

Effects of different dietary levels of AFB₁ on survival rate and growth factors of Beluga (*Huso huso*)

Sepahdari A.^{1,2*}; Ebrahimzadeh Mosavi H.A.²; Sharifpour I.¹;
Khosravi A.²; Motallebi A.A.¹; Mohseni M.³; Kakoolaki S.¹;
Pourali H.R.³ and Hallajian A.³

Received: January 2009

Accepted: February 2009

Abstract

In the present study, the impacts of various concentrations of Aflatoxin B₁ (AFB₁) on Beluga, *Huso huso*, under controlled conditions were investigated. Belugas (120±10g) were fed on diets containing 0, 25, 50, 75, and 100 ppb AFB₁/kg of diet for 3 months. Results showed various levels of AFB₁ do not significantly affect the specific growth ratio (SGR) (P<0.05) of fish in different treatments. However, weight gain and food conversion ratio (FCR) were varied significantly (P<0.05 between control and treatments with diets contaminated with 75 and 100ppb AFB₁/kg after 90 days). The increase AFB₁ level of did not affect on the percent of survival rate(SR) and no mortality was observed in treatments (SR=100%) suggesting that various AFB₁ levels under experimental conditions of the present study affect some growth factors, such as, weight gain and FCR but have no significant impact on SR and SGR. Histopathological studies showed that different level of AFB₁ can cause broad range of change in liver tissue, including progressive fat deposition, hepatocyte degeneration and necrosis, particularly at concentration of 75 and 100ppb AFB₁/kg of diets after 60 days.

Keywords: *Huso huso*, AFB₁, FCR, SGR, SR, Pathology, Liver

1-Iranian Fisheries Research Organization, P.O.Box:14155-6116 Tehran, Iran

2-Faculty of Veterinary Medicine, University of Tehran, P.O.Box:14155-6453 Tehran, Iran

3-International Sturgeon Research Institute, P.O.Box:41635-3464 Rasht, Iran

* Corresponding author's email: asepahdari@yahoo.com

Introduction

The primary objective in fish nutrition is to provide a nutritionally balanced mixture of ingredients to support the maintenance, growth, reproductive performance, flesh quality and health of the animals at an acceptable cost. The diet also has an important effect on water quality and culture systems. In order to achieve these goals the diet must provide all required nutrients in the correct balance and must be formulated to keep any anti-nutritional components below concentrations that would impede the performance and health of the fish (NRC, 1993). Over the last 10 years plant-based ingredients have been increasingly used in fish diets. This change is the product of increased economic/market pressures on the fish meal and oil manufacturing industries and animal feed compounders, and the drive to produce lower cost, sustainable alternatives by the aqua feed manufacturing sector (Tacon, 2004). The extent of the damage produced by aflatoxins depends on the toxin concentration present in foods or feeds and also on the time period of exposure, as well as animal species susceptibility (Stewart & Larson, 2002). Within a given species, the magnitude of toxicity is influenced by age, sex, weight, diet, and exposure to infectious agents. Fry are more susceptible to aflatoxicosis than adults, and certain fish species are more sensitive than others (Royes & Yanong 2002). Since the discovery of the nature of aflatoxins, aflatoxicosis has been investigated predominantly in freshwater aquatic species, especially in rainbow trout, (*Oncorhynchus mykiss*) (Halver, 1969; Lovell,

1989; Hendricks, 1994; Gallagher & Eaton, 1995) and, also, in American channel catfish (*Ictalurus punctatus*) (Lovell, 1984; Jantrarotai & Lovell, 1990; Jantrarotai *et al.*, 1990; Plakas *et al.*, 1991; Hendricks 1994; Gallagher & Eaton, 1995), Nile tilapia (*Oreochromis niloticus*) (Chavez-Sanchez *et al.*, 1994; Tuan *et al.*, 2002), Indian major carp (*Labeo rohita*) (Sahoo *et al.*, 2001; Sahoo *et al.*, 2003; Murjani 2003), mosquitofish (*Gambusia affinis*) (McKean *et al.*, 2006), guppy (*Lebistes reticulatus*) (Sato *et al.*, 1973), and in some invertebrate species such as *Penaeus monodon*, *Penaeus stylirostris*, *Penaeus vannamei* (Wiseman *et al.*, 1982; Lightner *et al.*, 1982; Boonyaratpalin *et al.*, 2001), brine shrimp (*Artemia salina*) (Reiss 1972a) and a copepod (*Cyclops fuscus*) (Reiss, 1972b).

The only research regarding the effects of AFB₁ on sturgeon fish was carried out by Farabi *et al.* (2006) on juvenile beluga (*Huso huso*). Therefore, the purpose of the present study was to assess the effect of different levels of AFB₁ on the growth and survival rate of *Huso huso* as economical and native Iranian species to provide additional information to the current knowledge.

Materials and methods

Five hundred fish of approximately the same size (100±10g) were obtained from the Shahid Beheshti Sturgeon Rearing Centre in north of Iran. The fish were acclimated to 500 liter tank and fed on a control diet for 30 days before the AFB₁ experiment began. Twelve fish of approximately the same size (120g average)

were stocked into each of the 15 tanks which contained 200 liter of water. Water from a reservoir was passed continuously into tanks and water exchange was about 200% during 24 hours. Water temperature was maintained at $18 \pm 2^\circ\text{C}$. Aeration was provided by an air blower and distributed continuously to each tank through an air-stone to maintain dissolved oxygen at a level of at least 6 mg/l. Three tanks were randomly assigned to each of the five dietary treatments. The fish were fed 3% body weight three times a day at 08:00, 12:00, and 16:00h for 3 month. The remained feed and feces siphoned daily at 16:00 and tanks were cleaned weekly.

The experimental diets were formulated to contain 0, 25, 50, 75, and 100ppb AFB/kg diet from pure SIGMA product. Ingredients were mixed and then AFB₁ which was dissolved in Methanol was sprayed during mixings process, for five minutes on each experimental diet. Then diets extruded to produce semi moist pellets and moisture content decreased to about 10%, through drying. Dried diets were stored in paper bags in cold room until fed. The amount of AFB₁ in experimental diets, confirmed by HPLC prior to feeding

Feed consumption was recorded daily. Fish were anesthetized in clove flower powder solution (250-300ppb in water) in two weeks intervals, then length and weight of each individual determined and recorded.

Growth rate was determined as weight gain (g) at 2-weeks intervals. WG, FCR, SGR and SR were calculated as follows:

$$\text{W.G.} = W_1 - W_0$$

W_0 =Initial weight

W_1 = Final weight

F.C.R. =Dry feed consumption/ $W_1 - W_0$

S.G.R. = $\ln W_1 - \ln W_0 / T$ (days of culture)

S.R. = (Number of live fish/ Initial number of fish) $\times 100$

For histopathology, at the end of each month the liver were removed from three fish from each tank and fixed in Bouin's solution, dehydrated in Isopropanol, cleared in Histosol, infiltrated in Paraffin, and sectioned at a thickness of 5μ . Sections were stained with hematoxylin and eosin (H & E), and examined with a light microscope.

Mean weight gain, feed conversion ratio, and specific growth rate were analyzed by Analysis of Variances. Duncan's multiple range tests were used to compare different treatment groups (P level = 0.05).

Results

Table 1 summarized average body weights of experimental fish after 90 days feeding with different levels of dietary AFB₁.

There was significant difference in average body weight (ABW) of fish between T₃ and T₄ with T₁ and control in third month biometry (P<0.05) (Table 1).

Table 1: Monthly comparison of the ABW \pm SD in different treatments

treatment►	Control	T ₁ (25ppb)	T ₂ (50ppb)	T ₃ (75ppb)	T ₄ (100ppb)
▼ time					
First month	362.2 \pm 42.9 ^c	339.2 \pm 46.8 ^{abc}	329.1 \pm 51.2 ^{ab}	356.2 \pm 58.2 ^{bc}	323.6 \pm 60.3 ^a
Second month	484.3 \pm 54.7 ^e	454.8 \pm 43.1 ^{de}	429.2 \pm 58.2 ^d	418.4 \pm 75.4 ^d	429.2 \pm 65.8 ^d
Third month	502.4 \pm 51.3 ^s	470.0 \pm 69.5 ^{fg}	443.4 \pm 69.7 ^f	421.9 \pm 86.9 ^f	431.5 \pm 76.9 ^f

Common scripts show no significant differences at the 0.05 level.

Similar results were acquired for FCR and SGR (Table 2). There was significant difference between FCR in treatment two (diet contained 75ppb AFB₁) and control diet (P<0.05). No significant difference was observed between other treatments. Also, there was no significant difference between treatments in relation with SGR. No mortality

observed in experimental fish and survival rate was 100% in all treatments.

Tissue liver of the experimental fish in different treatments was subject to various changes (Figs. 1 & 2). The range of changes varied widely and these were chronic manifestation (Table 3).

Table 2: Comparing of FCR and SGR in experimental treatments after 90 days (\pm SD)

Treatments	AFB ₁ levels (ppb)	SGR \pm SD	FCR \pm SD
Diet 1	100	0.83 \pm 0.03	2.22 \pm 0.16 ^{ab}
Diet 2	75	0.80 \pm 0.04	2.56 \pm 0.30 ^a
Diet 3	50	0.89 \pm 0.04	2.02 \pm 0.20 ^{ab}
Diet 4	25	0.91 \pm 0.04	1.88 \pm 0.13 ^{ab}
Control	0	0.94 \pm 0.02	1.70 \pm 0.07 ^b
P -Value		0.034	0.017

Common scripts show no significant differences at the 0.05 level

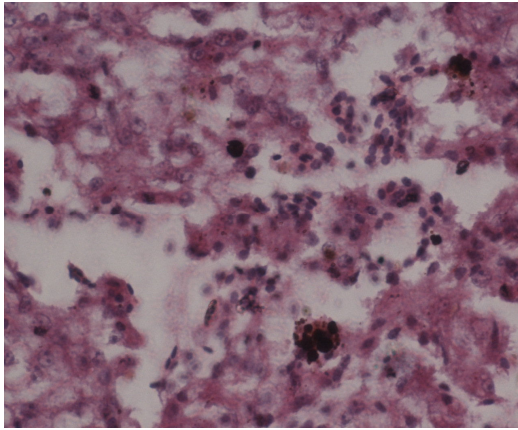


Figure 1: Hemorrhage, fat deposition, degeneration of hepatocytes, mild necrosis, presence of MMC in liver parenchyma in T₁ at 2nd sampling (H&E ×40).

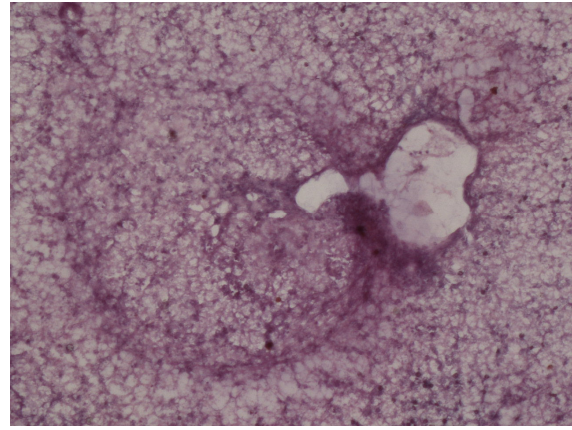


Figure 2: Degeneration of hepatocytes, necrosis, increase MMC and fibrocytes, granuloma like structure, T₄ at 2nd sampling (H&E ×20).

Table 3: Histopathological changes of the liver tissue in the present study

Time Treatment	First month	Second month	Third month
T1	Congestion, hepatocyte swelling, fat deposition	Hemorrhage, fat deposition, hepatocyte degeneration, mild necrosis, presence of MMC,	Hepatocyte degeneration, mild necrosis, increase MMC
T2	Congestion, fat deposition, hepatocyte degeneration, nuclear pyknosis and mild necrosis	Hemorrhage, fat deposition, hepatocyte degeneration, nuclear pyknosis and mild necrosis	Fat deposition, hepatocyte degeneration, focal necrosis, increase inflammatory cells and MMC
T3	Severe congestion, fat deposition, hepatocyte degeneration, nuclear pyknosis and mild necrosis	Fat deposition, hepatocyte degeneration, focal necrosis, increase MMC	Fat deposition, hepatocyte degeneration, confluent necrosis, increase MMC
T4	Severe congestion, fat deposition, hepatocyte degeneration, nuclear pyknosis and mild necrosis	Hepatocyte degeneration, confluent necrosis, increase MMC and fibrocytes, granuloma	Hepatocyte degeneration, confluent necrosis, increase MMC, granuloma, fibrosis

Discussion

Farabi *et al.* (2006) in a study on juvenile *Huso huso* reported 8.6% mortality during 15 days feeding contaminated diet (amount of contamination in this study was not defined). Similarly, there was a significant difference in the survival rate between Nile tilapia fed with the diet containing 100mg AFB/kg and the other diets (Tuan *et al.* 2002). In this study, survival of fish fed with 100mg AFB/kg was 55% after 6 weeks of feeding, and at the end of the feeding period (8 weeks) it was 40%. In contrast, in the present study, however, Beluga was fed with various levels of AFB₁ (25, 50, 75 and 100ppb/kg of diet) for three months at 18±2°C, no mortality occurred. This may be due to the sensitivity of sturgeon fish to aflatoxins. Further research on sensitivity of sturgeons is necessary to clarify this matter. Since early 1990s, channel catfish have been shown to be refractory to AFB₁ toxicity. Also, Coho salmon and Zebra fish have been reported to be resistant to AFB₁ hepatocarcinogenesis (Jantrarotai & Lovell, 1990; Plakas *et al.* 1991; Hendricks, 1994; Gallagher & Eaton, 1995; Tsai, 1996). Experimental studies conducted in catfish exposed to a wide range of AFB₁ concentrations in diets (ranged from 100 to 2,154ppb AFB₁), revealed that there was no significant reduction in weight gains or histopathological findings (Jantrarotai & Lovell, 1990). Our study showed that there was significant reduction in weight gain in 75 and 100ppb concentrations of AFB₁ with control after two months and liver histo-

pathological changes from sever congestion to confluent necrosis is noticeable in all treatments after three month. Early clinical signs of acute aflatoxicosis, e.g., decreased growth performance, anaemia, liver and gastric necrosis, were observed in catfish only when levels of 10,000ppb AFB₁ were fed (Jantrarotai & Lovell, 1990). The 10-day median LD50 of AFB₁ for channel catfish by i.p. administration is 11.5mg/kg body weight (Jantrarotai *et al.*, 1990), which is about 14- and 20-fold higher than the LD50 of rainbow trout by i.p. administration or oral administration, respectively.

Another fish species may be moderately resistant to AFB₁ is Nile tilapia, however, it is more susceptible than channel catfish to the acute effects of AFB₁ (Tuan *et al.* 2002). For instance, Chavez-Sanchez *et al.* (1994) reported that a dose of 30mg AFB/kg diet was not lethal to this species. When aflatoxin-contaminated feed (115.34ppb) was administered to Nile tilapia for 120 days, only chronic granulomatous inflammation was observed in the liver, with no tumor formation (Cagauan *et al.*, 2004). These results were close to our finding in relation to lethal dose. However, we did not observe tumor formation but liver damages were more progressive in Beluga. Furthermore, Nile tilapia that were fed diets with 10mg AFB/kg for 8 weeks had 90% reduction in growth rates compared to 24% growth reduction in channel catfish fed the same amount (Jantrarotai & Lovell, 1990; Tuan *et al.*, 2002).

According to Tuan *et al.* (2002) the intensity of adverse effects induced in Nile tilapia, by AFB₁ dietary administration for 8 weeks, increased proportionally along with the aflatoxin concentration ingested: fish fed diets containing 2.25mg AFB/kg had changes in growth rate, whereas 10mg AFB/kg produced hepatic lesions, and 100mg AFB/kg caused severe hepatic necrosis and 60% mortality, as reviewed by Gallagher and Eaton (1995).

Average body weight and FCR of Beluga were significantly affected by 75 and 100ppb AFB₁ in diet but not by 50ppb or less. Although, there was significant differences between FCR in treatment two (Diet with 75ppb AFB₁) with control diet and no statistical different observed between other treatments, but functional differences could have economical effects on large farm production. There was no significant difference in SGR between experimental treatments and control after three months. SGR has a close relation with weight gain, but it was not affected. Cha´vez-Sa´nchez *et al.* (1994) reported similar findings for Nile tilapia; growth was not affected by the 0.94mg/kg diet, but was reduced by diets containing 1.88mg AFB/kg or higher. However, these authors reported that FCR was not affected by AFB levels as high as 30mg/kg. An inverse relation between AFB concentration and growth rate of Nile tilapia was reported by El-Banna *et al.* (1992).

In conclusion, diets containing 100mg AFB/kg were not lethal to Beluga, 25, 50, 75 and 100mg/kg caused different degree of

hepatic lesions. The severity of hepatic lesions in 75 and 100ppb after two months suppressed appetite and food digestion which could affected weight gain and FCR significantly.

Aquatic species have shown dissimilar susceptibility to the hepatotoxic and carcinogenic effects of AFB₁ that depends on the particular species. The differential susceptibility seems to be correlated with interspecies variation in the biotransformation efficiency of AFB₁. Even though the problem of aflatoxosis in fish was discovered about 40 years ago, sudden outbreaks of fish mortality continue to be reported, suggesting that the problem is still misunderstood and that scant preventive measures have been adopted (Maria Pia Santacroce *et al.* 2008).

This was one of the first studies in regard to *Huso huso* and further studies are required for more details.

References

- Boonyaratpalin, M., Supamattaya, K., Verakunpiriya, V., Suprasert, D., 2001.** Effects of Aflatoxin B₁ on growth performance, blood components, immune function and histopathological changes in black tiger shrimp (*Penaeus monodon* Fabricius). *Aquaculture Research*, **32**:388–398.
- Cha´vez-Sa´nchez, M.C., Martinez Palacios, C.A. and Osorio Moreno, I., 1994.** Pathological effects of feeding young *Oreochromis niloticus* diets supplemented with different levels of Aflatoxin B₁. *Aquaculture*, **127**:49–60.

- Cagauan, A.G., Tayaban, R.H., Somga, J. and Bartolome, R.M., 2004.** Effect of aflatoxin-contaminated feeds in Nile *tilapia* (*Oreochromis niloticus* L.). In: Abstract of the 6th International Symposium on Tilapia in Aquaculture (ISTA 6) Section: Health Management and Diseases Manila, Philippines. 12–16 September.
- EI-Banna, H.M., Teleb, M.M. Hadi, and Fakhrt, F.M., 1992.** Performance and tissue residue of tilapias fed dietary aflatoxin. *Veterinary Medicine Journal*. **40**:17-23, Food and Drug Administration. 1994. Sec. 683.100—Action levels for aflatoxins in animal feeds (CPG 7126.33).
- Farabi, M., Yousefian, M. and Hajimoradloo, A., 2006.** Aflatoxiosis in juvenile fed a contaminated diet. *Journal of Applied Ichthyology*, **22**(Suppl.):234-234.
- Gallagher, E.P. and Eaton, D.L., 1995.** *In vitro* biotransformation of Aflatoxin B₁ in channel catfish liver. *Toxicological Applied Pharmacology*, **132**:82–90.
- Halver, J.E., 1969.** Aflatoxicosis and trout hepatoma. In: (L.A. Goldblatt ed.), *Aflatoxin: scientific background, control and implications*. Academic Press, New York, USA. pp.265–306.
- Jantrarotai, W. and Lovell, R.T., 1990.** Subchronic toxicity of dietary Aflatoxin B₁ to channel catfish. *Journal of Aquatic Animal Health*, **2**:248-254.
- Jantrarotai, W., Lovell, R.T., Grizzle, J.M., 1990.** Acute toxicity of Aflatoxin B₁ to channel catfish. *Journal of Aquatic Animal Health*, **2**:237-247.
- Lightner, D.V., Redman, R.M., Price, R.L. and Wiseman, M., 1982.** Histopathology of aflatoxicosis in the marine shrimp *Penaeus stylirostris* and *P. vannamei*. *Journal of Invertebrate Pathology*, **40**:279-291.
- Lovell, R.T., 1984.** Use of soybean products in aquaculture diets. *Animal nutrition research highlights*. American Soybean Association, St. Louis, Mo. USA.
- Lovell, R.T., 1989.** Nutrition and feeding of fish. Van Nostrand Reinhold, New York, USA.
- McKean, C., Tang, L., Billam, M., Tang, M., Theodorakis, C.W., Kendall, R.J., and Wang, J.S., 2006.** Comparative acute and combinative toxicity of Aflatoxin B₁ and T-2 toxin in animals and immortalized human cell lines. *Journal of Applied Toxicology*, **26**:139–147.
- Murjani, G., 2003.** Chronic aflatoxicosis in fish and its relevance to human health. Central Institute of Freshwater Aquaculture, India.
- NRC., 1993.** Nutrient requirements of fish. National Academy Press. Washington D.C.
- Ostrowski-Meissner, H.T., B.R. LeaMaster, E.O. Duerr and W.A. Walsh. 1995.** Sensitivity of the pacific white shrimp, *Penaeus vannamei*, to Aflatoxin B₁. *Aquaculture*, **131**:155–164.
- Plakas, S.M., Loveland, P.M., Bailey, G.S., Blazer, V.S. and Wilson, G.L., 1991.** Tissue deposition and excretion of ¹⁴C-labeled Aflatoxin B₁ after oral administration in channel catfish. *Food Chemistry Toxicology*, **29**:805–808.
- Reiss, J., 1972a.** Comparing investigations on the toxicity of some mycotoxins to the larvae of the brine shrimp (*Artemia salina* L.).

- Zentralblatt Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene. Erste Abteilung Originale, **155**:531–534.
- Reiss, J., 1972b.** Toxic effects of mycotoxins Aflatoxin B, Rubratoxin B, patulin and diacetoxyscirpenol on the crustacean *Cyclops fuscus*. Journal Association of Official Analytical Chemists, **55**:895–896 .
- Royes, J.B. and Yanong, R.P.E., 2002.** Molds in fish and aflatoxicosis (<http://edis.ifas.ufl.edu/FAO95>).
- Sahoo, P.K., Mukerjee, S.C., Nayak, S.K. and Dey, S., 2001.** Acute and subchronic toxicity of aflatoxin B₁ to rohu (*Labeo rohita*, Hamilton). Indian Journal of Experimental Biology, **39**:453–458.
- Sahoo, P.K., Mukherjee, S.C., Jain, A.K. and Mukherjee, A., 2003.** Histopathological and electron microscopic studies of gills and opisthonephros of rohu (*Labeo rohita*) to acute and subchronic Aflatoxin B₁ toxicity. Asian Fisheries Science, **16**:257–268.
- Santacroce, M.P., Conversano, M.C., Casalino, E., Lai O., Zizzadoro, C., Centoducati, G. and Crescenzo, G., 2008.** Aflatoxins in aquatic species: metabolism, toxicity and perspectives, Reviews in Fish Biology and Fisheries, **18**:99-130.
- Sato, S., Matsushima, T., Tanaka, N., Sugimura, T. and Takashima, F., 1973.** Hepatic tumours in the guppy (*Lebistes reticulatus*) induced by Aflatoxin B₁ dimethylnitrosamine and 2-acetyl amino-fluorene. Journal of National Cancer Institute, **50**:765–778.
- Stewart, D. and Larson, E., 2002.** Aflatoxicosis in wildlife. Information Sheet 1582. Mississippi State University. Extension Service, Cooperating with U.S. Dept. of Agriculture Steyn PS (1995) Mycotoxins, general view, chemistry and structure. International Journal of Molecular Sciences, Volume 10,82/83:843–851.
- Tacon A.G.J., 2004.** Fish meal and fish oil use in aquaculture: Global overview and prospects for substitution. In: Nutritional Biotechnology in the Feed and Food Industries, Proceedings of Alltech's 20th Annual Symposium (T.P. Lyons and K.A. Jacques, eds). Nottingham University Press, Nottingham, UK. pp.433–448.
- Tsai, H.W., 1996.** Evaluation of zebrafish (*Brachydanio rerio*) as a model for carcinogenesis. Ph.D. dissertation. Oregon State University, Corvallis. USA.
- Tuan, N.A., Grizzle, J.M., Lovell, R.T., Manning, B.B. and Rottinghaus, G.E., 2002.** Growth and hepatic lesions of Nile tilapia (*Oreochromis niloticus*) fed diets containing Aflatoxin B₁. Aquaculture, **212**:311–319.
- Wiseman, M.O., Price, R.L., Lightner, D.V. and Williams, R.R., 1982.** Toxicity of Aflatoxin B₁ to Penaeid shrimp. Applied Environmental Microbiology, **44**:1479–1481.

اثرات سطوح مختلف تغذیه ای آفلاتوکسین B₁ بر روی میزان بقا و فاکتورهای رشد فیل ماهی (*Husu huso*)

ابوالفضل سپهداری^{۱*}؛ حسینعلی ابراهیم زاده موسوی^۲؛ عیسی شریف پور^۱؛ علیرضا خسروی^۲؛ عباسعلی مطلبی^۱؛ محمود محسنی^۳؛ شاپور کاکولکی^۱؛ حمید رضا پور علی^۳ و علی حلاجیان

تاریخ دریافت: دی ۱۳۸۷ تاریخ پذیرش: بهمن ۱۳۸۷

چکیده

در این مطالعه تاثیر غلظتهای مختلف آفلاتوکسین B₁ بر روی فیل ماهی (*Husu huso*) تحت شرایط کنترل شده مورد بررسی قرار گرفت. در این بررسی تعداد ۱۸۰ عدد ماهی با میانگین وزنی ۱۰±۱۲۰ گرم و در پنج تیمار آزمایشی با جیره‌های حاوی مقادیر (۱۰۰ ppb و ۷۵ppb و ۵۰ppb و ۲۵ppb و ۰ ppb) آفلاتوکسین B₁ طی ۳ ماه مورد تغذیه قرار گرفتند. نتایج نشان داد که سطوح مختلف آفلاتوکسین B₁ تاثیر معنی داری بر روی سرعت رشد ویژه (SGR) نداشته ولی اختلاف معنی داری در ضریب تبدیل غذا (FCR) در بین گروه کنترل و تیمارهای ۷۵ و ۱۰۰ ppb بعد از ۹۰ روز را نشان داد (P<0.05). با افزایش سطح آفلاتوکسین درصد بقا ثابت بوده و هیچ تلفاتی در گروههای مختلف مشاهده نگردید (SR=100%). این مطالعه نشان داد که مقادیر مختلف آفلاتوکسین مورد استفاده در شرایط آزمایشی بر روی برخی از فاکتورهای رشد از قبیل ضریب تبدیل غذا تاثیر گذاشته در حالی که تغییرات معنی داری در بقا و سرعت رشد ویژه مشاهده نشد.

طیف گسترده‌ای از تغییرات هیستوپاتولوژی از پرخونی تا نکروز در کبد ماهیان مورد تغذیه با مقادیر مختلف آفلاتوکسین B₁ مشاهده گردید. دخیله گسترده چربی و دژنره شدن سلولهای کبدی بخصوص در مقادیر ۷۵ppb و ۱۰۰ آفلاتوکسین B₁ بعد از ۶۰ روز مشاهده گردید.

کلمات کلیدی: فیل ماهی، آفلاتوکسین B₁، ضریب تبدیل غذا، سرعت رشد ویژه، بقا

۱- موسسه تحقیقات شیلات ایران، تهران صندوق پستی: ۶۱۱۶-۱۴۱۵۵
 ۲- دانشکده دامپزشکی، دانشگاه تهران، صندوق پستی: ۶۴۵۳-۱۴۱۵۵
 ۳- انستیتو تحقیقات بین‌المللی ماهیان خاویاری دکتر دادمان، رشت صندوق پستی: ۳۴۶۴-۴۱۶۳۵

* پست الکترونیکی نویسنده مسؤل: asephadari@yahoo.com