ACTA UNIVERSITATIS AGRICULTURAE ET SILVICULTURAE MENDELIANAE BRUNENSIS

Volume LVIII 29 Number 4, 2010

IMPACTS OF DIETARY CYANOBACTERIA ON FISH

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Received: March 2, 2010

Abstract

ZIKOVÁ, A., PALÍKOVÁ, M., MAREŠ, J., NAVRÁTIL, S., KOPP, R.: *Impacts of dietary cyanobacteria on fish.* Acta univ. agric. et silvic. Mendel. Brun., 2010, LVIII, No. 4, pp. 277–284

Development of cyanobacterial water bloom became a common issue all over the world. Cyanobacteria are the most important primary producers in aquatic ecosystems but in some abundant species their secondary metabolites called cyanotoxins seem to be harmful for many animal groups especially mammals but also fish. In fishes, adverse effects have been demonstrated in several studies applying cyanotoxins by unnatural injection. However, cyanobacteria and fish coevolved during ages and therefore the question arises whether cyanobacteria might be even used for fish via oral application (fish diet). The use of cyanobacteria for fish diets is varying including applications of pure cyanobacteria biomass as well as incorporation of cyanotoxin containing cyanobacteria biomass into commercial fish diet. The impacts of cyanobacteria in fish diets administered via the oral route revealed contradictory findings ranging from moderate negative to growth promoting impacts and it seems that any bioaccumulation of microcystins can become depurated by rearing fish in clean water for a short period. According to the results obtained from various experiments, cyanobacteria as primary producers might be used as a component of fish diets especially concerning partial replacement of fish meal. However, the determination of nutrition value and the bioavailibility of nutrients present in cyanobacteria for different fish species needs to be determined. Furthermore thorough research is needed to exclude any harmful problem for the final consumers – humans.

blue-green algae; fish diet; cyanotoxins

Cyanobacteria development became recently the most discussed topic all over the world (Carmichael, 1989). Besides negative effects such as deterioration of physicochemical parameters of aquatic environment accompanied by high pH due to photosynthetic activities, oxygen depletion and bad odor from decaying cyanobacterial biomass, cyanobacteria are able to produce a wide range of bioactive compounds (Carmichael, 2001). The natural functions of these secondary metabolites of cyanobacteria, for example, in cell signaling, environmental signaling and defense against zooplanktic predators still remain unclear. These metabolites are named cyanotoxins and can be classified into three groups regarding their structure: cyclic peptides e.g. microcystins, nodularins; and alkaloids e.g. anatoxins, saxitoxins; and lipopolysaccharides that are produced by all cyanobacterial species (Sivonen and Jones, 1999). Cyanotoxins may have severe effects on vertebrates. Cyclic peptides are mainly associated with hepatotoxicity whereas alkaloids are known to be neurotoxic and lipopolysaccharides have the potential to be irritants. Microcystins, the most common cyanotoxins in freshwater systems, are a family of toxins produced primarily by the species *Microcystis aeruginosa* but also by other *Microcystis* species and other genera, namely *Anabaena*, *Planktothrix*, *Oscillatoria* and *Nostoc* (Dawson, 1998). The toxic mechanism of microcystin (MC) is the specific inhibition of protein phosphatases type 1 and 2A (Goldberg et al., 1995; MacKintosh et al., 1990). MCs are reported to have severe impacts on mammals including humans (Weng et al., 2007; Jochimsen et al., 1998).

Cyanobacteria and fish coevolved in same habitats and thus the question arises whether cyanotoxins containing cyanobacteria given via the natural exposure route as a component of fish diet might affect fish physiology e.g. growth and cause toxin accumulation in fish. Despite the fact that fishes are primarily exposed to cyanobacteria in their environment, most studies concerning effects of cyanotoxins, especially microcystin-LR (MC-LR), were performed using mammals (Ziková and Kopp, 2008). By far most of the studies in fish deal with exposure

Type of feeding (fish diet)	Fish	Duration	Observed effects (growth, further physiological parameters)	Reference
Toxic and non-toxic strains of *Microcystis aeruginosa*	**Nile tilapia** (*Oreochromis niloticus*) **silver carp** (*Hypophthalmichthys molitrix*) mean weight 5 g	2 hours of acclimatation 10–14 hours of exposure (fish deprived of food for 12 hours)	For both species, grazing on toxic *M. aeruginosa* was depressed significantly to grazing rates on the non-toxic strains ($P<0.05$). Grazing rates of *O. niloticus* were significantly higher ($P<0.05$) than those for *H. molitrix*. Very few particles ($<10^3$) of toxic *M. aeruginosa* had been ingested by both fish species compared with those of the non-toxic starins ($>10^7$). Increased opercular beat rates at fish held in non-toxic strains. No detectable levels of microcystin were present in the water (<250ng/l).	Beveridge et al. (1993)
Toxic and non-toxic strains of *Microcystis aeruginosa* (100%, 50%, 25% and 0% toxic) at particle concentrations (1×10^6 and 5×10^5 particles/ml)	**Nile tilapia** (*Oreochromis niloticus*) 10 ± 1 g	2 hours of acclimatation 10–14 hours of exposure (fish deprived of food for 12 hours)	At both concenctrations progresive decrease in grazing rate and opercular beat rates as the percentage of toxic cells increased. Water samples from tanks with toxic cells revealed detectable levels of microcystin-LR (<250ng/l). No apparent relationship between extracellular and cell-bound microcystin-LR levels.	Keshavanath et al. (1994)
Microcystis aeruginosa (PCC 7806), 400 µg MC-LR/kg body weight	**carp** (*Cyprinus carpio*)	single sublethal bolus dose, 72 hours monitoring	Damage of renal proximal tubular cells and hepatocytes was observed as early as 1 hour, followed by pathological ganges in the intestinal mucosa at approximately 12 hours postdosing. MCs-immunopositive staining was obserdved in hepatocytes and the promixal tubular cells; the staining increasing in the hepatopankreas in intensity with increasing time postdosing. The analysis of carp tissue extracts (hepatopankreas, kidney, GI tract, skeletal muscle, brain, heart, spleen and gills) demonstrated MC-LR adducts having molecular weights of 38 kDa (putatively catalytic subunit of protein phosphatases-1 and -2A) and 28 kDa.	Fischer & Dietrich (2000)
Aphanizomenon, *Microcystis*, *Daphnia*, unfed 20% of *M* per day	**roach** (*Rutilus rutilus*) 2.54 ± 0.60 g	10 days	Fish fed on cyanobacterium *Aphanizomenon* maintained liver glycogen and muscle protein concentrations. Internal energy stores of fish fed on cyanobacterium *Microcystis* were degraded. Anyway liver glycogen was higher than in starved fish.	Kamjunke et al. (2002a)
Aphanizomenon, *Microcystis*, *Daphnia*, unfed 20% of fish weight	**roach** (*Rutilus rutilus*) 2.54 ± 0.6 g	10 days	Growth rate with *Aphanizomenon* was lower than *Daphnia* but significantly higher than without food, whereas growth rate with *Microcystis* was as low as without food. Cultivation of roach faeces showed that *Microcystis* has not been digested and grew exponencially after passing trough the gut.	Kamjunke et al. (2002b)
Microcystis aeruginosa, *Arthrospira fusiformis*, *Scenedesmus quadricauda*, comercial diet (control), unfed (algae is not icluded into commercial diet – pellets)	**Nile tilapia** (*Oreochromis niloticus*)	8 weeks (*Microcystis* group-3 weeks)	Condition factor decreased in all algal fed fish groups except the one fed on *Microcystis* colonies. Saturated FA and monounsaturated FA were higher in the control vs. treatment groups (included unfed one). Fish decreased their weight during the experiment.	Tadesse et al. (2003)
Diet 1 fish food + toxic cells *Microcystis aeruginosa* (strain NPLJ-4) Diet 2 only toxic cells Diet 3 fish food + previously disrupted toxic cells (simulation of a senescent bloom)	juvenile *Tilapia rendalli* 5 ± 1 cm	**1.** 15 days +15 days only fish food-depuration period **2.** 28 days **3.** 42 days	The highest accumulation in liver (2.8 µg/g) in the second experiment. In comparison accumulation of MCs in liver (0.6 µg/g) in the first experiment was observed during the accumulation period while the highest concentration of MCs in muscle (0.05 µg/g) was obtained during the depuration period where at the same time elimination of toxin through faeces was observed. Accumulation of MCs in muscle (in all three experiments) was above the tolerable limit for human consumption.	Soares et al. (2004)

Type of feeding (fish diet)	Fish	Duration	Observed effects (growth, further physiological parameters)	Reference
Diet 1 lyophilized cyanobacterial cells of *Microcystis aeruginosa* mixed with a commercial food (pellets) Diet 2 a) crushed lyophilized cyanobacterial cells + commercial food (pellets) b) non-crushed lyophilized cyanobacterial cells + commercial food (dusted on the water surface)	male **tilapia** (*Oreochromis sp.*) 49.92 ± 9.1 g	**1.** 14 days **2.** 21 days	ACP and ALP activities changed in response to MCs in time-dependent manner. Changes were prominent in liver and kidney. The way the MCs were administred had no influence.	Molina et al. (2005)
Bloom scum (*Microcystis*) mixed with a commercial eel food powder (sticky pellets)	**loach** (*Misgurnus mizolepis*) 9.64 ± 2.27 g	28 days	Activities of antioxidant enzymes (SOD, CAT, GHX) significantly increased. Lipid peroxidation remained stable.	Li et al. (2005)
sun-dried cyanobacteria (90% *Microcystis aeruginosa*) in diet (1.19%;2.34%;3.51%;4.68%;5.85%)	**Nile tilapia** (*Oreochromis niloticus*) cca 5.64g	12 weeks	Fish fed by algae meal diets had higher SGR (except the highest content-5.85%), no significant differences in mortality and FCR. MC-LR content: liver > spleen > gall bladder > muscle.	Zhao et al. (2006a)
Diet 1 was used as control – no blue-green algae meal only soybean meal, the content of blue-green algae (90% *Microcystis aeruginosa*) in Diets 2-6 was 15.15; 29.79; 44.69; 59.58 and 74.48%, respectively.	**gibel carp** (*Carassiuus auratus gibelio*) cca 1.22g	12 weeks	Final body weight and SGR of fish fed on diet 5 were significantly lower than the control diet. Mortality increased with increase in algae meal inclusion. FCE decreased with the increase in algae meal inclusion. Fish fed on diet 6 showed the highest feeding rate. GOT activity– no significant difference among groups. GPT activity of fish fed on diets 4, 5 and 6 was significantly lower copared to control diet. Microcystins in the muscle, liver, gallbladder and spleen increased with increasing algae inclusion.	Zhao et al. (2006b)
Diet 1 with air-dried cyanobacteria from Lake Taihu Diet 2 with air-dried cyanobacteria fron Lake Dianchi	**hybrid tilapia** (*Oreochromis niloticus* × *Oreochromis aureus*) cca. 2.2g	30+30 days exposure 55 days recovery	Both cyanobacterial biomasses showed negative effects on growth, feed utilization and nutrient retention during the exposure phase.	Dong et al. (2009)
Diet 1 commercial fish diet, Diet 2 5% dried cyanobacterial biomass (90% *Microcystis aeruginosa*, 5% *M. flos-aquae*, 5% *M. ichtyoblabe*) + commercial fish diet Diet 3 20% dried cyanobacterial biomass + commercial fish diet Diet 4 20% *Arthrospira* + commercial fish diet	female **Nile tilapia** (*Oreochromis niloticus*) 30-50g	28 days	No significant changes were observed among the experimental groups concerning stress and growth.	Ziková et al. (2010)

SGR – specific growth rate; FCR – feed conversion efficiency; FA – fatty acids; FCE – feed conversion efficiency; GOT - asparate aminotransferase; GPT – alanine aminotransferase; ACP – acid phosphatase; ALP – alkaline phosphatase; MCs – microcystins;
SOD – superoxide dismutase; CAT – catalase; GPX – glutathione peroxidase; DM – dry matter; M – wet mass; FA – fatty acids; GI – gastrointestinal tract; detailed description concerning microcystins cf. Tab. II

II: *Microcystins concentrations*

MCs content in fish diet	MCs intake	MCs in target organs	Analysis	Reference
	Diet 1: 20.4 µg MCs/fish/d Diet 2: 14.6 µg MCs/fish/d Diet 3: 29.2 µg MCs/fish/d	**L**: 0.28 ± 0.19 *AP*; 0.10 ± 0.05 *DP*; **M**: 0.007 ± 0.01 *AP*; 0.012 ± 0.02 *DP* µg MCs/g **L**: 2.8 µg/g; **M**: 0.08 µg/g (highest MCs concentration) **L**: 1.7 µg/g; **M**: 0.1 µg/g (highest MCs concentration)	ELISA	Soares et al. (2004)
	control Diet 1: 60.0 µg MC-LR/fish/d	**L**: 0.086 ± 0.010; **K**: 0.018 ± 0.002; **G**: 0.293 ± 0.050 g/10 g b.w. **L**: 0.099 ± 0.015; **K**: 0.018 ± 0.003; **G**: 0.311 ± 0.030 g/10 g b.w. (after 14 d) **L**: 0.097 ± 0.019; **K**: 0.021 ± 0.006; **G**: 0.300 ± 0.040 g/10 g b.w. (after 21 d)-*CC* **L**: 0.105 ± 0.023; **K**: 0.016 ± 0.006; **G**: 0.320 ± 0.020 g/10 g b.w. (after 21 d)-*NC*	quantitative HPLC	Molina et al. (2005)
	Diet 1: 10 µg MC-RR/kg b.w.		quantitative HPLC	Li et al. (2005)
Diet 2: 1253.52 ng/g DM Diet 3: 2210.07 ng/g DM Diet 4: 3363.02 ng/g DM Diet 5: 4440.64 ng/g DM Diet 6: 5460.06 ng/g DM	Diet 2: 327.83 ± 10.99 ng/fish/d Diet 3: 630.67 ± 74.85 ng/fish/d Diet 4: 884.06 ± 83.20 ng/fish/d Diet 5: 1259.64 ± 93.05 ng/fish/d Diet 6: 1570.98 ± 75.59 ng/fish/d	**L**: 423.55 ± 123.77; **S**: 22.85 ± 4.22; **GB**: 17.18 ± 2.71 **M**: 0.77 ± 0.43 ng/g **L**: 538.44 ± 11.62; **S**: 25.58 ± 3.39; **GB**: 28.26 ± 4.58 **M**: 1.69 ± 0.26 ng/g **L**: 784.13 ± 45.23; **S**: 51.52 ± 12.42; **GB**: 36.22 ± 2.08 **M**: 2.59 ± 0.26 ng/g **L**:1596.14 ± 300.12; **S**: 53.91 ± 3.84; **GB**: 41.21 ± 4.16 **M**: 6.47 ± 2.46 ng/g **L**: 3007.33 ± 631.54; **S**: 54.6 ± 4.19; **GB**: 38.66 ± 6.38 **M**: 14.62 ± 2.54 ng/g	Monoclonal antibodies	Zhao et al. (2006a)
MC-LR equivalents Diet 2: 39.12 ng/g Diet 3: 124.14 ng/g Diet 4: 174.50 ng/g Diet 5: 203.03 ng/g Diet 6: 228.92 ng/g		**L**: 1.69 ± 0.14; **S**: 1.47 ± 0.53; **GB**: 1.79 ± 0.32 **M**: 0.019 ± 0.003 ng/g **L**: 4.14 ± 0.36; **S**: 3.09 ± 0.22; **GB**: 4.14 ± 0.91 **M**: 0.030 ± 0.002 ng/g **L**: 6.58 ± 0.67; **S**: 5.32 ± 0.99; **GB**: 3.56 ± 0.32 **M**: 0.062 ± 0.019 ng/g **L**:16.81 ± 1.56; **S**: 7.72 ± 0.77; **GB**: 6.25 ± 0.20 **M**: 0.147 ± 0.035 ng/g **L**: 40.80 ± 6.22; **S**: 8.99 ± 3.02; **GB**: 10.56 ± 0.59 **M**: 0.171 ± 0.030 ng/g MC-LR equivalents	Monoclonal antibodies	Zhao et al. (2006b)
Diet 1: 80.0 µg/g Diet 2: 410.0 µg/g			ELISA	Dong et al. (2009)
Diet 1 commercial fish diet< DL Diet 2 (MC-5%): 4.92 µg/g Diet 3 (MC-20%): 19.54 µg/g Diet 4 (Arthr-20%): < DL		L+G+M < DL L: 0.433 ± 0.421 (1d), 0.494 ± 0.362 (7d), 0.223 ± 0.069 µg/g (28d); G+M < DL L: 0.118 ± 0.069 (1d), 0.443 ± 0.445 (7d), 0.162 ± 0.153 µg/g (28d); G+M < DL L+G+M < DL	quantitative HPLC	Ziková et al. (2010)

DM – dry matter; L – liver; S – spleen; GB – gall bladder; M – muscle; K – kidney; I – intestine; G – gills; ELISA – enzyme-linked immunosorbent assay; AP – accumulation period; DP – depuration period; CC – crushed cyanobacterial cells; NC – non-crushed cyanobacterial cells; b.w. – body weight; < DL – bellow the detection limit; 1d, 7d, 28d – after one, 7 and 28 days of exposure

to MC via intraperitoneal injection, which is an artificial exposure route (Liang et al., 2007; Wang et al., 2006; Prieto et al., 2006). However, under natural conditions cyanobacteria are also ingested by fish and thus it is of special interest to investigate the impact of cyanobacteria containing fish diets on performance of fish because they are at least in part a source of natural nutrition. To our knowledge, only few papers dealing with cyanobacteria containing diets on fish performance exist but because of ecological importance to reset fish meal it is important to investigate the potential impacts of cyanobacteria biomass on fish as well as on fish consumers. Therefore this review aims to summarize what is known about the use of cyanobacteria as a component of fish diet to point out whether it might be feasible to make use of cyanobacteria in fish diets.

RESULTS AND DISCUSSION

Table I gives an overview about the papers available concerning (1) type of feeding and (2) observed effects. Microcystin analyses are summarized in table II. Cyanobacteria were used in several ways to feed fish such as dried biomass or cyanobacterial meal (Zhao et al., 2006a,b) in fish diet, pure cyanobacterial cells or lyophilized cyanobacteria used in diets (Molina et al., 2005). Various fish species in different life stages were used in cyanobacteria feeding experiments. Duration of fish exposure to cyanobacteria varied between hours and weeks.

Comparison of sensitivities to MC between the predatory rainbow trout and the omnivorous carp revealed that carp was more sensitive to orally applied toxic *Microcystis* cells. Compared to trout, the longer lasting and more thorough digestion process of the carp might lead to a greater uptake of MC via the gut epithelial cells. The pathology in carp develops rapidly and at lower toxin concentrations in comparison to the pathological events in salmonids exposed to MC, where a slower development of pathology and primarily necrotic cell death prevails (Fischer and Dietrich, 2000).

Nile tilapia (*Oreochromis niloticus*) and silver carp (*Hypophthalmichthys molitrix*) were shown to exhibit the capacity for food selection. Both prefer to feed on a non-toxic *Microcystis* strain rather than a toxic strain, thereby avoiding MC uptake (Beveridge et al., 1993; Keshavanath et al., 1994). According to Dong et al. (2009) dietary cyanobacteria from Lake Taihu and Lake Dianchi showed negative effects on growth, feed utilization and nutrient retention of hybrid tilapia during the exposure period. Fish showed recovery in growth when they were free of dietary cyanobacteria, but the clearence of microcystins in fish muscle was slow. *Tilapia rendalli* is able to accumulate MCs but the accumulation rate depends on the availability of other feeding sources besides toxic cyanobacteria (Soares et al.; 2004). Nile tilapia fed on toxic cyanobacteria was not suitable for human food (Zhao et al., 2006a) but on the other hand the accumulation of MCs in the liver of gibel carp did not reach the critical limit for human consumption of 0.04 µg/kg/day given by WHO (Zhao et al., 2006b). Further studies using Nile tilapia revealed that cyanobacteria containing diets did not exceed the critical WHO value in fish fillet detected by HPLC even after four weeks of continuous feeding (Ziková, 2008; Ziková et al., 2010). Nile tilapia seems to have rather great capacity to modify fatty acids (FA), found in algal food, into their own species specific FA patterns. Therefore, the observed influence appears to be derived from both the amount and the type of food (Tadesse et al., 2003). Low and repeated doses of MC-LR from cyanobacterial cells induce toxicity in tilapia fish although no adverse effects were detected. When tilapia were exposed to cyanobacterial cells under laboratory conditions (60.0 µg MC-LR/fish/day) the enzymatic activities of acid and alkaline phosphatases (ACP and ALP) changed in a time-dependent manner, but adapted to the toxic environment over time. At this concentration, MC-LR is only moderately toxic in tilapia fish (*Oreochromis sp.*), especially in liver and kidney and they can derive energy from alternative pathways and survive. In addition, both organs underwent histopathological changes, which could be correlated to the significant increases in ACP and ALP, the gills and gastrointestinal tract were also affected. These findings suggest that low and repeated doses of MC-LR from cyanobacterial cells induce toxicity in tilapia fish although no adverse effects were perceived (Molina et al., 2005).

According to the study of Kamjunke et al. (2002b), cyanobacterium *Aphanizomenon* can be considered as a suitable alternative food source for juvenile roach (*Rutilus rutilus)* when zooplankton food is in short supply. Growth rate with *Aphanizomenon* was significantly higher than without food. Roach were able to maintain their weight by using this cyanobacterium, which may allow them to bridge over periods when no other food is available. In contrast, *Microcystis* was obviously not a suitable food probably because of its poor digestibility (Kamjunke et al., 2002b). It was suggested that the incomplete digestion of *Microcystis* was the main reason for the negative growth rates of roach when fed on this cyanobacterium species. The *Microcystis* mucus cover protects these cyanobacteria against digestion in the intestine of roach that lacks pepsin- and acid-secreting cells (Persson, 1983). During gut passage some carbohydrates may be released from the mucus, which are absorbed by the fish, but the *Microcystis* cells are not destroyed and digested. An assimilation of *Microcystis* into muscle tissue of 0+year roach was detected. Therefore, at least some parts of this cyanobacterial species were utilized. Assimilation increased, however, with proportion of *Aphanizomenon* in cyanobacterial food indicating a more effective utilization of the filamentous cyanobacterium (Kamjunke et al., 2002a).

Li et al. (2005) suggested that antioxidant enzymes were able to eliminate oxidative stress induced by low concentrations of microcystins and to

prevent increased lipid peroxidation in the liver of loach (*Misgurnus mizolepis*). According to Tab. II. cyanotoxin concentrations were detected by ELISA or HPLC and from the findings it seems that the highest concentrations were found in liver > spleen, gall bladder, kidney > muscle.

CONCLUSIONS

Toxic effects and modes of action of hepatotoxins in fish have been investigated in detail at several levels (whole fish, eggs, organs and enzymes). In some case the reasons for species specific susceptibilities of MC remain unclear. However, these various MC sensitivities have to be investigated from an ecological point of view as well as for the potential to impact human health via the food chain. Cyanotoxins present in fish can depurate fast after putting fish into clean water (Adamovský et al., 2007). More investigations are needed to verify that all cyanotoxins and potential harmful metabolites can become depurated easily and might not bear any harm for consumers.
It is likely that at least in part cyanobacteria even containing cyanotoxins might be used for fish diets. However, the determination of digestebility, nutrition value and the bioavailibility of nutrients present in cyanobacteria for different fish species needs to be determined.

Acknowledgements

This work was supported by the National Agency for Agricultural Research (project QH71015) and by the Research Project of the Ministry of Education, Youth and Sports of the Czech Republic „Veterinary aspects of food safety and quality“ (MSM 62 15712402).

SOUHRN

Vliv sinic jako krmiva na ryby

Masivní rozvoj vodního květu sinic se stal v mnoha ohledech často diskutovaným celosvětovým tématem. Sinice jsou ve vodním prostředí sice významnými primárními producenty, mohou ale produkovat široké spektrum metabolitů s často negativním účinkem na ostatní organismy. Běžné druhy planktonních sinic jsou producenty toxických metabolitů – cyanotoxinů, které jsou toxické pro poikilotermní i homoiotermní obratlovce včetně člověka. Sinice a ryby se však vyvíjejí po mnoho let společně ve stejném prostředí bez zjevných negativních vlivů na rybí organismus. Navzdory experimentům, které potvrzují škodlivý vliv toxinů sinic na ryby aplikovaných převážně intraperitoneálně, tedy nepřirozenou cestou, pokusy s orální aplikací sinic rybám přinesly často protikladné výsledky. A to od mírné podpory růstu ryb, až po skutečnost, že microcystiny se mohou kumulovat v tkáních ryb a přenášet se v rámci potravního řetězce. Další experimenty však prokazují snížení koncentrace sinicových toxinů po přesunu ryb do čisté vody bez sinic. Na základě získaných výsledků by sinice jako primární producenti mohly být vzhledem ke svému složení využity jako komponent krmných směsí pro ryby. Tyto úvahy nabývají významu v souvislosti s hledáním náhrady za v současnosti nedostatkovou a drahou rybí moučku. Podmínkou je stanovení nutriční hodnoty použitých sinic a biodostupnost v nich obsažených živin pro jednotlivé skupiny ryb. Z pohledu spotřebitelů rybího masa je potřeba další výzkum, který prokáže, že při použití sinic obsahujících cyanotoxiny v krmivech pro ryby nemůže dojít k ohrožení zdraví konzumentů takto krmených ryb.

sinice, krmivo pro ryby, cyanotoxiny

REFERENCES

ADAMOVSKÝ, O., KOPP, R., HILSCHEROVÁ, K., BABICA, P., PALÍKOVÁ, M., PAŠKOVÁ, V., NAVRÁTIL, S., MARŠÁLEK, B. and BLÁHA, L., 2007: Microcystin kinetics (bioaccumulation and elimination) and biochemical responses in common carp (*Cyprinus carpio*) and silver carp (*Hypophthalmichthys molitrix*) exposed to toxic cyanobacterial blooms. *Environ. Toxicol. Chem.*, 26: 2687–2693.

BEVERIDGE, M. C. M., BAIRD D. J., RAHMATULLA, S. M., LAWTON, L. A., BEATTIE, K. A. and CODD, G. A., 1993: Grazing rates on toxic and non-toxic strains of cyanobacteria by *Hypophthalmichthys molitrix* and *Oreochromis niloticus*. *J. Fish Biol.*, 43: 901–907.

CARMICHAEL, W. W., 1989: Freshwater cyanobacteria (blue-green algae) toxins. In: Owenby CL, Odell GV, eds, *Natural Toxins: Characterization, Pharmacology and Therapeutics,* Pergamon Press, Oxford: pp 3–16.

CARMICHAEL, W. W., 2001: Health effects of toxin-producing cyanobacteria: "The Cyanohabs". *Hum. Ecol. Risk Assesov.*, 7: 1393–1407.

DAWSON, R. M., 1998: The toxicology of microcystins. *Toxicon*, 36 (7): 953–962.

DEMIR, N., 2007: Changes in the phytoplankton community of a costal, hyposaline lake in western Anatolia, Turkey. *Limnology*, 8: 337–342.

DONG, G., ZHU, X., HAN D., YANG, Y., SONG, L. and XIE, S., 2009: Effects of dietary cyanobacteria of two different sources on growth and recovery of hybrid tilapia (*Oreochromis niloticus x O. aureus*). *Toxicon*, 54: 208–216.

FISCHER, W. J. and DIETRICH, D. R., 2000: Pathological and biochemical characterization of microcystin-induced hepatopancreas and kidney damage in carp (*Cyprinus carpio*). *Toxicol. Appl. Pharmacol*., 164: 73–81.

GOLDBERG, J., HUANG, H., KWON, Y., GREENGARD, P., NAIRN, A.C. and KURIYAN, J., 1995: Three dimensional structure of the catalytic subunit of protein serine/threonine phosphatase-1. *Nature*, 367: 745–753.

JOCHIMSEN, E. M., CARMICHAEL, W. W., AN, J. S., CARDO, D. M., COOKSON, S. T., HOLMES, C. E., ANTUNES, M. B., DE MELO FILHO, D. A., LYRA, T. M., BARRETO, V. S., AZEVEDO, S. M. and JARVIS, W. R., 1998: Liver failure and death after exposure to microcystins at a hemodialysis center in Brazil. *N. Engl. J. Med*., 338: 873–878.

KAMJUNKE, N., MENDONCA, R., HARDEWIG, I. and MEHNER, T., 2002a: Assimilation of different cyanobacteria as food and the consequences for internal energy stores of juvenile roach. *Journal of Fish Biology*, 60: 731–738.

KAMJUNKE, N., SCHMIDT, K., PFLUGMACHER, S. and MEHNER, T., 2002b: Consumption of cyanobacteria by roach (*Rutilus rutilus*): useful or harmful to the fish? *Freshwater Biol*., 47: 243–250.

KESHAVANATH, P., BEVERIDGE, M. C. M., BAIRD D. J., LAWTON, L. A., NIMMO, A. and CODD, G. A., 1994: The functional grazing response of a phytoplankton fish *Oreochromis niloticus* to mixture of toxic and nontoxic strains of the cyanobacterium *Microcystis aeruginosa*. *J. Fish Biol*., 45: 123–129.

LI, X. Y., CHUNG, I. K., KIM, J. I. and LEE, J. A., 2005: Oral exposure to *Microcystis* increases activity-augmented antioxidant enzymes in the liver of loach (*Misgurnus mizolepis*) and has no effect on lipid peroxidation. *Comparative Biochemistry and Physiology*, 141: 292–296.

LIANG, X. F., LI, G. G., HE, S. and HUANG, Y., 2007: Transcriptional responses of alphaand rho-class glutathione-S-transferase genes in the liver of three freswater fishes intraperitoneally injected with microcystin-LR: relationship of inducible expression and tolerance. *J. Biochem. Mol. Toxicol*., 21 (5): 289–298.

MACKINTOSH, C., BESTIE, K. A., KLUMPP, S., COHEN, P. and CODD, G. A., 1990: Cyanobacterial microcystin-LR is a potent and specific inhibitor of protein phosphatases 1 and 2A from both mammals and higher plants. *FEBS Lett*., 264: 187–192.

MOLINA, R., MORENO, I., PICHARDO, S., JOS, A., MOYANO, R., MONTERDE, J. G. and CAMEÁN, A., 2005: Acid and alkaline phosphatase activities and pathological changes induced in Tilapia fish (*Oreochromis* sp.) exposed subchronically to microcystins from toxic cyanobacterial blooms under laboratory conditions. *Toxicon*, 46: 725–735.

PERSSON, L., 1983: Food consumption and the significance of detritus and algae to intraspecific competition in roach *Rutilus rutilus* in a shallow eutrophic lake. *Oikos* 41**:** 118–125.

PRIETO, A. I., JOS, Á., PICHARDO, S., MORENO, I. and CAMEÁN, A. M., 2006: Differential oxidative stress responses to microcystins LR and RR in intraperitoneally exposed tilapia fish (*Oreochromis sp*.). *Aquatic Toxicol*., 77: 314–321.

SIVONEN K. and JONES G., 1999: Cyanobacterial toxins. In: Chorus, I., Bartram, J., (eds.), *Toxic Cyanobacteria in Waters,* St. Edmundsbury Press: Suffolk, Great Britain, pp 41–111.

SOARES, R. M., MAGALHÃES, V. F. and AZEVEDO, S. M. F. O., 2004: Accumulation and depuration of microcystins (cyanobacteria hepatotoxins) in *Tilapia rendalli* (Cichlidae) under laboratory conditions. *Aquatic Toxicol*., 70: 1–10.

TADESSE, Z., BOBERG, M., SONESTEN, L. and AHLGREN, G., 2003: Effects of algal diets and temperature on the growth and fatty acid content of the cichlid fish *Oreochromis niloticus* L. – A laboratory study. *Aquatic Ecology*, 37: 169–182.

WANG, L., LIANG, X. F, LIAO, W. Q., LEI, L. M. and HAN, B. P., 2006: Structural and functional characterization of microcystin detoxification-related liver genes in a phytoplanktivorous fish, Nile tilapia (*Oreochromis niloticus*). *Comp. Biochem. Physiol*., Part C 144: 216–227.

WENG, D., LU, Y., WEI, Y., LIU, Y. and SHEN, P., 2007: The role of ROS in microcystin-LR-induced hepatocyte apoptosis and liver injury in mice. *Toxicol*., 232 (1–2): 15–23.

ZHAO, M., XIE, S., ZHU, X., YANG, Y., GAN, N. and SONG, L., 2006A: Effect of dietary cyanobacteria on growth and accumulation of microcystins in Nile tilapia (*Oreochromis niloticus*). *Aqaculture*, 261: 960–966.

ZHAO, M., XIE, S., ZHU, YANG, Y., GAN, L. and SONG, L., 2006B: Effect of inclusion of blue-green algae meal on growth and accumulation of microcystins in gibel carp (*Carassius auratus gibelio*). *J. Appl. Ichthyol*., 22: 72–78.

ZIKOVÁ, A. and KOPP, R., 2008: Impacts of microcystin, a cyanobacterial toxin, on laboratory rodents *in vivo*. *Acta Universitatis agriculturae et silviculturae Mendelianae Brunensis* 5: 263–274.

ZIKOVÁ, A., 2008: Impact of microcystin containing diets on Nile tilapia (*Oreochromis niloticus*), doctoral thesis, 115.

ZIKOVÁ, A., TRUBIROHA, A., WIEGAND, C., WUERTZ, S., RENNERT, B., PFLUGMACHER, S., KOPP, R., MAREŠ, J. and KLOAS, W., 2010: Impact of microcystin containing diets on physiological performance of Nile tilapia (*Oreochromis niloticus*) concerning stress and growth. *Environ. Toxicol. Chem*. 29: 561–568.

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