al., 2005). Mycotoxins differ in their structure which explains the great variation of symptoms. They produce a wide range of adverse and toxic effects, in this respect mycotoxins are considered to be among most important feed born stress factors (Surai, 2006) which impose an oxidative stress and have stimulating effect on free radical formation (Iheshiulor et al.,2011) .

So, the current work is an attempt made to investigate the real impact of aflatoxin B1 and zearalenone contaminated ration on dairy cows to detect its effect on redox status, clinicopathological parameters and cytogenetic changes.

MATERIAL AND METHODS

Animals and ration:

A total number of thirty three (33) lactating multiparious Holstein Friesian

cows were used in the current study. Their body weight ranged from 650 to 750 kg. Feed and water were provided ad-libitum. Cows were milked three times per day. Animals fed Total Mixed Rations (TMR) consists of corn silage, Egyptian clover hay, ground corn, soybean meal, rice brain, minerals and vitamin premxis. Nutrient concentrations met nutritional requirements for lactation according to the NRC (2001). Animals were randomly classified into two groups : control group (11 cows) received TMR mixture where the percentage of aflatoxin B1 and zearalerone not exceed the permissible limits which is 5 ppb and 200 ppb for aflatoxins and zearalenone respectively (FAO,2004) .The other group (22 cows) received the same ration but with using corn silage and clover containing high levels of both aflatoxins B1 and zearalenone .The total concentration of mycotoxins in TMR fed to this group was (13.10 ppb) and (281 ppb) for aflatoxinsB1and zearalenone respectively.

Sampling:

Three blood samples were collected from each animal by jugular vein puncture. The first on plain centrifuge tubes for serum separation and evaluation of serum copper and zinc levels, liver enzymes (ALT- AST), urea and creatinine . The second on heparinized vacum tubes for plasma separation and evaluation the activity enzymatic antioxidant (glutathione peroxidase, super oxide dismutase and catalase)and malondiadehyed. The third sample was taken on sterile heparinized vaccutainers for cytogenetic analysis. Representative feed samples were taken for analysis of feed born mycotoxins by HPLC.

Redox , biochemical and mycotoxin analysis:

Each sample was analyzed using commercial diagnostic kits (bio-diagnostic) for the following parameters: glutathione peroxidase according to Paglia and Valentine (1967), catalese according to Aebi (1984), super-oxide dismutase according to Nishikimi et al. (1972), lipid peroxidase (Malondialdehyde) concentration according to Ohkawa et al. (1979). Copper and zinc levels were estimated by using atomic absorption sepectrophotomemter (Mod, 3300, Parkin Elmar USA). AST (Aspartate Aminotransferase) and ALT (Alanine Aminotransferase) according to Reitman and Frankel (1957).Blood urea nitrogen was determined according to Henery et al. (1974). Serum creatinine was determined according to Faulkner and King (1976). Mycotoxins were analyzed by High Performance Liquid Chromatography (HPLC-CBC-7210H

Austerila) with fluorescence detection (FLD). We offer a varity of clean up columns according to Scott (1997).

Cytogenetic analysis:

It was carried on 5 cows from group one and 12 cows from group two.1 ml of blood samples were culture in flattened side tubes containing 5 ml RPMI media 1640, 1ml fetal calf serum and o.1 ml phytohaemagglutinine (PHA.).The samples incubated in CO_2 incubator at 37° C. and 5 % Co_2 for 72 hrs .One and half hour before the end of incubation period, the cells were treated with 0.01 ml colchicines then reincubated till the end of incubation period. The cells were exposed to hypotonic solution of 0.56 % KCL for 30 min / 37° C. Then the cells fixed by carnoy`s fixative (1 part glacial acetic acid + 3 parts absolute methanol) for three to four times. The cells suspension was splashed on wet chilled slides then flamed to dry (Macgregor, 1993). The slides were stained by 10 % Giemsa stain ( diluted with sorenson`s buffer) and covered by DPX mounting media (Ram and Arvind ,1995).The slides were scanned by inspsection of 50 good metaphase for each sample according to Nicholas (1996).

Statistical analysis:

The biochemical parameters were subjected to T-test analysis while the percentages of chromosomal aberrations among groups were compared using chi square test according to Senedecor and Cochran (1982).

RESULTS

Redox status Prameters

Table (1) revealed that cows received contaminated ration showed a significant ($P<0.05$) decrease in serum activities of glutathione peroxidase, superoxide dismutase and catalase meanwhile, the decrease recorded by copper and zinc was statistically non significant. On the other hand, malondiadehyde showed a significant ($P<0.05$) increase in the same group compared with control one.

Clinicopthological Pameters

Table (2) showed that there were a significant ($P <0.05$) increase in urea, ALT and AST, while the increase in serum creatinine was non significant in group of cow that feed contaminated ration compared with control group.

Cytogenetic Parameters

Table (3) recorded the results of numerical and structural aberrations in the two groups under investigation. Photo (1) showed normal metaphase spread of cows 58 acrocentric autosomes and two submetacentric gonosomes (60, xx). Our results revealed many significant changes in the group of cattle received contaminated ration recorded by

significant increase in structural aberrations represented by deletions (P<0.04) and fragments (P<0.02) (photo 2). Concerning the gonosome aberrations, the current study revealed a clear significant (P<0.02) increase in gonosome anomalies (59, xo) (photo 3). The second group showed also a significant increase in numerical chromosomal aberrations (peridiploidy-photo 4). Globally, the later group recorded a significant (P<0.02) increase in the total percent of aberrated cells.

Table (1): Redox status in control cows compared with those received contaminated ration.

Parameters	Control cows	Cows received Contaminated ration
Glutathione Peroxidase mU/ml	88.74 ± 5.83	70.70 ± 5.70*
Superoxide Dismutase U/ml	845.6 ±31.20	753.40 ± 28.10*
Catalase U/ml	8.10 ±0.40	6.80 ±0.20*
Malondialdehyde m mol/l	3.41 ± 0.15	5.36 ± 0.38*
Copper µg /dl	1.27 ± 0.10	0.90 ± 0.06
Zinc µg /dl	2.30± 0.08	1.90± 0.05

* Means significant from control at (P< 0.05).

Table (2): Clinicopathological parameters in control cows compared with those received contaminated ration.

Parameters	Control cows	Cows received Contaminated ration
ALT IU/L	5.10 ± 1.04	11.47 ± 2.80 *
AST IU/L	18.45 ± 2.02	33.81 ± 3.09 *
Urea (mg/dl)	28.27 ± 2.50	36.1 ± 2.80 *
Creatinine (mg/dl)	1.45 ± 0.54	2.52 ± 0.60

* Means significant from control at (P< 0.05).

Table (3): Percentage of chromosomal aberrations in control cows compared with those received contaminated ration.

Chromosomal aberrations %	Control cows	Cows received Contaminated ration
Peridiploidy	0.8	5.2*
Breaks	0.4	1.3
Gaps	0.4	0.6
Deletions	1.2	3.2 *
Fragments	0.4	2.5 *
Gonosome aberration (59,xo)	0.0	1.8 *
Total %	3.2	14.6 *

*Means significant from control.

Discussion

Mycotoxins contamination of crops is an inevitable part of animal production system (Sultan and Hanif, 2009). Ruminant's diet may have an increased probability of multiple mycotoxins contamination (Azam et al., 2009). Recent studies showed that in many cases membrane active properties of various mycotoxins determine their toxicity ,indeed, incorporation of mycotoxins into membrane structures causes various detrimental changes (Voss et al ., 2006).

In the present study, our results in relation to redox balances revealed a significant decrease in enzymatic antioxidant and increase in lipid peroxidation parameter (Malondialdehyde) in the group of cattle received contaminated ration .These results were in agreement with Umarani et al. (2008), Cavin et al. (2007), Peter et al. (2007), Rumora et al. (2007) and Surai (2006). This alteration in redox balance may be due to tissue specific activation and expression of redox sensitive signaling

molecules as recorded by Rumora et al. (2007). Peter et al. (2007) pointed out that pro-oxidant effect of mycotoxins may stimulate lipid peroxidation by enhancing free radical production and decreasing concentration of enzymatic antioxidant. More explanation was quoted by Surai (2006) who explained that mycotoxins induces membrane structural alteration ,induction of oxidative stress and lipid peroxidation .The same author illustrated that there was a delicate balance between antioxidants and pro – oxidants in the body in general and specifically in the cells responsible for regulation of various metabolic pathways , consequently nutritional mycotoxins (aflatoxins and zearalenone) have a negative impact on this antioxidant / pro-oxidant balance and have stimulating effect on lipid peroxidation and hydroxyl radical formation . Furthermore, Umarani et al. (2008) elucidated that the harmful effect of aflatoxin B1 are consequence of its being metabolized to AFB1 - 8, 9 epoxide that serve as an alkylating agent and mutagen which is efficiently conjugated with reduced glutathione. Meki et al. (2001) in their study on hepatic malondialdehyde (bio-marker of oxidative stress and cellular damage) attributed the increase in the level of malondiadehyde(MAD) to the fact that AFB1 - 8, 9 epoxide which in turn react with macromolecules such as lipid and DNA leading to lipid peroxidation and cellular injury.

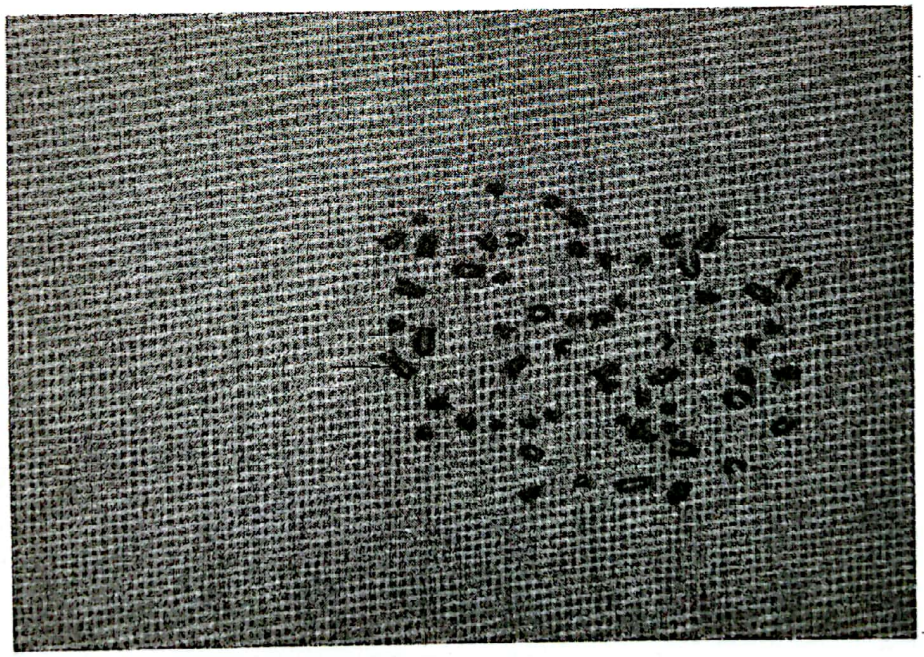
Regarding to the clinicopathological changes, the present study recorded a disturbance in liver enzymes and kidney function. As mycotoxins can cause damage to target organs mainly liver and kidneys (Aydin et al., 2008). As the reactive aflatoxin -8,9 epoxide induce hepatocellular damage in addition to kidney lesions so, blood biochemical parameters were altered reflecting the degree of liver damage (EFSA,2004). Gremmrls (2008) explained that conversion of zearalerone to alpha-zearalenone in addition to the increase rate of passage of food through the rumen may possible overwhelm the ability of rumen to completely denaturate the toxins. So, the author added that some of the rumen metabolites of mycotoxins are more toxic than the parent mycotoxins. Conflicting results have been reported by Umarani et al. (2008) regarding hypothesis that aflatoxin found in rumen may be ascribed to hepatic biotransformation and subsequent recycling to the rumen via rumino-hepatic pathway , because biodegradation capacity of rumen microbs towards aflatoxin is poor and the toxicity of aflatoxicol is close to the toxicity of the native toxins . Additionally, Towner et al.

(2002) stated that aflatoxins are oxidized in the liver into very reactive molecules capable of binding criteul molecules such as nucleic acid or functional proteins in cells thus originating the initial steps of cancer formation. Korosteleva et al. (2007) gave an explanation to the elevation of the blood urea as microbial protein synthesis in the rumen is inhibited by mycotoxins , resulting in presence of more free ammonia remains in rumen , absorbed into the blood and is metabolized to urea , resulting in elevated blood urea concentrations . Our results is also in corroborated with Doorten et al. (2004) in their study on ochratoxins as they stated that ochratoxins induced cell damage of liver and kidney tissue due to increase levels of superoxide radical , leading to an increase in oxidative stress enhancing early cell death , probably by apoptotic mechanisms . Meanwhile, our results recorded insignificant decreased levels of copper and zinc which may be due to that they are components of antioxidant system that affected by mycotoxins feed born stress factors.

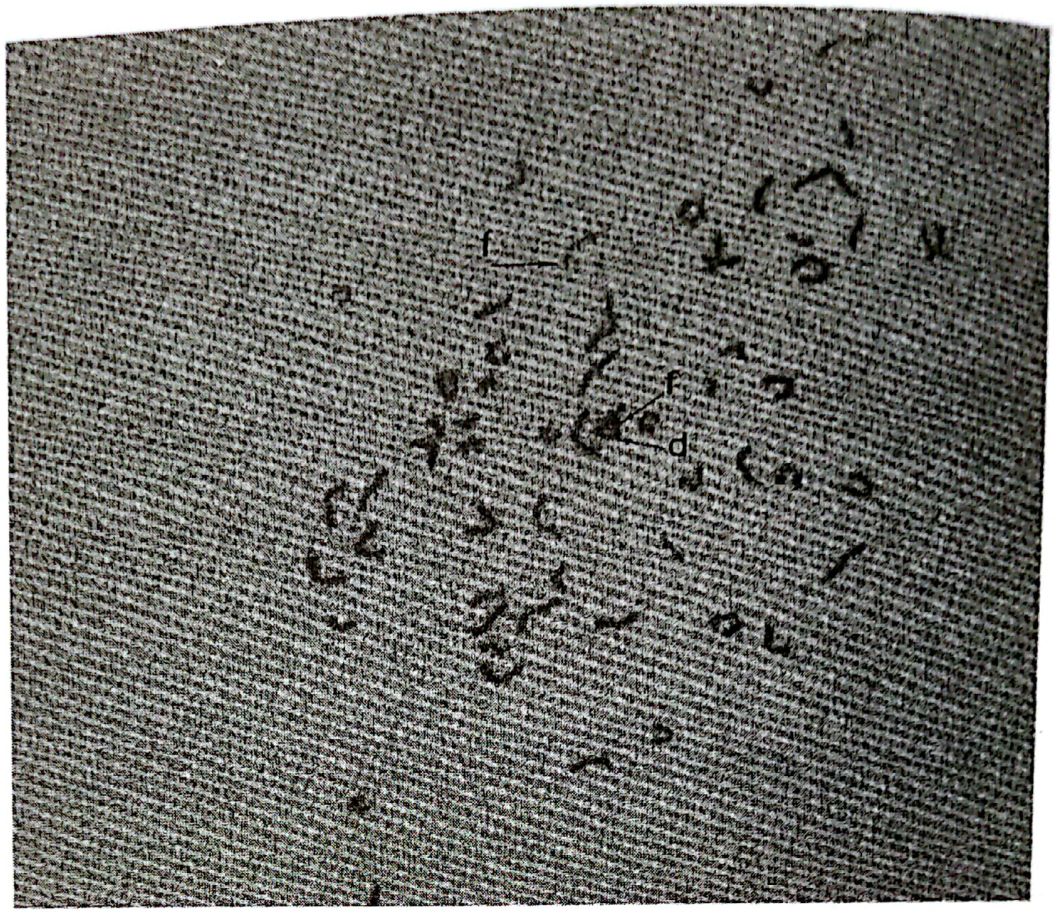
With respect to the chromosomal analysis, the commonly used test for genetic abnormality, the current work revealed numerical and structural aberrations in addition to gonosomal anomolies (59, xo) .this results are in contrast with those of Stopper et al. (2005) who stated that the genotxicity is reported concerned with zearalenone which induced chromosomal anomolies in some lymphocytes and oocytes. The authors attributed these changes to that zearalenone metabolite which is alpha-zearalenone, has about three folds more estrogenic potency than zearalenone. Carrano and Natarajan (1988) reported that chromosome aberrations are thought to arise from misrepair of lesions in the GO stage of circulating lymphocytes as well as from precursor cell in bone marrow and thymus. Also Yi jang et al. (2005) stated that aflatoxins induce differential subset distribution and functional alteration of specific lymphocyte subset, major changes in the constitutions of lymphocytes and decrease in activated T- cells and B- cells. Cosimi et al. (2009) in a parallel study on ochratoxin A (OTA) elucidated that some types of mycotoxins might interfere with chromosome distribution during cell division which may explain the numerical anomalies that occur in the current study on aflatoxins and zearalenone. Additionally, the generated free radicals and lipid peroxidation has been linked to genotoxicity expressed by DNA breakage and damage which give another explanation to the significant occurrence of structural chromosomal aberrations. Eaton

and Gallagher (2004) added that aflatoxin B1 (AFB 1) is metabolized by liver through cytochrome P-450 enzyme system to the major carcinogenic metabolite AFB1- 8, 9 epoxide (AFBO) or to less mutagenic forms such as AFM1, Q1, or P1 and there are several pathways that AFBO can take, one resulting in cancer, another in toxicity the exo-form of AFBO readily bind to cellular macromolecules including genetic material leading to gene mutation and cancer. Groopman and Kensler (2005) showed that AFBO induces conversion from G (guanine) to T (thyamine) making it a mutational hotspot. Recently Emna et al. (2010) elucidated that AFB1 caused a marked decrease of cell viability and increase damage and fragmentation of DNA.

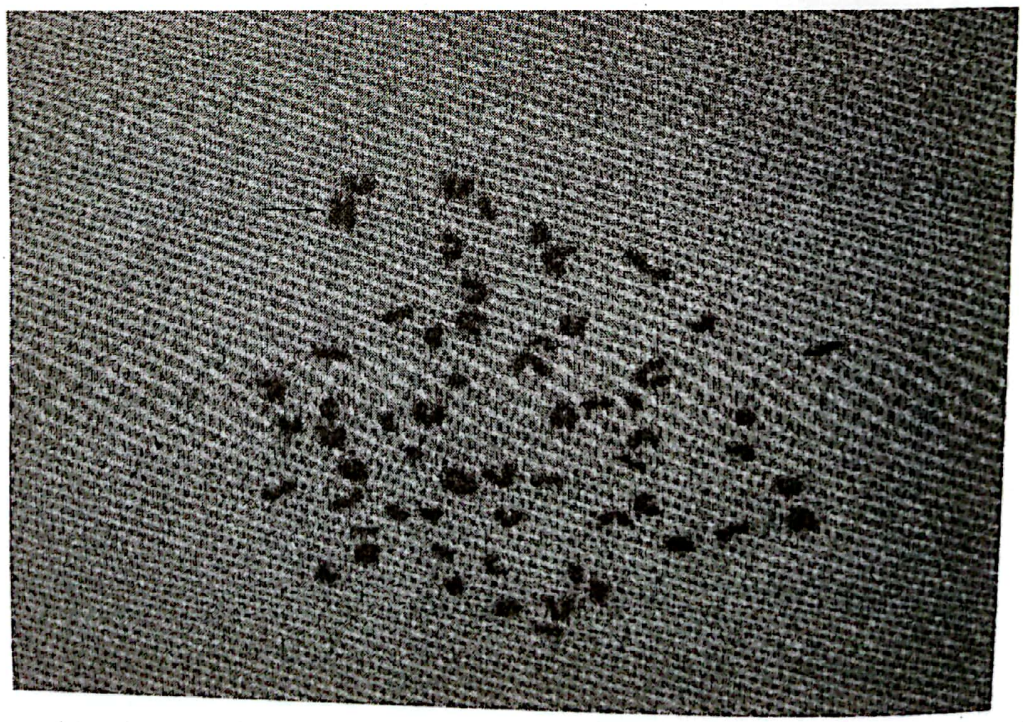
Finally the present investigation concluded that aflatoxin B1and zearalenone contaminated ration cause detrimental changes in oxidant / antioxidant balance, impaired liver function in addition to cytogenetic changes including gonosomal aberrations. So, the ideal means of dealing with mycotoxins is to control and prevent it from its contamination to the ration as even under the best prevention and control programs mycotoxins will still present.



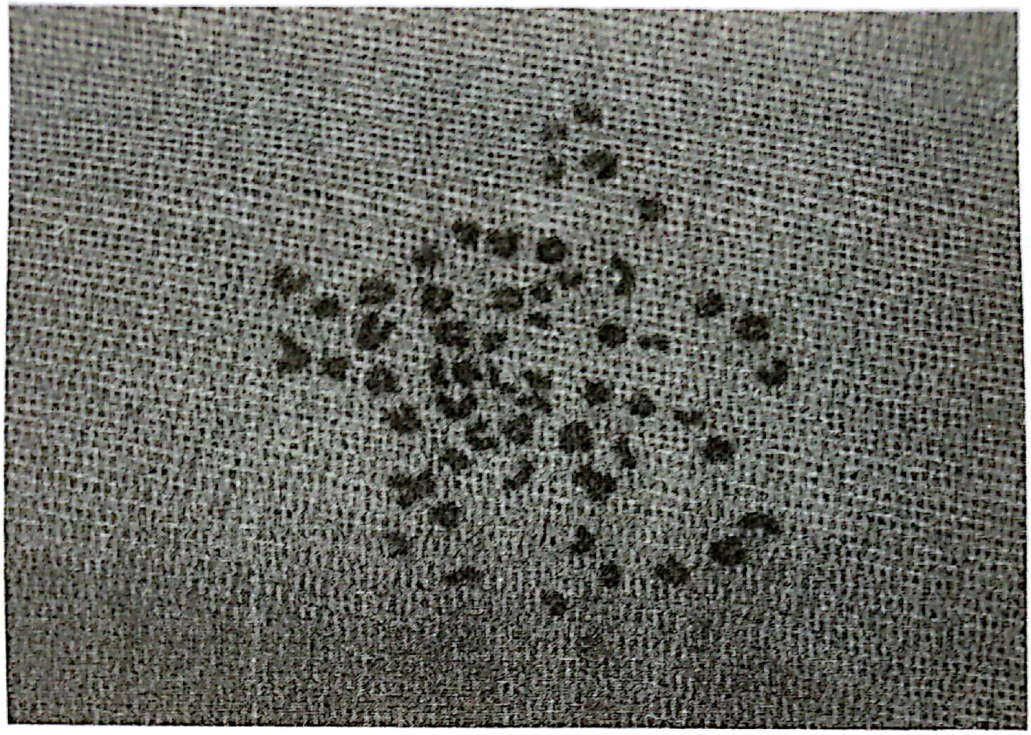
1- Normal metaphase spread of cows (60, xx) the arrows refer to sex chromosomes.



2- A metaphase spread showing deletion (d) and fragments (f).



3- A metaphase spread showing sex –chromosome monosomy (59, xo)



4- A metaphase spread showing Peridiploidy (2n-2).

References:

Aebi, H. (1984): Methods Enzymol 105, 121-126 Fossati, P., et al., (1980) Clin. Chem. 26, 227-231.

Aydin, A.; Gunsenand, U.and Demirel,S. (2008): Total aflatoxin B1 and ochratoxin levels in Turkish wheat flour. J. food and Drug analysis, 16 (2): 48-53.

Azam, M.B; Khan, Z.H.; Yagoob, S. and Khan, R.A., (2009): Nature and extent of problems of agrograziers in Bhawal pur district, Pakistan. Pakistan Vet. J., 29 (1): 32-34.

Baht, R.V. and Vasanthi,S. (2003): Mycotoxin food safety risks in developing countries. In: Food safety in food security and trade food agriculture and environment. Focus Lovision.2020, p.p.1-2.

Carrano,A.V. and Natarajan, A.T. (1988): Consideration for population monitoring using cytogenetic techniques. Mutat.Res.204, 379-409.

Cavin, C.; Delatour, T.; Marin-Kuan, M.; Holzhauser, D.; Higgins, L.; Bezencon, C.; uignard, G.and Schilter, B. (2007): Reduction in antioxidant defenses may contribute to ochratoxin A toxicity and carcinogenicity. Toxicol. Sci., 96: 30-39.

Cosimi,S.; Orta,L.; Mateos,S. and FelipeCortes,F.(2009):The mycotoxin ochratoxin A inhibits DNA topoisomerase II and induces polyploidy in cultured CHO cells. Toxicol. In Vitro.23:1110-1115

Doorten,S.Y.; Bull.; Vander Poelen ,M.A. and Fink Germmels J. (2004): Metabolism – medisated cytotoxicity of ochratoxin A. Toxicol. In Vitro 18 (3): 271-277.

Eaton, D.L. and Gallagher, E.P. (2004): Mechanisms of aflatoxins carcinogenesis. Ann. Rev. Pharmacol. and Toxicol. 34: 135-72.

EFSA (2004): Opinion of the Scientific Panel on Contaminants in the Food Chain on a request from the Commission related to Aflatoxin B1 as undesirable substance in animal feed. The EFSA Journal 39, 1-27. www.efsa.eu.int.

Emna,G.B.; Bochra,K.; Amel ,B.; Salwa,A.E.; Wafa,H. and Hassen B. (2010): Cytotoxicity and genotoxicity induced by aflatoxin B1 ochratoxin A and their combination in cultured vero cells. Journal of Biochemical and Molecular toxicology. Volume 24, Issue 7, Pages 24-50.

FAO, Food and Agriculture Organization (2004): Worldwide regulation for mycotoxins 1995.A compendium FAO Food and Nutrition, Rome, Italy.

Faulkner,W.R.and King,J.W. (1976): Determination of serum creatinine. In "fundamental of Clinical Chemistry". 2nd Ed. (N.W. Tietz Ed.), Saunder, Philadelphia, P.994.

Gremmrls, J.F. (2008): The role of mycotoxins in the health and performance of dairy cattle. Vet.J. 176: 84-92.

Groopman, J.D. and Kensler, T.W. (2005): "Role of metabolism and viruses in aflatoxin- inducel liver cancer. Toxicol. Appl. Pharmacol. 206: 131-70.

Henery,J.B.; Sanford, T.and Davidsohn: T.(1974):" Clinical Diagnosis and Measurment by Laboratory Method".16th Ed.W.B. Saunder, Philadelphia, P: 260.

Iheshiulor,O.O; Esonu,B.O.; Chuwuka, O.K; Omeda ,A.A.; Okoli,I.C.and Ogbuewu, I.P. (2011): Effects of mycotoxins in animal nutrition. A review: Asian Journal of Animal Sciences, 5: 19-33.

Korosteleva,S.N.; Smith, K.; and Boermans (2007): Effect of feedborn fusarium mycotoxins on the performance, metabolism, and immunity of dairy cows. J.Dairy Sci., 90:3867-3873.

Lanyasunya, T.P.; Wamae, L.W.; Musa, H.H.; Owofeso, O.I. and Lokwaleput, I.K. (2005): The risk of mycotoxins contamination of dairy feed and milk on small holder dairy farms in Kenya. Pakistan Journal of Nutrition, 4 (3): 162-169.

Lawlor, P.G. and Lynch, P.B. (2005): Mycotoxin management. Afr. Farming food process. ub: 12-13.

Macgregor,H.C.(1993): "An Introduction to Animal Cytogenetics" 1st Ed. Champman and Ital. London Glasgow NewYork-Tokyo-Melbourne- Madras.

Meki, A.R.; Abdel- Ghaffar, S.K; and El-Gibaly, I. (2001): Aflatoxin B1 indnecs apoptosis in rat liver: protective effect of melatonin. Neuroerdocrirol. Lett., 22: 417-26.

Nicholas, F.W. (1996): "Introduction of Veterinary Genetics". Oxiford Univ. Press. Inc. New York.

Nishikimi, M.; Roa, N.A. and Yogi, K. (1972): Biochem. Bioph. Res. Common., 46, 849-854.

NRC: National Research Council (2001): Nutrient requirements of dairy cattle. 7th. Ed. 242-248.

Ohkawa, H.;Ohishiw, and Yagi, K. Anal. Biochem. (1979) 95, 351.

Paglia, D.E. and Valentine, W.N. (1967): studies on the quantitative and qualitative characterization of glutathione peroxidase. J. Lab. Clin. Med. 70: 158-169.

Peter,F.; Julia, E.; Niek, H; and kata, J. (2007): Impact of mycotoxins on the body's antioxidant defence. Avian Science Research Center. Sumystate Agrarien University Allech North American Biosciences Center.

Ram, S.V. and Arvind, B. (1995): "Human Chromosomes Principles and Techniques" 2nd Ed. International Edition Mc Graw Hill, Inc.

Reitman, S.and Frankel, S. (1957): Calorimetric determination of GOT and GPT activity. Am.J.Clin.Path., 28:26-59.

Rumora, L.; Dsomijan, A.M.; Sic, T.Z. and Peraica, M. (2007): Mycotoxin fumonisin B1 alters cellular redox balance and signaling pathways in rat liver. Toxicology. Vol. 242: 31-38.

Scott,P.M.(1997): Natural Toxins. In: Cunnif, P. (ed). Official Methods of Analysis of Association of official Analytical Chemists. Gaithersburg, Maryland, 970.44, 971.22, 986.16.

Snedecor, G.W.and Cochran,W.C. (1982): "Statistical Methods".8th Ed.,Iowa State Univeristy Press.Ames.Iowa,USA

Stopper, A.; Schmitt, E. and Kobras, K. (2005):Genotoxicity of phytoetrogens. Mutat. Res., 574: 139-55.

Sultan, N. and Hanif, N.Q. (2009): "Mycotoxin contamination in cattle feed and feed ingredients" Pakistan Vet.J., 29 (4): 211-213.

Surai, P.F. (2006): Selenium in Nutrition and health. Nottinghan University press. Nothtinghan, U.K. task force Report, cited on page 2.

Towner,R.; Mason,R. and Reinke ,L. (2002): In vivo detection of aflatoxin induced lipid free radicals in rat bile. Biochim. Biophys.Acta, 1573: 55-62.

Umarani, M.; Shanthi, P. and Sachalanadam, P. (2008): Protective effect of kalpaamruthaa in combating the oxidative stress posed by aflatoxin B (1) induced hepato-cellular carciroma with special reference to flavonoid structure activity relationship. Liver int; 28: 200-13.

Voss, K.A.; liu J.; Anderson, S.P.; Dunn, C.; Miller, A.S.; Owen, J.D. and Bacon, R.T. (2006): Toxic effect of fumonisin in mouse liver and independent of the peroxisome proliperator activated receptor alpha. Toxicol. Sci., 89: 108-119.

Yi Jiang,P.; Joll,E.; William,O.; Jian-Sheno,W. and Jonathan,H.W. (2005) : AflatoxinB1 albumin adduct levels and cellular immunstatus in Ghanaians. Int.Immuno., 17(6):807-814.

تقييم حالة الأكسدة و بعض العناصر الكلينيكوباثولوجية والوراثية الخلوية في الأبقار الحلابة المعرضة للعليقة الملوثة بالأفلاتوكسين (ب١) والزيرالينون

الملخص العربي

اجريت هذه الدراسة علي الابقار الحلابة بهدف تقييم تأثير بعض أنواع السموم الفطرية (الأفلاتوكسين و الزيرالينون) الملوثة للعلائق علي الاتزان بين المؤاكسدات ومضادات الأكسدة وعلي وظائف الكبد والكلي بالأضافة إلي تأثيرها علي الصورة الكروموسومية .وقد تم إجرء هذه الدراسة علي ٣٣بقرة حلابة متعددة الولادات اوزانها تتراوح بين ٦٥٠-٧٥٠كيلوجرام. تم تقسيمها إلي مجموعتين الاولي تتكون من ١١ حيوان وهي المجموعة الضابطة والتي تتغذي علي عليقة لا تتجاوز الحد المسموح من الأفلاتوكسين و الزيرالينون. المجموعة الثانية تتكون من ٢٢ حيوان وتتغذي علي عليقة ملوثة بالأفلاتوكسين و الزيرالينون . تم أخذ ثلاثة عينات دم من كل حيوان الاولي لفصل السيرم لقياس عنصري النحاس والزنك بالأضافة إلي انزيمات الكبد (ALT-AST) و مستوي كل من البولينا والكرياتينين. أما العينة الثانية فقد أستخدمت لفصل البلازما لقياس نشاط إنزيمات الجلوتاثيون بيروكسيديز، السوبر أكسيد ديسميوتيزوالكاتاليزبالأضافة الي المالوندالدهيد وبالنسبة للعينة الثالثة فقد تم استخدامها في التحليل الكروموسومي . وقد أوضحت النتائج وجود نقصاً معنوياً في نشاط إنزيم الجلوتاثيون بيروكسيديز، إنزيم السوبر أكسيد ديسميوتيز، إنزيم الكاتاليز وفي المقابل كان هناك زيادة معنوية في مستوي المالوندالدهيد اما النحاس والزنك فقد سجلا نقصاً غير معنوياً . كما أظهرت إنزيمات الكبد والبولينا زيادة معنوية أما زيادة الكرياتينين فقد كانت غيرمعنوية. أما بالنسبة للتحليل الكروموسومي فقد أوضحت الدراسة زيادة معنوية في الاختلالات العددية والتركيبية (الأجزاء الممحاه والأجزاء المكسورة) وكذلك زيادة معنوية في عدد الخلايا المحتوية علي كروموسوم جنسي واحد وعموماً كان هناك زيادة معنوية في النسبة المئوية للخلايا الغير طبيعية . ونستخلص من هذه الدراسة أن سموم الأفلاتوكسين و الزيرالينون الملوثة للعليقة لها آثار سلبية علي مستوي المؤاكسدات ومضادات الاكسدة وعلي وظائف الكبد وكذلك علي الصورة الكروموسومية للابقار الحلابة . لهذا ننصح برفع مستوي وسائل التحكم ومنع تلوث العلائق بهذه السموم تجنباً لآثارها السلبية والاقتصادية علي انتاجية المزارع.