Vitenskapskomiteen for mattrygghet
Norwegian Scientific Committee for Food Safety

Opinion of the Panel on animal feed of
The Norwegian Scientific Committee for Food Safety
25 april 2005

Comments on maximum content for cadmium in fish feed

BACKGROUND

In February 2005 FEFAC (the European Feed Manufacturers Federation) submitted a document to the European Commission proposing to increase the maximum permitted content for cadmium in fish feed from 0.5 to 1.0 mg/kg (88% dry matter). The reasons given for this proposed increase were primarily related to background occurrence of cadmium in fish feed.

The Norwegian Food Safety Authority has therefore requested the Norwegian Scientific Committee for Food Safety, Panel on animal feed, to comment whether the increase in the maximum content for cadmium in fish feed proposed by FEFAC would affect fish health or pose a threat to the consumer.

TERMS OF REFERENCE

The Norwegian Food Safety Authority requests the Norwegian Scientific Committee for Food Safety, Panel on animal feed, to comment on whether increasing the maximum permitted content of cadmium in fish feed from 0.5 to 1.0 mg/kg (88 % dry matter) would affect fish health or pose a threat the consumer.

COMMENT

Toxicity in fish

General cadmium toxicity in animals and man include damage to liver and kidney, anaemia, osteoporosis, reduced growth and increased mortality. Whereas several studies have been performed on waterborne exposures of cadmium, few relevant studies exist on dietary exposures in fish. Effects of exposure in fish to elevated dietary cadmium include reduced osmoregulatory capacity, impaired digestibility, increased intestinal cell turn-over, disturbance of calcium metabolism, anaemia, and altered liver histology.

Interactions with other elements in fish

Cadmium is a well known calcium mimic, and disturbance of calcium metabolism is considered to be one of the main mechanisms of cadmium toxicity in fish (e.g Pratap et al. 1989; Hwang and Yang 1997). Dietary cadmium disturbs the calcium handling of the fish intestine. Little is known about the possible antagonistic interactions of dietary cadmium with

iron, zinc and copper in fish. However, feeding rainbow trout with elevated levels of dietary cadmium (70 mg/kg) did not affect whole body levels of copper and zinc (Mount et al. 1994).

Dietary accumulation in fish

The route of uptake (food or water) affects the organ distribution and accumulation of cadmium. The retention of dietary cadmium is low (1-5%), and organ contamination depends on the level of contamination as well as the duration of exposure (Berntssen et al. 2001; Berntssen and Lundebye 2001). Most dietary cadmium will be retained in the intestine. Of the internal organs, kidney and liver are contaminated over time while relatively little cadmium accumulates in the muscle tissue (Handy 1992; Handy 1993; Berntssen et al. 2001).

Rainbow trout fed 150 mg Cd/kg for over 1 month showed elevated levels in kidney and liver, but not in muscle tissue (Handy 1992). After exposure of dietary cadmium up to 204 mg/kg for four months the mean cadmium content in Atlantic salmon filets was 0.04 mg/kg fresh weight (Berntssen and Lundebye 2001; Berntssen et al. 2001). Atlantic salmon parr fed 7-21 mg Cd/kg feed had elevated muscle levels after 4 months of exposure, with muscle levels of 0.02 mg/kg wet weight compared to 0.01 mg/kg wet weight in control fish. No significant muscle accumulation was observed when feeding Atlantic salmon parr with 0.8 mg Cd/kg for four months (Berntssen et al. 2001). After an initial redistribution to liver and kidney (first 2-4 weeks), accumulated dietary cadmium is eliminated from trout and salmon when subsequently fed on uncontaminated feed (Handy 1992; Harrison and Klaverkamp 1989).

Dietary toxicity in fish

Dietary exposure to cadmium has low acute toxicity in fish, in contrast to waterborne exposure. For example, no mortality or other apparent signs of acute toxicity occurred in rainbow trout or Atlantic salmon fed 150 or 250 mg Cd/kg feed, respectively (Handy 1992; Berntssen and Lundebye 2001). Only levels up to 10 g Cd/kg caused some mortality in rainbow trout (Handy 1993). Adverse effects on health (such as organ damage) only become apparent after prolonged exposures.

In juvenile Atlantic salmon parr, a dietary concentration of 6.7 mg Cd/kg induced synthesis of the metal detoxifying protein metallothionein (whereas no induction was detected at 0.8 mg Cd/kg) and a level of 22 mg Cd/kg affected overall apparent energy digestibility after 4 months exposure (Berntssen et al. 2001; Berntssen and Lundebye 2001). Dietary levels of 22 mg Cd/kg affected intestinal calcium uptake mechanisms and caused mobilisation of calcium from the scales, but bone calcium and phosphorus were not affected and calcium homeostasis was maintained in Atlantic salmon parr (Berntssen et al. 2003). Exposure of dietary cadmium up to 204 mg/kg, did not affect growth or bone formation in Atlantic salmon (Berntssen and Lundebye 2001; Berntssen et al. 2001).

The non-observed effect level (NOEL) and lowest observed effect level (LOEL) for sub-chronically (four months) exposed Atlantic salmon parr can be set at 0.8 mg Cd/kg and 6.7 mg Cd/kg, respectively. The information available in the literature on the effect of exposure to dietary cadmium on fish health parameters (with respect to establishing a NOEL) on species other than salmonids is limited. Furthermore, information regarding the sensitivity of start feeders (fish fry) to dietary cadmium is lacking.

Food Safety

For Food, the current EU maximum level for cadmium in fish meat is 0.05 mg/kg or 0.1 mg/kg depending on the species. The FAO/WHO Joint Expert Committee of Food Additives

and Contaminants (JECFA) has established a provisional tolerable weekly intake (PTWI) of 7 µg Cd/kg body weight (WHO, 2001), which was recently confirmed (WHO, 2003).

CONCLUSION

Food safety

The above-mentioned studies show that relatively little cadmium accumulates in the muscle tissue. Rainbow trout fed 150 mg Cd/kg for over 1 month showed no elevated levels in muscle tissue (Handy 1992). Atlantic salmon parr fed 7-21 mg Cd/kg feed had elevated muscle levels after 4 months of exposure, with muscle levels of 0.02 mg/kg wet weight compared to 0.01 mg/kg wet weight in control fish.

No significant muscle accumulation was observed when feeding Atlantic salmon parr with 0.8 mg Cd/kg for four months (Berntssen et al. 2001). However, this was not evaluated for a life cycle production and/or slaughter size fish. Even after an exposure of dietary cadmium up to 204 mg/kg for four months the mean cadmium content in Atlantic salmon filets was 0.04 mg/kg fresh weight (Berntssen and Lundebye 2001; Berntssen et al. 2001) which is still below the maximum permitted level for cadmium, which is 0.05 mg/kg for most fish species and 0.1 mg/kg in certain fish species (commission reg. (EC) No 466/2001).

Fish health

A dietary cadmium concentration of 0.8 mg/kg did not cause an onset of sensitive biological responses (e.g. induction of detoxification proteins) following sub-chronic exposure. The risk that a dietary cadmium level of 1mg/kg will cause adverse health effects after chronic (life time) exposure is low.

A dietary cadmium concentration of 6.7 mg/kg has been shown to cause biochemical and physiological responses in Atlantic salmon parr following 4 months exposure. These responses are indicators of toxic exposure and do not necessarily indicate adverse health effects. Whether a dietary cadmium level of 6.7 mg/kg will cause adverse and irreversible effects on fish health following chronic exposure is not known. High dietary cadmium levels (up to 204 mg/kg feed) were not found to cause increased indices of bone deformities or reduced growth in Atlantic salmon parr following 4 months of exposure.

The information available in the literature on the effect of exposure to dietary cadmium on fish health parameters (with respect to establishing a NOEL) on species other than salmonids is limited. Furthermore, information regarding the sensitivity of start feeders (fish fry) to dietary cadmium is lacking.

Based on the aforementioned, increasing the maximum limit for cadmium from 0.5 to 1.0 mg/kg would not pose a risk to fish health (for Atlantic salmon parr) or pose a risk to the consumer. No information exits on the sensitivity of start feeders to dietary cadmium exposure. Furthermore, no clear no observed effect limit (NOEL) can be established for other cultured fish species such as sea bream, bass or common carp.

ASSESSED BY

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