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Toxicological effects of the fumonisin B_1 on some physiolgical and biochemical parameters in the rainbow trout fish, *Tilapia florida*

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Abstract

Fumonisin FB_1 is a mycotoxin produced naturally by the fungi, *Fusaium moniliforme* and *F. proliferatum*, on corn and corn-based products. This study is the first to evaluate toxicity of the purified FB_1 in rainbow trout fish, *Tilapia florida*. Fish (10/group) were injected intrapretonially with a single dose of either saline vehicle (control) or FB_1 (2.5, 5.0 or 7.5 mg/kg bw). After 24 hrs, the response to the mycotoxin based on relative organ weights, haematology and biochemistry was assayed. The results showed that the treated fish reflected alterations in relative organ weights. FB_1–treated fish exhibited significant elevation in erythrocyte count, hemoglobin (Hb) content, as well as plasma levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), albumin and total bilirubin. In contrast, plasma levels or acid phosphatase (ACP), alkaline phosphatase (ALP), glucose and cholesterol were significantly reduced, particularly with the 7.5 mg FB_1 /kg bw dose. These results suggest mainly that FB_1 induces profound signs of toxicities in *T. florida* where the liver and kidney seem to be major target organs. The study provides that *T. florida* is a highly sensitive species to FB_1 and thus can be used successively as an experimental model in the future studies.

Key words: *Tilapia florida*, fumonisin FB_1, physiological parameters, biochemical parameters

Introduction

The mycotoxin fumonisin (FB_1) is produced by the ubiquitous fungi, *Fusarium moniliforme* and *F. proliferatum* a worldwide contaminant of major grain crops, especially abundant in corn and corn-based products for both animal and human consumption (Bacon and Nelson, 1994). Animals, as well as humans, are exposed to mycotoxins through consumption of contaminated food in the diet, which can be considered the gateway to cases of natural intoxication by these compounds (Gutema *et al.*, 2000). Contamination with FB1 has been detected in different countries (e.g. Brazil, Canada, Egypt, Italy, New Zealand, Peru, South Africa and USA) in cereals and corn-oased food products (Machinsk and Soares, 2000).

Diseases induced by mycotoxins cause acute, chronic and subchronic toxicities, which depends on different factors such as the animal species, age, sex, strain, dosage and administration route (Hengtler *et al.*, 1999). Ingestion of *Fusarium moniliforme* contaminated food or application of purified FB_1 have been linked to fatal animal diseases including: leukoencephalomalacia in horses (Wilson *et al.*, 2008), pulmonary edema in pigs (Osweiler *et al.*, 1992), hepatocellular carcinoma in rats (Gelderblom *et al*. 1994) and cardiovascular toxicity (Smith *et al.*, 1996). Effects of FB1 on human are unclear, but epidemiologic evidence suggests that consumption of FB_1 contaminated corn contributes to human esophageal cancer in regions of South Africa, China, Italy and USA (Sydenham *et al.*, 1990: Voss *et al.*, 2001).

Acute, sub-acute and chronic toxicities of purified FB_1, singly or in combination with other mycotoxins, have been widely studied in different domestic and laboratory animals, such as rats (Bondy *et al.*, 1995: Pozzi *et al.*, 2001), mice (Voss *et al.*, 1995 and Casado *et al.*, 2001), goats (Gurung *et al.*, 1998). Turkeys, chickens and ducks (Baily *et al.*, 2001 and Broomhead *et al.*, 2002). General toxicities of liver, kidney and hematopoetic toxicity, immune toxicity reproduction toxicity, foetal toxicity and teratogenicity, and mainly catcinogeicity have been mostly known in these experimental models.

Based on the fact that FB_1 is highly polar (Murphy *et al.*, 1993), the water resources can be easily spoiled from the contaminated corn and corn-based products. Moreover, samples of fishmeal were analyzed for FB_1 and 94% of them were found to be positive in Natal, South Africa (Dutton and Kinsey, 1995). No available data are documented on the toxicity of FB_1 in fish.

The main objective of this study was to evaluate the acute toxicity of a single dose of FB_1 in *T. florida*. This fish was chosen because it is a highly sensitive species to the toxicity of other mycotoxins such as (T-2, diacetoxyscripenol (DAS), deoxynivalenol (DON), aflatoxin B_1ochratoxin A and tricothecenes) (Valsta *et al.*, 1988; Abd-Allah *et al.*, 1999; El-Sayed *et al.*, 2003 and Galtier, 2007). Besides, it is usually considered as a model for other toxicological studies (Bailey *et al.*, 2004). Additionally, this study aims also to demonstrate whether the rainbow trout can be used as an experimental model for studying the toxicity of FB_1.

Materials and Methods

Fishes:

A total number of 40 adult of rainbow trout fish, *Tilapia florida* were collected from the Tawarga pond at Misurata city, Libya. The average of sample weight ranged between 100-120g. The fish were immediately transported after catching to special aquaria in the laboratory (100 liter capacity) filled with aerated fresh water. The water temperature was adjusted thermostatically at 22±0.05°C. Fish were divided into 4 equal groups, acclimatized in well aerated aquaria at 11°C, and deprived of food for one week prior to initiation of the experiment.

Chemicals:

A purified FB_1 and diagnostic kits of alanine aminotransferase ALT, aspartate aminotransferase AST, acid phosphatase ACP, and alkaline phosphatase ALP were purchased from Sigma Chemical Co., St. Louis, MO, USA.

Dosage and Sampling:

The purified FB_1 was dissolved in sterilized saline 0.85%. Three doses were prepared at concentrations of 2.5, 5.0 and 7.5 mg FB_1 /ml saline/kg bw. The fish were injected intrapretonially (ip) with a single appropriate dose of the toxin (treated) or an equal volume of sterilized saline (control). 24-hrs after treatment, fish were anesthetized with tricaine methanestlfonate (1/2000 wt/vol) and the blood was immediately collected from the caudal region into two tubes, the first 5 ml Becton Dickinson vacutainer tube containing sodium heparin for haematological studies. A portion of heparinized blood was taken into a micro-tube, and the remaining was centrifuged at 3000 rpm for 15 min to obtain the plasma. After blood collection, liver and kidneys were excised and weighed.

Haematology and Biochemistry:

The total erythrocyte count was calculated according to Dacie and Lewis (1984). Haemoglobin content was determined according to Drabkin's method (1932) using a test kit. Spectrophotometric methods using Sigma diagnostic kits were applied for the determination of the following plasma enzymes: ALT, AST. ACP and ALP. The concentrations of plasma glucose and cholesterol were determined using commercially available enzymatic kits while plasma total protein, albumin and total bilirubin were assayed spectrophotometrically using commercial kits.

Statistical Analysis:

Data are represented as means ± SD, % change from control and *p*-value. For each parameter, Student t-test was used for the comparison between treated and control values. Differences were considered significant at 3 levels $P<0.05$, $P<0.01$ or $P<0.001$.

Results

No mortality was obtained after 24 hrs in all *Tilapia florida* treated with the 2.5 5.0 or 7.5 mg FB_1 /kg bw doses. In general, FB_1 –treated fish appeared lethargic, particularly at the 7.5 mg FB_1 /kg bw level, as compared with the controls.

Relative weights of the liver and kidney (expressed as a percentage to the body weight) for all control and treated fish groups are summarized in Table (1). Relative liver weight was significantly elevated ($P<0.05$) in fish groups treated with 5.0 or 7.5 mg FB_1 /kg bw above that of the control. In contrast, relative kidney weight was significantly reduced in all treated fish groups, and the effect was more pronounced as the FB_1 dose increased.

Table (2) presents enzyme activities of plasma ALT, AST, ACP and ALP for the control and treated fish groups. Activities of both aminotransferases were higher in FB_1 - treated fish than that obtained from the control group. Plasma ALT was significantly higher ($P<0.01$) with only the 7.5 mg FB_1 /kg bw level, whereas plasma AST was significantly higher in all treated groups and increased with the increase of the FB_1 dose. In contrast, plasma ACP and ALP levels were significantly lower than those of the control with the 5.0 and 7.5 mg FB_1 /kg bw doses. In general, it seems that FB_1 induces a dose-dependent effect on the activities of tested plasma enzymes.

Data of plasma concentrations of glucose, cholesterol, total protein, albumin and total bilirubin are given in Table (3). Glucose concentration was significantly reduced ($P<0.05$) with the 5.0 and 7.5 mg FB_1 /kg bw levels, and the plasma cholesterol was significantly ($P<0.001$) decreased with the 7.5mg FB1 /kg bw dose. No significant difference was observed in the total protein concentration of FB_1 –treated fish groups as compared with the control one, while a significant ($P<0.05$) increase was obtained in albumin concentration of the group treated with the 7.5mg FB_1 /kg bw. The concentration of total plasma bilirubin was significantly higher in fish groups treated with 5.0 and 7.5 mg FB_1 /kg bw in comparison with the corresponding control values. No significant changes were generally observed in the tested plasma metabolites in the group treated with the lower toxin dose (2. 5mg FB1 /kg bw).

Table (4) summarizes the haematological results. *T. florida* treated with FB_1 showed dose- dependent elevations in both erythrocyte count and Hb content as compared with the control group. The increase was significant at $P<0.05$ and $P<0.01$ in erythrocyte count with the 5,0 and 7.5 mg FB_1 /kg bw doses, respectively, but it was significant at $P<0.05$ in Hb-content with the 7.5 mg /kg bw dose.

Discussion

Because of the greater absorption of FB_1 following ip dosing than after oral dosing, increased amounts of toxin reach the general circulation (Shephard *et al.*, 1992). Moreover, Bondy *et al.* (1995) used ip injection of FB_1 in rats to attain greater toxin bioavailability and to maximize effects on potential target organs using small quantities of toxin over a short time period. In the present study, the intraperitoneal route of administration was also applied to attain the same effect. In rats, a single ip dose of 7.5 mg FB_1 /kg bw was used to study its fate (Shephard *et al.*, (1992). Bondy *et al.* (1995) used 7.5 and 10.0 mg FB_1 /kg bw for 4 consecutive days to evaluate FB_1 toxicity in rats, as well. Because *T. florida* is well known as a sensitive species to other mycotoxins (Valsta *et al.*, 1988; Abd-allah *et al.*, 1999), doses of 7.5 mg FB_1 /kg bw or less (2.5 and 5.0 mg FB_1 /kg bw) were used herein for a comparative purpose. The results revealed a number of FB_1-related changes that have not previously been reported in fish and deserve consideration in future subchronic and long-term studies.

Inhibition of ceramide synthase and disruption of membrane phospholipids have been shown to be mechanisms of FB_1 toxicity (Ramljak *et al.*, 2000). Galvano *et al.* (2002), described the first mechanism where FB_1 structurally resembles sphingoid basis an inhibitor of ceramide synthase, a key enzyme involved in *de novo* sphingolipid biosynthasis and in the reacylation of free sphingoid basis derived from sphingolipid turnover. This inhibitory effect leads to accumulation of free sphinganine and sphinosine, and subsequent induction of cell death or damage. The sphingolipid analysis of liver, kidney, and heart tissues of FB_1-treated goats showed elevated free sphinganine (Gurung *et al.*, 1998). Voss *et al.* (2001) reported also that liver sphingolipid-induced effects of $FB_1$and toxicity are correlated, and ceramide synthase inhibition occurs in liver and kidney at doses below their respective no-observed effect level. Toxicity of FB_1 may also be due to its own hydroxyl radicals, which initiate the nuclear membrane lipid peroxidation and produce peroxyl radicals, leading to cell death or damage (Sahu *et al.*, 2010).

The measurement of enzyme activities, particularly ALT and AST, in serum is frequently used as a diagnostic tool in human medicine (Dufour *et al.*, 2000a &b). Bucher and Hofer (2009) reported also that disturbance of blood enzymes is indicative of intoxication in fish. Because the elevation of plasma ALT and AST is usually correlated with cell damage of the liver, elevations of both enzymes associated with the 7.5 mg FB_1 /kg bw dose herein indicate that the hepatotoxic effects are induced in *Tilapia florida* at this level of the toxin. The damaging effect of FB_1 in liver cells may be attributed to its impacts on ceramide synthase and membrane phospholipids mentioned above. Several previous studies also confirmed that the liver is a target organ for toxicity of FB_1in different animal species. These results revealed that the toxicity was also concomitant with elevations in serum aminotransferases (Bondy *et al.*, 1995; 2000; NTP, 2001). Moreover, Gelderblom *et al.*, (1997) reported that the disturbance in phospholipid, cholesterol and fatty acid metabolic pathways might also be important factors in the FB_1-induced hepatictoxicity. Consequently, the reduction of plasma glucose and cholesterol, obtained herein with the high level of the toxin, might have been ascribed to the intervention of FB_1 with the lipid biosynthesis in the liver cells as mentioned by Gelderblom *et al.*, (1997). The perturbations in ACP activity are considered a sensitive index of environmental pollution and a good biomarker for toxic effects and stress response in fish. Consequently, the perturbations in plasma ACP and ALP activities, as well as plasma bilirubin, induced herein by FB_1, affirm the impact of the toxin on the hepatocytes in *T. florida*.

The nephrotoxicity of FB_1 in rodents has been extensively documented (Suzuki *et al.*, 1995 and NTP, 2001) and the kidney was early considered the primary target organ for the toxin effect (Riley *et al.*, 1994). Riley *et al.* (1994) reported that following FB_1 exposure, similar to other established target organs of toxicity such as liver, metabolism of sphingolipids was altered in the kidney and that these alterations preceded changes in renal pathology. It is conceivable that FB_1 might have affected certain renal functions, particularly the water retention capability. The reduction of relative kidney weights, even with the small levels of the toxin observed herein, as well as the elevations of erythrocyte count, Hb-content, total protein and albumin could have been attributed, at least in part, to dehydration, which is a nephrotoxic indicator. Besides, the increased activity of plasma AST, rather than plasma ALT, observed herein for the small doses of the toxin may be attributed to the primary effect of the toxin on the kidney (Riley *et al.*, 1994) or to cardiovascular toxicity (Smith *et al.*, 1996) prior to its effect on the liver.

Finally, this study indicates mainly that FB_1 induces profound signs of toxicity in *Tilapia florida*, in which the liver and kidney seem to be major target organs. By comparing the present results with some of the previous ones in rats (Bondy *et al.*, 1995), mice (NTP, 2001), and poultry (Henry *et al.* 2000; Bailly *et al.* 2001 and Broomhead *et al.* 2002), it is evident that *T. florida* is a highly sensitive species to FB_1 toxicity. Consequently, this fish species may be used successfully as an experimental model in detecting waters polluted with FB_1 in the future studies.

Table 1. Relative organ weights of *T. florida* injected ip with a single dose of sterile saline (control) or fumonisin B_1.

parameters		Control	Fumonisin B_1(mg/kg b.wt)		
			2.5	5.0	7.5
Relative liver weight	Mean ± SD	0.89± 0.029	0.90± 0.024	0.93± 0.053	0.90± 0.044
	%change from control	---------	+20	+47	+50
	p-value	---------	NS	<0.05	<0.05
Relative kidney weight	Mean ± SD	0.58± 0.057	0.53± 0.039	0.49± 0.066	0.47± 0.055
	%change from control	---------	-95	-158	-192
	p-value	---------	<0.05	<0.01	<0.001

NS = non- significant

Table 2. Activities of some plasma enzymes of *Tilapia florida* injected ip with a single dose of sterile saline (control) or fumonisin B_1.

parameters		Control	Fumonisin B_1(mg/kg b.wt)		
			2.5	5.0	7.5
ALT (IU/l)	Mean ± SD	14.50± 2.95	15.45± 2.67	16.12± 3.09	40.36± 3.25
	%change from control	---------	+5.5	+112	+178.3
	p-value	---------	NS	NS	<0.001
AST (IU/l) (IU/l)	Mean ± SD	151.2± 14.7	172.0± 21.2	180.0± 20.0	215.0± 29.7
	%change from control	---------	+13.8	+19.0	+42.9
	p-value	---------	<0.05	<0.01	<0.001
ACP (IU/l)	Mean ± SD	22.90± 2.91	20.50± 2.98	17.10± 3.99	15.80± 2.70
	%change from control	---------	-10.5	-25.3	-31.0
	p-value	---------	NS	<0.01	<0.001
ALP (IU/l)	Mean ± SD	82.40± 5.45	75.80± 7.54	55.50± 4.55	54.40± 3.37
	%change from control	---------	- 8.0	- 20.4	- 21.8
	p-value	---------	NS	<0.001	<0.001

ALT = alanine aminotransferase AST = aspartate aminotransferase
ACP = acid phosphatase ALP = alkaline phosphatase
NS = non- significant

Table 3. Some plasma constituents of *Tilapia florida* injected ip with a single dose of sterile saline (control) or fumonisin B_1.

parameters		Control	Fumonisin B_1(mg/kg b.wt)		
			2.5	0.5	7.5
Glucose (mg/dl)	Mean ± SD	119.0 ± 6.15	115.4 ± 4.30	111.4 ± 6.98	112.2 ± 6.03
	%change from control	---------	-30	-64	-56
	p-value	---------	NS	<0.05	<0.05
Cholesterol (mg/dl)	Mean ± SD	171.0 ± 15.8	170.0 ± 12.8	169.8 ± 12.2	140.0 ± 14.9
	%change from control	---------	-0.6	-0.7	-18.1
	p-value	---------	NS	NS	<0.001
Total protein (g/dl)	Mean ± SD	5.24 ± 0.288	5.26 ± 0.310	5.34 ± 0.227	5.48 ± 0.469
	%change from control	---------	+0.4	+1.9	+4.6
	p-value	---------	NS	NS	NS
Albumin (g/dl)	Mean ± SD	3.41 ± 0.241	3.44 ± 0.331	3.52 ± 0.181	3.78 ± 0.454
	%change from control	---------	+0.9	+3.2	+10.9
	p-value	---------	NS	NS	<0.05
Bilinrubin (mg/dl)	Mean ± SD	0.11 ± 0.023	0.12 ± 0.024	0.13 ± 0.023	0.17 ± 0.023
	%change from control	---------	+13.0	+22.2	+55.6
	p-value	---------	NS	<0.05	<0.001

NS = non- significant

Table 4. Erythrocyte count and Hb content of *Tilapia florida* blood injected ip with a single dose of sterile saline (control) or fumonisin B_1.

	parameters	Control	Fumonisin B_1(mg/kg b.wt)		
			2.5	0.5	7.5
Erythrocyte count×10^2	Mean ± SD	1.10 ± 0.15	1.27 ± 0.23	1.29 ± 0.22	1.45 ± 0.30
	%change from control	---------	+15.1	+17.3	+31.5
	p-value	---------	NS	<0.05	<0.01
Hemoglobin Content ,mg/dl	Mean ± SD	4.34 ± 0.24	4.36 ± 0.28	4.48 ± 0.14	4.73 ± 0.44
	%change from control	---------	+06	+3.3	+9.1
	p-value	---------	NS	<0.05	<0.01

NS = non- significant

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التأثيرات السامة للسم الفطرى فيومونيزين ب 1 علي بعض الجوانب الفسيولوجية والبيوكيميائية في سمك تلابيا فلوريدا

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الفيومونيزين ب1 هو سم فطري ينتج طبيعيا بواسطة فطرفيوزاريوم مونليفورمى وفيوزاريوم بروليفراتم علي حبوب الذرة ومنتجاتها. وتعد هذه الدراسة لتقييم السمية الحادة **للفيومونيزين ب1** النقي في سمكة تلابيا فلوريدا . لذلك حقنت الأسماك (المجموعة تشمل عشرة سمكات) في التجويف البريتوني بجرعة وحيدة إما من المحلول الملحي (الضابطة) او **الفيومونيزين ب1** وذلك في ثلاث مستويات هي 2.5، 5.0 و7.5 مجم/كجم من وزن الجسم، ثم اختبرت الإستجابة لهذا السم بعد 24 ساعة بفحص الوزن النسبي للأعضاء وبعض معايير الدم والكيمياء حيوية.

وقد أوضحت النتائج حدوث تغييرات في الوزن النسبي للأعضاء في الأسماك المعاملة، كما سبب **الفيومونيزين ب1** ارتفاعا معنويا في كل من عدد كرات الدم الحمراء والمحتوى الهيموجلوبينى وكذلك مستويات البلازما من الإنزيمات الناقلة للامين (AST وALT) والالبيومين والبليروبين، وقد كانت الزيادة مرتبطة بزيادة الجرعة وفي المقابل انخفضت معنويا مستويات البلازما لكل من ALP و ACP والجلوكوز والكوليسترول في الأسماك المعاملة، خاصة مع الجرعة 7.5 مجم/كجم من وزن الجسم.

ويستخلص من هذه النتائج ان**الفيومونيزين ب1** يحدث نطاق واسع من التأثيرات السامة في سمكة تلابيا فلوريدا ، ويبدو ان الكبد والكلية هما عضوين مستهدفين رئيسيين، كما تقدم الدراسة دليلا علي حساسية سمكة تلابيا فلوريدا **للفيومونيزين ب1** ، لذلك يمكن استخدامها بنجاح كنموذج تجريبي في الدراسات المستقبلية.