Acute Toxicity and Bioaccumulation of Arsenic in Tilapia (*Oreochromis mossambicus*) from a Blackfoot Disease Area in Taiwan

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ABSTRACT: The general objective of our work was to determine the acute toxicity and bioaccumulation of arsenic (As) in tilapia (*Oreochromis mossambicus*) from the blackfoot disease (BFD) area in Taiwan. The average concentration of As in pond water ranged from 17.8 to 49 μg L^{-1}. Acute toxicity tests showed that the As concentration that caused toxicity to tilapia ranged from 69 060 μg As L^{-1}, in the 24-h toxicity test, to 28 680 μg As L^{-1}, in the 96-h toxicity test. We measured As concentrations in various tissues of tilapia to identify the affinities of tissues for As. Significant correlations were found among the As concentrations in all tissues. The highest bioconcentration factor (BCF) was found in the intestine (maximum value: 2270). The order of BCFs was: intestine $>$ stomach $>$ liver $\simeq$ gill $>$ muscle. Arsenic concentrations in all tissues were allometric, negatively correlating with fish body weight [$r^2 = 0.63 \pm 0.045$ (mean $\pm$ *SE*), $p < 0.05$]. Our results also revealed that As concentrations in muscle tissue were positively correlated with As accumulation in the viscera ($r^2 = 0.85$, $p < 0.05$). Significantly higher concentrations of As were obtained in the viscera of tilapia [12.65 $\pm$ 10.17 μg g^{-1} dry wt (mean $\pm$ *SD*)] than in the muscle tissue (3.55 $\pm$ 0.42 μg g^{-1} dry wt). Our results suggest that a simple way of reducing the health risk associated with consuming tilapia is to trim and cook the fish properly, that is, removing the viscera of tilapia can greatly reduce the amount of As ingested and consequently reduce the health risks. Environ Toxicol 18: 252–259, 2003.

Keywords: arsenic; acute toxicity; bioaccumulation; tilapia; blackfoot disease

INTRODUCTION

Arsenic (As) is widespread in the environment as a consequence of both anthropogenic and natural processes. It is ubiquitous but is potentially a toxic trace element. Inorganic as well as organic forms of As are present in the environment, and the former seems to be more toxic and slightly more accumulated in some freshwater aquatic species than

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TABLE I. Sampling date and size ranges of fish samples collected from fish ponds situated in BFD area in southwestern Taiwan (Fig. 1)

Sampling Date	Fish Pond	No. of Samples	Length (cm)	Weight (g wet wt)
January 1999	Y-1, Y-2	6	15.68[a] (13.55–17.01)[b]	82.41 (51.43–109.50)
August 1999	Y-1, Y-2	6	23.50 (22.31–23.78)	302.15 (255.89–310.38)
November 1999	Y-1, Y-2, Y-3, H-1, H-2	15	25.59 (24.09–26.18)	396.62 (326.80–427.31)
January 2000	Y-1, Y-2, Y-3, H-1, H-2	15	18.37 (16.89–19.48)	137.33 (104.28–165.72)

[a] Average value.
[b] Minimum and maximum values.

the latter (Spehar et al., 1980). Trivalent As may show an adverse effect on aquatic biota and is considered more toxic than the inorganic pentavalent form (Hall and Burton, 1982). Humans are exposed to As from many sources such as food, water, air, and soil. The U.S. Food and Drug Administration (FDA, 1993), in examining this food category, indicated that fish and other seafood account for 90% of total As exposure. Donohue and Abernathy (1999) reported that total As in marine fish, shellfish, and freshwater fish tissues ranged from 0.19 to 65, from 0.2 to 125.9, and from 0.007 to 1.46 $\mu g\ g^{-1}$ dry wt, respectively. Koch et al. (2001) demonstrated that total As in freshwater fish ranged from 0.28 to 3.1 $\mu g\ g^{-1}$ dry wt for whitefish (*Coregonus clupeaformis*), from 0.98 to 1.24 $\mu g\ g^{-1}$ dry wt for sucker (*Catostomus commersoni*), from 0.46 to 0.85 $\mu g\ g^{-1}$ dry wt for walleye (*Stizostedion vitreum*), and from 1.30 to 1.40 $\mu g\ g^{-1}$ dry wt for pike (*Esox lucius*).

Chen et al. (2001) indicated that long-term exposure to ingested inorganic As in artesian well water has been found to induce human blackfoot disease (BFD), a unique peripheral vascular disease that ends with dry gangrene and spontaneous amputation of affected extremities in the southwestern coastal area of Taiwan, which consists mainly of four towns, Putai, Yichu, Peimen and Hsuehchia, in Chiayi and Tainan counties. There exists a dose–response relationship between the As concentration in drinking water and the risk of BFD. Recently, a number of studies on acquired and genetic susceptibility to As have been carried out in the BFD-endemic areas of southwestern Taiwan to find out the cause of BFD (Chen et al., 2001). Nowadays, most people living in these areas do not drink water from artesian wells because tap water has been made available in this area. However, artesian well water is still used for aquaculture.

Han et al. (1998) reported that the consumption of contaminated fish and shellfish has been an important route of human exposure to trace elements (As, Cu, Zn, Pb, Cd, Hg) in Taiwan, in that oysters (*Crassostrea gigas*), tilapia, tuna, and shrimp are the most popular foods. Farming tilapia (*Oreochromis mossambicus*) is a promising practice in the BFD area because of its high market value. The fish are fed with artificial bait, which does not contain As. These fish are maintained in the ponds for at least 6 months (from April to October) before harvest. At present, data on the actual effects of As to tilapia are limited. Generally, the accumulation of metals in aquatic organisms has been linked to decreased survival and reduced reproductive ability. If As levels in pond water are higher, there may be severe effects on cultured fish, reducing their market price and leading to the closure of fish farms. Suhendrayatna et al. (2002) suggested that tilapia could be used as a bioindicator for studying the accumulation and transformation of As in freshwater organisms.

The process of accumulation of waterborne metals by fish and other aquatic animals through nondietary routes is defined as bioconcentration (Hemond and Fechner-Levy, 2000). The bioconcentration factor, which relates the concentration of metals in water to their concentration in an aquatic animal at equilibrium, is generally used to estimate the propensity to accumulate metals in the organism (Hemond and Fechner-Levy, 2000). Fish are targets for BCF assessments because of their importance as a human food source and the availability of standardized testing protocols. Measured or predicted BCFs are a requisite component for both aquacultural ecosystems and human health risk assessment (Liao et al., 2003).

The aims of the present study were to assess toxicity thresholds and to investigate As accumulation in tilapia from the BFD area in southwestern Taiwan. Specifically, the objectives were: (a) to establish the acute toxicity of As to tilapia, (b) to investigate the relationship between As accumulation in fish tissues and As in pond water, and (c) to determine the allometric relationship between As concentrations in fish tissues and fish body size.

MATERIALS AND METHODS

Sample Collection and Preparation

Details about the sampling are shown in Table I. Tilapia (*Oreochromis mossambicus*) were collected from two fish-

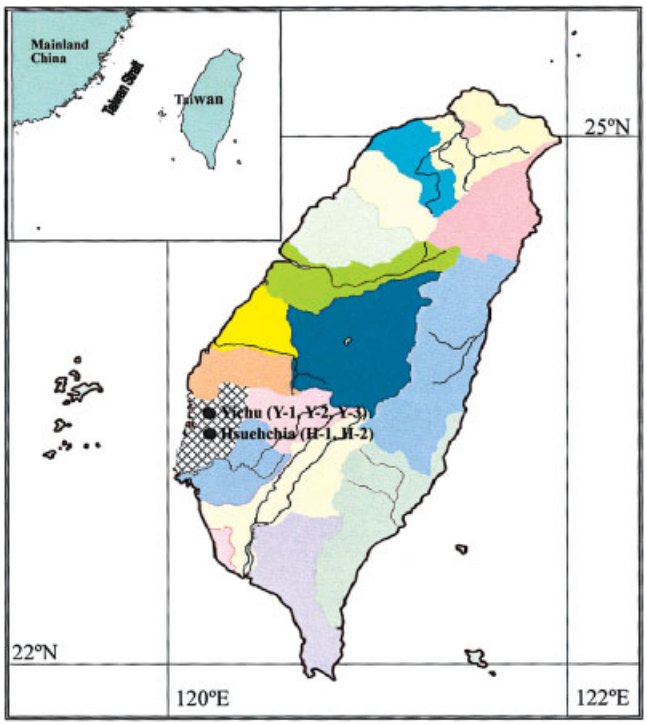

Fig. 1. Map showing the locations of sampling sites in the blackfoot disease (BFD) area (shaded area) in the southwestern Taiwan region. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]

ponds in Hsuehchia (H-1 and H-2) and three fishponds in Yichu (Y-1, Y-2, and Y-3), all of which are in the BFD area of southwestern Taiwan, in January, August, and November 1999 and in January 2000. The geographic locations of the sampling sites are given in Figure 1. After the fish were stocked in the ponds, we used nylon nets to collect the samples of stocked tilapia from the fishponds. Fish samples were kept in a 0°C cooler and were transported to the laboratory as quickly as possible. Thus, four sets of fish samples were collected from Yichu and Hsuehchia. Each time three 500-mL water samples per pond were collected. One-liter polyethylene bottles cleaned with 10% nitric acid and then rinsed with deionized water were used as containers for the collection of water samples. The collected water was filtered through a 125-μm nylon mesh to remove large suspended particles and macroinvertebrates immediately after collection and then was acidified by adding 5 mL of 1*N* HNO_3 before As analysis.

Tissue Distribution Assays

After measuring its length and weight, each fish was individually wrapped in a plastic bag and stored frozen. Dissections were performed on a clean bench on unfrozen material using a titanium knife and Teflon forceps. Adequate portions of the gill, intestine, liver, stomach, and muscle of each individual were collected. The contents of the intestine were removed. The dissected tissues samples were cleaned with deionized water, freeze-dried overnight, and then ground into fine powder in a grinder (Tai-Hsiang S36-89, Taiwan). A 500-mg portion of the powder was digested in 10 mL of concentrated HNO_3 (65% wt) overnight at room temperature. The resulting solution was evaporated and the residue redissolved in 0.1*N* HCl. Arsenic uptake results were described in terms of the bioconcentration factor (BCF). BCF was calculated from the concentration of the As accumulated in the fish (C_f, μg g^{-1}) divided by the As concentration in the pond water (C_w, μg g^{-1}) as: $BCF = C_f/C_w$.

Acute Toxicity Assays

Laboratory bioassays were conducted to determine the 24-h, 48-h, 72-h, and 96-h LC_{50} values for tilapia exposed to As. The experimental design and calculations for the acute toxicity were based on well-known procedures given by Finney (1978) and Sparks (2000). The tests were carried out in 50-L rectangular fiberglass aquaria filled with well-aerated and reconstituted dilution water (pH 7.8–8.0). The tested fish were collected from fishponds Y-1 and Y-2. Six fish of a specific size class [mean body length = 17.67 ± 1.65 cm (mean ± *SD*) and mean body weight = 148.72 ± 6.5 g] were randomly selected and transferred into each test aquarium. Dissolved oxygen in each tank was maintained at close to saturation by aeration. The temperature in each aquarium was maintained at 24.7°C ± 0.2°C using submerged heaters. The photoperiod was 16 h light:8 h dark, with an intensity of 1400 ± 100 lux. The sodium arsenite ($NaAsO_2$) stock solution was prepared with deionized water. The fish were visibly free of any deformities, lesions, or disease and were acclimated in tap water for 1 week prior to the experiment. The nominal concentrations of As tested were 0 (control), 1, 2, 4, 10, 30, 50, and 80 mg L^{-1} (Hwang and Tsai, 1993). Gross mortality of fish to each concentration was recorded every 1 h for the first 12 h and every 2 h thereafter for 96 h, with dead fish removed every 3–8 h. Tilapias were not fed throughout the test. The control and each test concentration were tested in duplicate. No mortality occurred in the controls.

The LC_{50} values were determined from maximum likelihood estimates of linear functions relating log As concentration to probit transformations of percent mortality (Finney, 1978). The LC_{50} values were determined using mean assayed As concentrations and cumulative mortality. Statistical comparisons between LC_{50} values were based on the standard error of the difference. When it became apparent that no statistically significant differences in LC_{50} values had occurred between bioassay replicates ($p > 0.05$), the replicates were pooled, and a single LC_{50} was calculated for As. Chi-square tests were performed to test the homogeneity of mortality between replicates.

Chemical and Statistical Analyses

A Perkin–Elmer Model 5100PC atomic absorption spectrometer equipped with an HGA-300 graphite furnace atomizer was used to analyze As. Analytical quality control was achieved by digesting and analyzing identical amounts of rehydrated (90% H_2O) standard reference material (dog fish muscle, DORM-2, NRC-CNRC, Canada). Recovery rate was 94.6% ± 3.6%, and the level of detection was 0.62 μg As L^{-1} for water samples and 0.05 μg As g^{-1} for tissue samples.

Curve fitting was performed using the nonlinear regression option of the Statistica® software (StatSoft, Tulsa, OK, USA). We also employed Statistica® to determine the coefficient of determination (r^2) and to perform such statistical analyses as analysis of variance and the student's t test. Statistical significance was determined if p values were less than 0.05.

RESULTS AND DISCUSSION

Acute Toxicity Study

LC_{50} values for 24 h, 48 h, 72 h, and 96 h are listed in Table II. The LC_{50} became progressively smaller as the duration of exposure increased. A limited number of studies have investigated As toxicity to tilapia. Our 96-h LC_{50}, 28.68 mg L^{-1} (95% CI: 24.92–32.44 mg L^{-1}) was close to the range of the 96-h LC_{50} of As to seawater tilapia (26.5 mg L^{-1}; 95% CI: 23.2–33.8 mg L^{-1}) yet was lower than that to freshwater tilapia (71.7 mg L^{-1}; 95% CI: 67.8–76.4 mg L^{-1}) reported by Hwang and Tsai (1993). Thus, our results indicate that tilapias reared in water from artesian wells (fishponds Y-1 and Y-2; mean length = 17.67 ± 1.65 cm) are more sensitive than the freshwater tilapias (mean length = 9.51 ± 0.81 cm) used by Hwang and Tsai (1993), which were first collected from seawater ponds in the Tainan branch of Taiwan Fisheries Research Institute and then reared in freshwater for more than 1 month.

Several studies have reported acute toxicity of As to other fish species. Our result for the 96-h LC_{50} of As to tilapia was close to the range of the 96-h LC_{50} of As to the rainbow trout *Oncorhynchus mykiss*—23–26.6 mg L^{-1} (Spehar et al., 1980), to the bluegill *Lepomis macrochirus* (29–35 mg L^{-1}), and to the stonefly (*Pteronarcys californica*)—38 mg L^{-1} (Johnson and Finley, 1980).

TABLE II. LC_{50} values (with 95% confidence intervals of arsenic to tilapia for selected time intervals in parentheses)

Time (h)	LC_{50} (mg L^{-1})
24	69.06 (65.81–72.31)
48	51.52 (48.11–54.93)
72	38.44 (34.85–42.03)
96	28.68 (24.92–32.44)

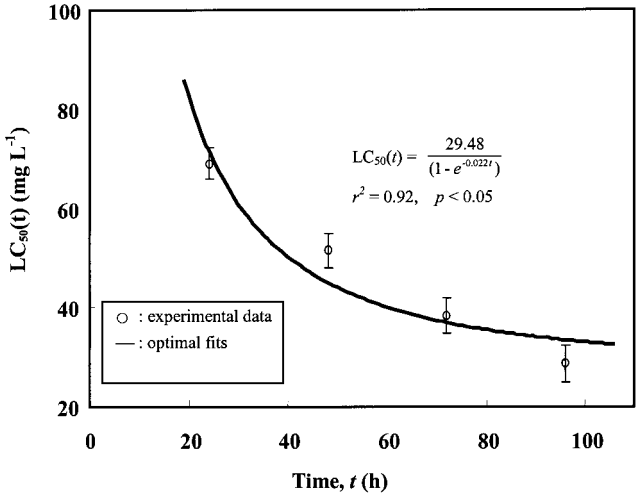

Fig. 2. Optimal fit of the acute toxicity model to the $LC_{50}(t)$ data (mean ± 95% CI) listed in Table I.

To obtain the concentration at which 50% of the test population can live for an indefinite time (incipient LC_{50}), we used an acute toxicity model presented by Liao and Lin (2001) to estimate incipient LC_{50}. The mathematical expression of the acute toxicity model can be written as: $LC_{50}(t) = LC_{50}(\infty)/(1 - e^{-k_2 t})$, where t is the exposure time, $LC_{50}(\infty)$ is the incipient LC_{50}, and k_2 is the depuration rate constant (d^{-1}). The optimal fit ($r^2 = 0.92$, $p < 0.05$) of the model to the LC_{50} values listed in Table II resulted in the incipient

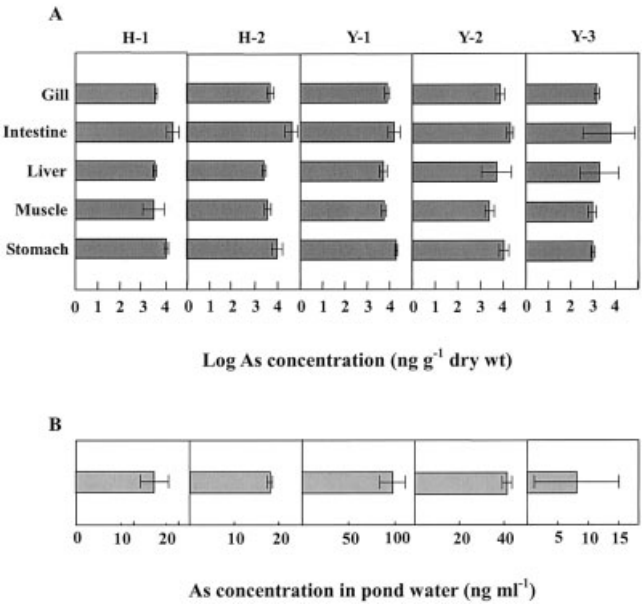

Fig. 3. Arsenic concentrations: (A) in various tissues of tilapia and (B) in pond water collected from fish farms Y-1, Y-2, Y-3, H-1, and H-2, in the BFD area. Error bars show 1 standard deviation from the mean.

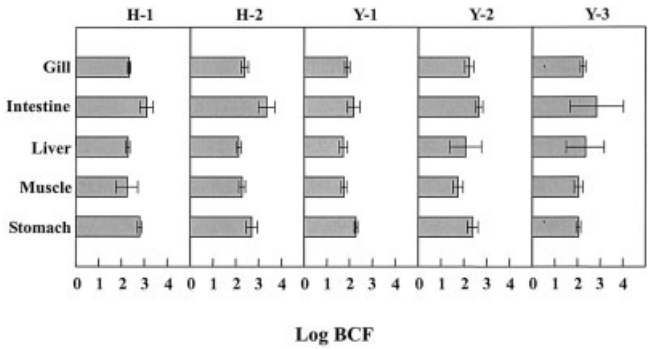

Fig. 4. Bioconcentration factors (BCFs) for arsenic for various tissues of tilapia collected from fish farms Y-1, Y-2, Y-3, H-1, and H-2, in the BFD area. Error bars show 1 standard deviation from the mean.

LC_{50} of 29.48 ± 6.9 mg L^{-1} (mean ± *SE*) at a depuration rate constant of 0.53 ± 0.19 d^{-1} (Fig. 2). Hence, if the whole-body BCF is available, the uptake rate constant (k_1 in mL g^{-1} d^{-1}) from k_2 and BCF may be estimated as: $k_1 = BCF \times k_2$.

Bioaccumulation and Tissue Distribution

Arsenic concentrations in pond water and in various tissues of tilapia from fish farms in the BFD area are shown in Figure 3. Figure 4 gives the BCF values for various tissues. The mean As pond water concentrations in Hsuehchia (H-1, H-2) and in Yichu (Y-1, Y-2, Y-3) were 17.8 ± 1.86 and 49 ± 7.49 $\mu g\ L^{-1}$ (Table III), respectively, and were below both the maximum concentration allowed in the current drinking water standard, 50 $\mu g\ L^{-1}$, and the U.S. EPA established water quality criterion for protection of aquatic biota, 100 $\mu g\ L^{-1}$ (Hellawell, 1988). However, because of increasing concerns for potential problems in aquatic-receiving systems, as have been related, current drinking water standards are under scrutiny.

Our study found that the As concentrations that were toxic to tilapia in the 24- to 96-h LC_{50} tests ranged from 69 060 $\mu g\ L^{-1}$, in the 24-h LC_{50} test, to 28 680 $\mu g\ L^{-1}$, in the 96-h LC_{50} test (Table II), indicating a low risk of toxicity to tilapia in these aquacultural ecosystems. The average As concentrations in fish tissues were 29.3, 10.9, 5.37, 5.04, and 3.55 $\mu g\ g^{-1}$ in intestine, stomach, liver, gill, and muscle, respectively (Table III). The overall mean BCF of As was found to be highest in the intestine (1394). The other BCFs, of the stomach, liver, gill, and muscle, were 421, 180, 163, and 143, respectively. Takatsu and Uchiumi (1998) and Mason et al. (2000) also showed that As concentrations are lower in muscle than in other tissues in *Tribolodon bakonensis*.

No statistically significant differences between As concentrations in fish and pond water were found ($p > 0.05$), yet these appeared to be linearly related ($r^2 = 0.5$). Schendrayatha et al. (2002) indicated that the direct accumulation of As by tilapia was proportional to the concentration of arsenicals in water. Hence, in general, As concentration in fish tissues increased with As concentration in pond water. Results of a two-way ANOVA show that BCFs differed significantly in various tissues ($F = 3.19$, $df = 123$, $p < 0.05$). No significant variations ($F = 1.20$, $df = 123$, $p > 0.05$) between As concentrations and individual fish were found.

The BCF represents the capacity for a species to accumulate a compound to an extent that is greater than the background level. In our study BCFs ranged from 2 to 3.5 log units for tilapia (Fig. 4), indicating that a process of bioconcentration occurred with As. The BCFs in our study were comparable to the ones reported by Mason et al. (2000) for small brook trout exposed to As, which had bioaccumulation factors that ranged from 3 to 3.5 log units.

Chen et al. (2001) indicated that tilapia potentially could be able to regulate the concentrations of metals in their tissues with time by combining the processes of absorption, excretion, detoxification, and storage and that this could be checked by analyzing the tissues of individuals exposed to different metals for different periods of time. In addition, the rate of metal uptake was organism specific and time dependent in fish. Our results show that more of the As was accumulated in the intestine, stomach, and liver (referred to as viscera) of fish than in the muscle. The order of tendency to accumulate As in tilapia tissues was: intestine > stomach > liver ≃ gill > muscle.

Allometry–Tissue Relationships

To study allometry–tissue relationships, the whole fish samples were divided into 10 subsamples, with each subsample containing 3 tilapias, based on the measured wet weight of four sampling sets. Two-way ANOVA analysis showed

TABLE III. Average arsenic concentrations (mean ± *SD*) in pond water ($\mu g\ L^{-1}$) and various tissues of tilapia ($\mu g\ g^{-1}$ dry wt) in Hsuehchia and Yichu fish farms in BFD area of southwestern Taiwan

	Arsenic Concentrations					
Study Site	Pond Water	Gill	Intestine	Liver	Muscle	Stomach
Hsuehchia	17.8 ± 1.86	4.28 ± 1.21	33.27 ± 14.44	3.00 ± 0.78	3.96 ± 1.56	11.11 ± 3.26
Yichu	49.0 ± 7.49	5.79 ± 3.76	25.34 ± 8.30	7.74 ± 2.73	3.13 ± 2.26	10.67 ± 5.92

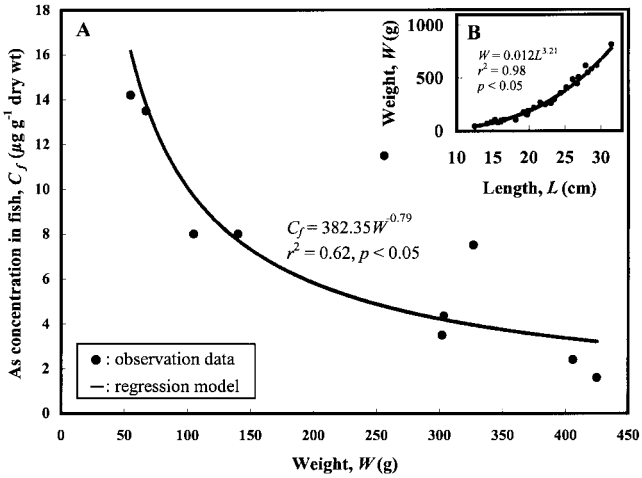

Fig. 5. Scatter plots showing (A) correlation between whole-body arsenic concentration in tilapia and body weight and (B) correlation between tilapia body weight and length.

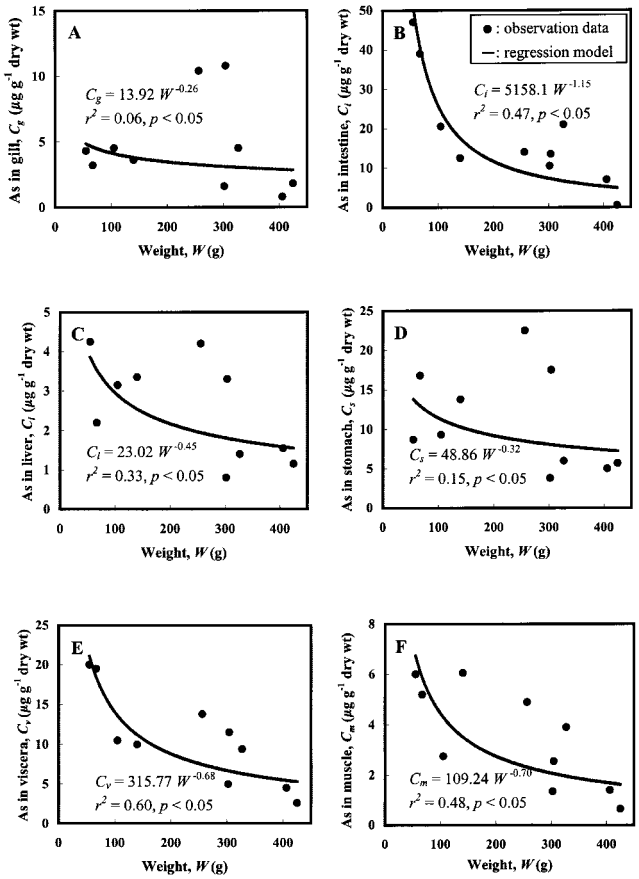

Fig. 6. Scatter plots showing relationships of body weight of tilapia to arsenic concentrations in (A) gill, (B) intestine, (C) liver, (D) stomach, (E) viscera, and (F) muscle.

significant variation between As concentration in fish and fish body weight collected from fish farms H-1, H-2, Y-1, Y-2, and Y-3 ($F = 28.99$, $df = 29$, $p < 0.05$). Significant allometric, negative correlations ($p < 0.05$) were obtained for As concentrations in various tissues with fish body weight (Figs. 5 and 6). Fish body weight and length were correlated in the tilapia ($r^2 = 0.982$, $p < 0.05$; Fig. 5(B)]. There was a significant positive correlation ($r^2 = 0.85$, $p < 0.05$) of As concentrations in fish muscle with the fish viscera (Fig. 7). Correlation analysis between As in various tissues revealed positive significant ($p < 0.05$) correlations, for most tissues ($r^2 = 0.15$–0.60).

Exponent values of the concentration-size correlations (Fig. 6) depend on several factors, such as bioavailability of As in the environment, the specific turnover rate of As in the fish, tissue type, and the competition between the opposing effects of aging and tissue growth on As accumulation within tissues. As a result, concentrations of most trace metals in tissues of small- or medium-sized fish species usually decrease with increase in fish size (Al-Yousuf et al., 2000). Our results match well with these findings; based on the relationships between As concentrations in tissues and fish weight, we have found statistically significant correlations between As content in viscera and fish weight [$r^2 = 0.60$, $p < 0.05$; Fig. 6(E)] and in muscle and fish weight ($r^2 = 0.48$, $p < 0.05$; Fig. 6(F)].

Several studies have demonstrated a significant correlation of As concentrations in tissues (muscle) with body weight in fish species: *Liza macrolepis* (Lin et al., 2001), *Mugil cephalus* (Maher et al., 1999), *A. forsteri* (Francesconi et al., 1989), *Thunnus thynnus* and *T. toggle* (Ashraf and Jaffer, 1988), *S. maculata* (Edmonds and Francesconi, 1981), *Boreogadus saida* (Bohn and McElroy, 1976), *Reinhardtius hippoglossoides* (Bohn, 1975), *Anarchichas minor* (Bohn, 1975), and *My. Scorpius* (Bohn, 1975; Bohn and Fallis, 1978). For some other species no dependence was found [e.g., *S. maculata* (Edmonds and Francesconi, 1981) and Bahama groupers (Taylor and Bright, 1973)]. Maher et al. (1999) reported that As con-

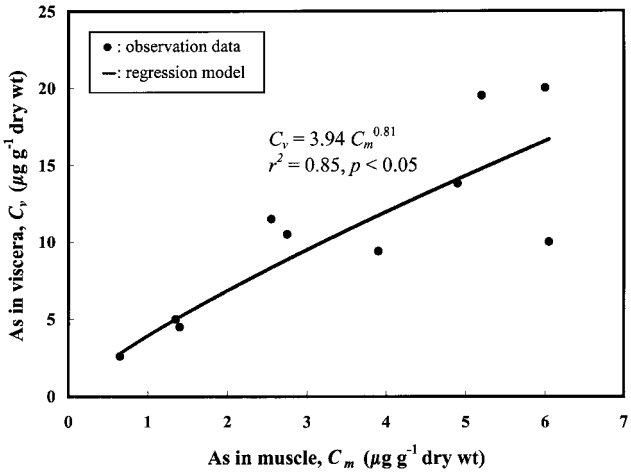

Fig. 7. Scatter plots showing relationship between arsenic concentrations in fish muscle and fish viscera.

centrations in the intestine, stomach, gill, and muscle of *M. cephalus* decreased with fish body weight ($p < 0.05$). They also pointed out that no significant differences in the As concentrations in fish were found by gender or by age.

Many laboratory and field studies have revealed that many trace metals (Zn, Cu, Cr, Ni, Hg, Cd, Pb) were accumulated in viscera than in muscle of tilapia (Kureishy and D'silva, 1993; Liang et al., 1999). The results of the present study also reveal that significantly higher concentrations of As are in the viscera than in muscle, demonstrating that viscera play a vital role in storing As in tilapia. Our results also indicate that As in viscera decreased with an increase in fish body weight. Cossa et al. (1992) wrote that higher metal concentrations in juvenile specimens are related to higher metabolic rates and insufficiently developed mechanisms for the neutralization of toxic trace metals. Furthermore, they explained, if organism growth is faster than metal accumulation, the observed trace metal concentration will decrease with age and weight, even though overall metal content may be increasing. Mackay (1991), Sijm et al. (1992), Landrum et al. (1994), and Wong et al. (1999) all reported that the processes that cause a decrease in metal concentrations in larger fish may result from a growth dilution effect, suggesting that the key factor determining metal accumulation in fish seemed to be metabolism, such as (a) metabolic rate being linked with uptake/depuration rates and (b) small fish having a more rapid short-term uptake of metals and the like.

A limited number of studies have investigated As content in the muscle of tilapia, especially that harvested from the BFD area. A comparison of the results of the present study with those of studies conducted by Han et al. (1998) in Taiwan demonstrate that mean As contents in muscle of tilapia were higher in the present study (3.13–3.96 $\mu g\ g^{-1}$ dry wt) than in the tilapia collected from supermarkets or grocery stores in Taipei city (0.13–1.45 $\mu g\ g^{-1}$ dry wt) in the Han et al. study. Our results show that As concentrations in pond water do not pose acute risks for As toxicity to cultured tilapia in selected fish farms from the BFD area of Taiwan. The highest BCF was found in the intestine. Significantly higher As concentrations were found in the viscera of tilapia than in the muscle, demonstrating that viscera play an important role in storing As in tilapia. Arsenic concentrations in the muscle of tilapia were positively correlated with As concentrations in the viscera.

These accumulation data coupled with additional acute toxicity data would provide a database to realistically pursue effective fish farm management practices and to reduce impairment from As or other metals, allowing aquaculture production to continue with minimum restraints. Of particular concern is the significant As accumulation detected in cultured tilapia harvested from the BFD area. Therefore, we cannot rule out the possibility of As contamination in tilapia because of its high BCFs in various tissues. Consequently, the consumption of cultured tilapia from the BFD area possibly poses a potential risk to human health. From the results of the present study, we suggest that to reduce the health risk associated with consuming tilapia, their viscera should be trimmed and removed, which can greatly reduce the amount of As. However, additional investigations into As content in tilapia muscle from different sources such as fish farms not in the BFD area and natural water bodies should be conducted. On the other hand, we recommend that the government set safety limits on As content in the muscle of fish. Effort is needed to update the knowledge base on the effects of consuming tilapia on human health and to determine acceptable exposure levels.

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