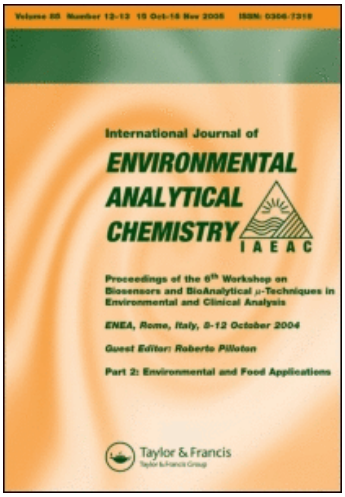

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Butyltin Residues in Fish From Australia, Papua New Guinea and the Solomon Islands

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BUTYLTIN RESIDUES IN FISH FROM AUSTRALIA, PAPUA NEW GUINEA AND THE SOLOMON ISLANDS

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Concentrations of mono- (MBT), di- (DBT) and tributyltin (TBT) were determined in the muscle and liver of fish collected from Australia, Papua New Guinea and the Solomon Islands. Butyltin concentrations ranged from below the limit of detection to 47 ng g^{-1} in muscle and 6.5 to 570 ng g^{-1} wet wt in liver. Liver was found to accumulate higher concentrations of butyltins than muscle. Butyltin residues in tissues were not positively correlated with lipid content. Monobutyltin was the predominant species in all samples. The daily dietary intake of butyltins by Australians via fish was estimated to be 377–416 ng $person^{-1}day^{-1}$, lower than is believed to cause health problems.

KEY WORDS: Butyltin, TBT, fish, dietary intake, Australia.

INTRODUCTION

Tributyltin (TBT) has been used extensively for almost 30 years in antifouling paints to prevent the accumulation of barnacles and slime on boat hulls. During the early 1980s to early 1990s, concern for the environmental impact of TBT and its degradation products, dibutyltin (DBT) and monobutyltin (MBT), grew and a number of studies of the fate and effect of butyltins in the aquatic environment were undertaken[1-4]. Several investigations have shown that TBT is toxic to non-target organisms such as molluscs (shell deformation) and gastropods (imposex) at water concentrations of a few ng l^{-1} [5,6]. Despite the high toxicity of organotin compounds to aquatic organisms, there are as yet few data on their contamination levels in fish.

Similar to other western countries, butyltin pollution in the aquatic environment and its detrimental effects on oyster culture have been documented in Australia[7-10], but not much is known about contamination levels in finfish.

Human exposure to organotin compounds as a result of consumption of contaminated seafood has only recently received attention. No known adverse human health effect due

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to eating TBT contaminated fish and shellfish has been documented so far, but organisms obtained from waters close to direct TBT input may contain concentrations that make them unsuitable for human consumption. A tentative tolerable daily intake (TDI) of 1.6 μg $kgbw^{-1}$ day^{-1} was established for TBT[11]. Recently, based on several toxicity studies, a more conservative TDI of 0.25 μg TBT $kgbw^{-1}$ day^{-1} was recommended[12]. These studies also suggested that fish are the prime sources of human exposure to organotins. Therefore, monitoring organotin residue levels in fish not only helps in understanding aquatic contamination levels and bioaccumulation but also in estimating human exposure.

The present study was conducted to determine MBT, DBT and TBT concentrations in fish muscle and liver collected from several locations in Australia and in Port Moresby, Papua New Guinea and Honiara, the Solomon Islands. Regional differences in butyltin contamination and human dietary exposures were assessed. Variations in butyltin accumulation pattern with fish feeding habits were examined.

MATERIALS AND METHODS

Muscle and liver from a range of fish species were collected in Sydney, Perth and Hobart from 28 August to 11 September, 1990. Another series of samples was collected in Brisbane, Atherton and Townsville between 26 February and 6 March, 1992. Samples from Papua New Guinea and the Solomon Islands were collected in Port Moresby and Honiara, respectively, between 31 August and 4 September, 1990. Sampling locations are shown in Figure 1. Fish samples used in this study were also analysed for organochlorine residues and the results have been reported elsewhere[13].

Fish samples consisted of the following species: rubberlip morwong (*Nemadactylus douglasii*), blue groper (*Achoerodus viridis*) and shovelnose ray (*Aptchotrema rostrata*) from near shore waters off Sydney; long-spined snapper (*Argyrops spinifer*), sea mullet (*Mugil cephalus*), spinytailed leatherjacket (*Bigener brownii*), striped seaperch (*Lutjanus vitta*), black bream (*Acanthopagrus butcheri*) and Australian herring (*Arripis georgianus*) from the western Australian coast (Rankin Platform-about 1,000 km north of Perth, Princess Royal Harbour, Albany- about 400 km southeast of Perth, Peel-Harvey Estuary and Swan River, Perth); rainbow trout (*Oncorhynchus mykiss*) and Atlantic salmon (*Salmo salar*) collected in Tyenna River and fish culture farm, respectively, near Hobart; sea mullet (*M. cephalus*), silver bream (*Acanthopagrus australis*) and mud flathead (*Platycephalus fuscus*) from Brisbane (seafood shop); silver trevally (*Caranx sexfasciatus*), stripey (*Lutjanus carponotatus*), black pomfret (*Apolectus niger*) and squid (*Loligo chinensis*) from Townsville and sea bass (*Dicentrarcus labrax*) from Atherton. Fish samples were either obtained from research institutes (Sydney), purchased in seafood shops (all cities except Sydney and Perth) or were caught specifically for this survey.

Samples collected in Port Moresby included sea mullet (*M. cephalus*), tilapia (*Tilapia nilotica*), mud crab (*Scylla serrata*) and an oyster (*Ostrea sp.*) from Koki market. Green spotted kingfish (*Caranx papuensis*), Indian mackerel (*Rastrelliger kanagurta*) and paddletail snapper (*Lutjanus gibbus*) were purchased in the local markets of Honiara. Muscle tissue from the left shoulder of each fish and the whole liver were removed and preserved in 10% formalin after dissection and stored at 4°C until analysis. The whole body of crab and the whole oyster were retained for analysis. Analysis of formalin, used for preserving the muscle, showed no butyltin residues suggesting that butyltins from tissues were not leached out by formalin preservation.

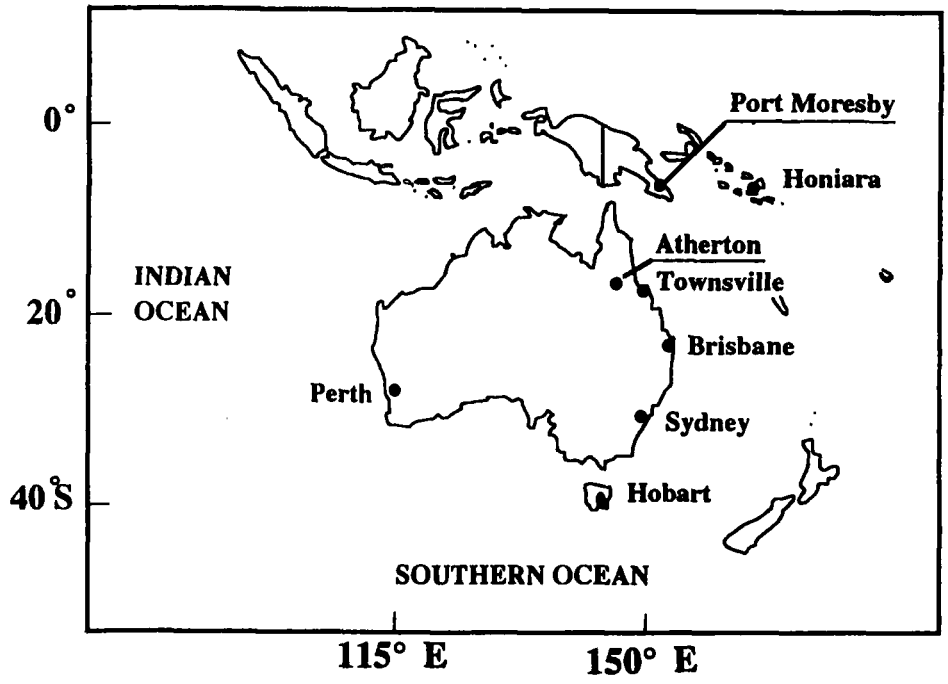

Figure 1 Map of Australia, Papua New Guinea and the Solomon Islands showing sampling locations.

Chemical analysis

The analytical method used for the determination of MBT, DBT and TBT, in fish tissue has been described elsewhere[14]. Briefly, acidified tissue samples were extracted with 0.1% tropolone-acetone and eluted through a Na_2SO_4 packed glass column to remove moisture. The concentrated extract was propylated via a Grignard reaction with *n*-propyl magnesium bromide (ca. 2 mol L^{-1} in THF solution, Tokyo Kasei Kogyo Co. Ltd., Japan). The derivatized extract was passed through a 20 g Florisil® packed dry column to remove lipids and then purified by passage through a 6 g Florisil® packed wet column. The eluate from the clean-up column was rotary evaporated to 5 ml.

Sample extracts were analysed for butyltins using capillary gas chromatography with flame photometric detection (GC-FPD). Chromatographic separation was performed on a Hewlett-Packard 5890 series II gas chromatograph with a 30 m × 0.25 mm (i.d.) DB-1 capillary column coated at 0.25 μm film thickness. The column oven temperature was programmed from 80°C (1-min hold) to 160°C at a rate of 15°C min^{-1} and then at a rate of 5°C min^{-1} to a final temperature of 260°C with a 5-min final hold time. Injector and detector temperatures were held at 200°C and 270°C, respectively. The flame photometer was operated using a hydrogen-air-nitrogen flame and was equipped with a 610 nm bandpass filter that is selective for tin-containing compounds. Butyltin trichloride, dibutyltin dichloride and tributyltin chloride of known amounts (0.1 μg) were spiked into the muscle of cod (*Gadus morhua*), caught off southern Japan, containing butyltins below the limit of detection, passed through the whole analytical procedure and used as an external standard. Only freshly derivatized external standards prepared along with every set of four samples were used to estimate concentrations. Concentrations were

estimated by comparing peak heights of butyltins in samples with those in external standards. A procedural blank was run with every set of four samples to check for interfering compounds and to correct sample values, if necessary. Monobutyltin was present at trace levels in procedural blanks. The values obtained for MBT in fish were, therefore, corrected for reagent blank concentrations. Detection limits of butyltins in samples were assigned twice the values of procedural blanks. Respective detection limits of MBT, DBT and TBT in fish muscle tissues were 4.0, 0.36 and 0.13 ng g^{-1} wet wt.

RESULTS AND DISCUSSION

Mean and range of concentrations of butyltin residues in samples of fish muscle and liver from Australia, Papua New Guinea and the Solomon Islands are shown in Table 1. For the Australian samples the total butyltin concentration ranged from below the limit of detection to 47 ng g^{-1} (mean: 20; n = 20) in muscle and 6.5 to 570 ng g^{-1} (mean: 150; n = 14) in liver on a wet weight basis. Butyltin concentrations in most liver samples were up to an order of magnitude higher than in muscle suggesting preferential accumulation of these compounds. This agrees with the finding of Suzuki *et al.*[15] who found higher concentrations of butyltin in liver than in muscle of fish from Japanese markets. Previously, fish exposed to different water concentrations of butyltins were found to accumulate higher concentrations of butyltins in liver[16]. TBT was shown to concentrate in the peritoneal fat, kidney and liver of rainbow trout (*Salmo gairdneri*) following exposure in tank experiments[17]. This study also found exceptions to the higher liver concentrations as the concentration of butyltins in muscle of black bream (Swan River, Perth), rainbow trout (Tyenna River, Hobart) and silver bream (from seafood shop in Brisbane), was higher than that in liver. The higher concentration of butyltin in muscle was contributed mainly by MBT.

The presence of butyltin in fish taken from various locations, including seafood shops, fish culture farms, rivers and harbors suggests the widespread occurrence of butyltin compounds in Australia. This is despite a ban on the sale and usage of TBT-based antifouling paints on boats under 25 m length being instituted in Australia in 1989. As the majority of Australia's population resides on the coastal strip, disposal of domestic and industrial effluents into coastal waters[18] may contribute significantly to butyltin contamination. More specifically, it was recently reported that the discharge of sewage and industrial outfalls is one of the major sources of pollutants, including organochlorines, in Australian coastal waters[19]. Wastewater may not be the only problem as, a sample of Atlantic salmon (*S. salar*) obtained from a fish farm near Hobart also contained butyltin (16 ng g^{-1} in muscle and 50 ng g^{-1} in liver) suggesting contamination from TBT-treated nets used in salmon farms. TBT compounds are widely used in various parts of the world as a sea pen antifoulant in the salmon aquaculture industry.

Concentrations of butyltin residues in muscle of four fish from Papua New Guinea ranged from below the limit of detection to 9.0 ng g^{-1} (Table 1). A liver sample of tilapia (*Tilapia nilotica*) contained 38 ng g^{-1} on a wet weight basis. Samples of three fish from the Solomon Islands contained a mean value of 2.7 ng g^{-1} butyltins in muscle (range: < 2.5–4.2) and 160 ng g^{-1} in liver (range: 89–220 ng g^{-1}) (Table 1). The liver of greenspotted kingfish (*Caranx papuensis*) collected in Honiara markets contained considerable concentrations (220 ng g^{-1}) of butyltins. Monobutyltin was the major species both in liver and muscle of fish from Papua New Guinea and the Solomon Islands (Table 1), suggesting that major sources of butyltin contamination in fish in these countries are from the disposal of municipal sewage into inland and coastal aquatic environment. The concentration of butyltin in the muscle of the fish from Papua New

Table 1 Mean and range of butyltin concentrations (ng g^{-1} wet wt) in fish muscle and liver from Oceanian countries.

Country	*Tissue*	*n*	*MBT*	*DBT*	*TBT*	*ΣBT*
Australia	Muscle	20	16 (< 4.0–42)	1.4 (< 0.36–3.1)	2.8 (< 0.13–13)	20 (ND-47)
	Liver	14	130 (< 4.0–470)	5.8 (0.42–24)	13 (1.2–72)	150 (6.5–570)
Papua New Guinea	Muscle	4	< 4.0 (< 4.0–8.0)	0.38 (< 0.36–0.98)	< 0.13 (< 0.13–0.15)	2.5 (ND-9.0)
	Liver	1	33	3.1	1.6	38
The Solomon Islands	Muscle	3	< 4.0 (< 4.0)	< 0.36 (< 0.36–0.4)	0.53 (0.2–1.0)	2.7 (< 2.5–4.2)
	Liver	2	130 (77–180)	14 (5.3–22)	13 (6.5–20)	160 (89–220)

Figures in parentheses indicate the range.
Concentrations below the detection limit were assigned half its value to calculate mean.
Samples with all the three butyltin concentrations below the detection limit were assigned ΣBT of ND (not detected).

Guinea and the Solomon Islands was lower than from Australia although this may have been a function of sample size.

The presence of organotins in tissues of fish did not appear to be related to lipid content (Figure 2), suggesting that some other mechanism regulates storage. Lipophilicity has traditionally been estimated by octanol-water partition coefficients (K_{ow}), and the log K_{ow} values for TBT, DBT and MBT are 2.2, 0.05 and 0.09, respectively[17]. These values are lower than those for highly lipophilic compounds such as organochlorine pesticides, which have the log K_{ow} values around 6.0[20]. Therefore, for future biological monitoring studies of butyltins, it is essential to identify suitable species and tissues, and whether there are species-specific patterns of organotin distribution within tissue.

The average composition of MBT, DBT and TBT relative to total butyltin concentration in fish muscle and liver is shown in Figure 3. Monobutyltin constituted > 80% of ΣBT, followed by TBT and DBT. Horseshoe crabs (*Tachypleus tridentatus*) collected from Japanese coastal waters also contained a high proportion (50–80%) of MBT[21]. A much higher composition of MBT to other butyltin compounds was observed in mussels from the Sado Estuary, Portugal[22]. These results suggest that TBT is degradable in the aquatic environment and biota. It has been suggested that degradation of TBT to MBT can occurr during storage of samples. However, mussel tissues stored at 4°C for over one year did not show any reduction in the total butyltin concentration, while the composition of TBT decreased with a concomitant increase (proportionally) in MBT[23]. Based on this information, it would appear that the concentration of total butyltin in Australian fish has not changed by long term storage, but degradation of TBT has increased the composition of MBT during storage. In addition, increased inputs of MBT from the effluent of municipal sewage treatment plants may also elevate the proportion of this compound in fish.

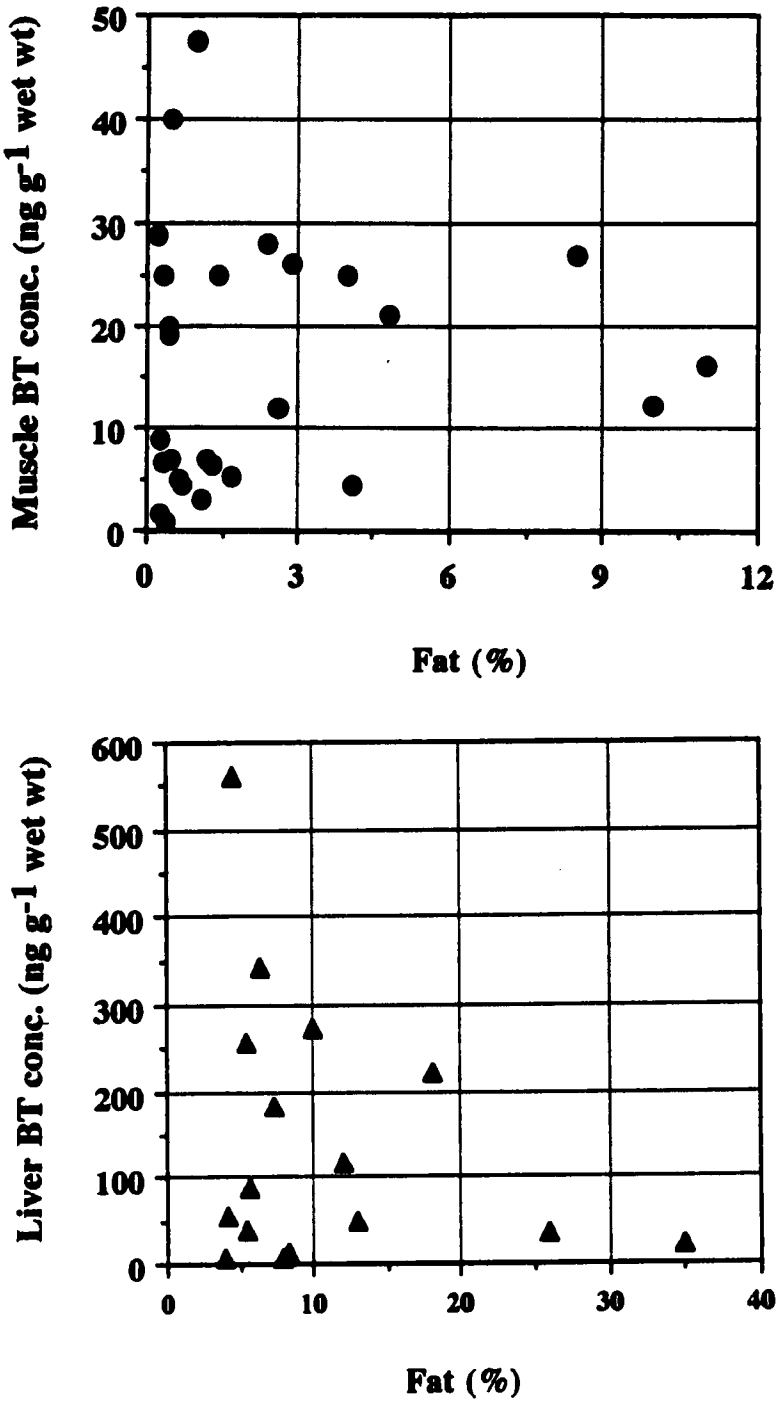

Figure 2 Relationship of butyltin concentrations (MBT + DBT + TBT) in fish muscle and liver to their fat content.

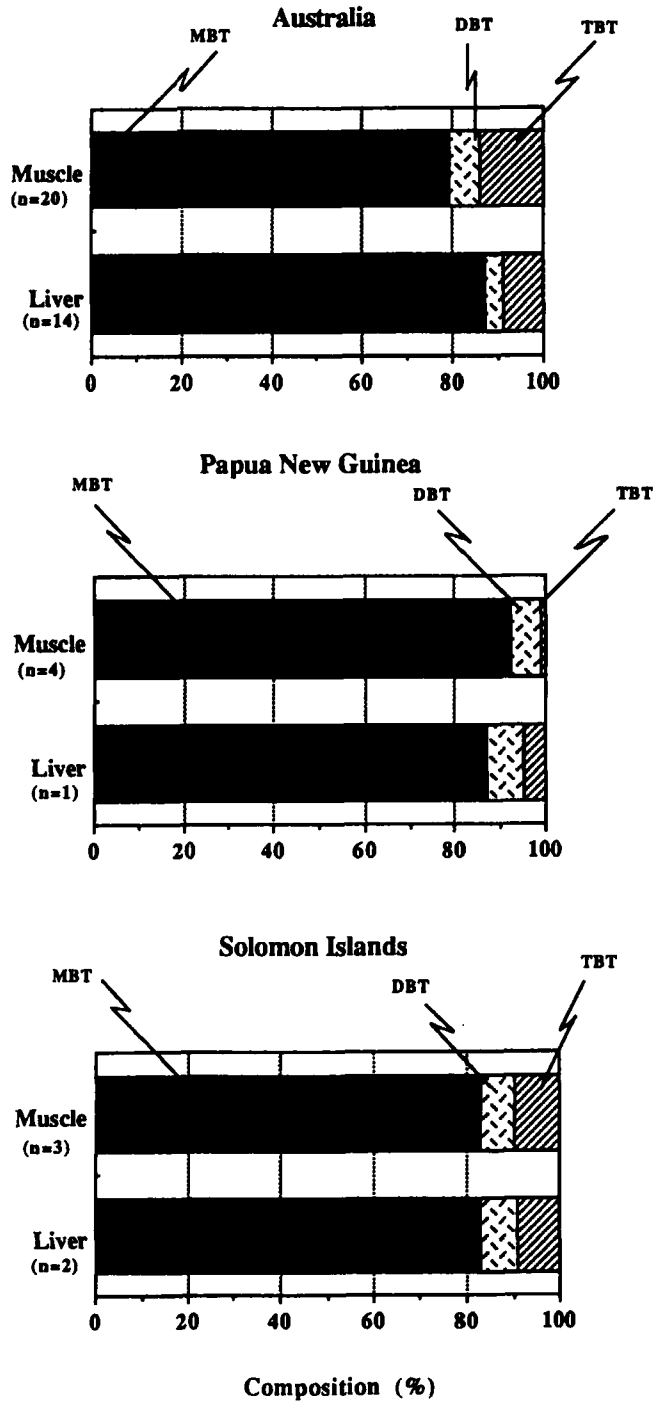

Figure 3 Average composition of butyltin compounds in fish muscle and liver from Australia, Papua New Guinea and the Solomon Islands.

Table 2 Regional differences in butyltin concentrations (ng g^{-1} wet wt) in fish muscle and liver from Australia, Papua New Guinea and the Solomon Islands.

Region	*n*	*Tissue*	*MBT*	*DBT*	*TBT*	*∑BT*
Western Australia	7	Muscle	17 (< 4.0–24)	1.3 (0.68–2.4)	1.1 (0.21–3.4)	19 (4.5–27)
	4	Liver	25 (4.7–52)	1.2 (0.42–1.5)	5.4 (1.5–16)	32 (12–56)
Southeast Australia	5	Muscle	18 (5.0–42)	1.1 (0.8–2.2)	3.4 (0.49–6.2)	22 (6.5–47)
	5	Liver	110 (4.0–310)	5.7 (0.7–9.7)	9.5 (1.5–23)	130 (6.5–340)
Northeast Australia	8	Muscle	14 (< 4.0–26)	1.7 (< 0.36–3.1)	3.8 (< 0.13–13)	20 (ND-40)
	5	Liver	220 (5.6–470)	9.5 (0.6–24)	22 (1.2–72)	250 (7.3–570)
PNG, SI	7	Muscle	< 4.0 (<4.0–8.0)	< 0.36 (<0.36–0.98)	0.28 (<0.13–1.0)	2.5 (ND-9.0)
	3	Liver	97 (33–180)	10 (3.1–22)	9.4 (1.6–20)	120 (38–220)

Southeast Australia comprises Sydney and Hobart; Northeast Australia comprises Brisbane, Townsville and Atherton; Western Australia includes Rankin Platform, Princess Royal Harbour-Albany, Peel-Harvey Estuary and Swan River-Perth.
Values below the detection limit were assigned half of its value to calculate mean.
Samples with all the three butyltin concentrations below the detection limit were assigned ∑BT of ND (not detected).
Figures in parentheses indicate the range.

There were comparable levels of butyltins in muscle taken from several Australian locations (Table 2); but the concentrations in liver varied according to the region, with the highest values recorded in Queensland (comprising samples from Brisbane, Townsville and Atherton) and the lowest in Western Australia (Perth). While the concentrations of butyltins in fish muscle from Papua New Guinea and the Solomon Islands were very low, the liver contained concentrations comparable to those observed for the southeast Australian region (comprising Sydney and Tasmania samples), the reason for this is unknown.

As feeding habits of the species analysed in different locations may also influence the magnitude of contamination, we compared butyltin concentrations in the muscle of water column (n = 5) and bottom feeding animals (n = 15) (Table 3). Most fish analysed in this study were bottom feeders, except for stripey, Australian herring, silver trevally, black pomfret and squid which feed near surface or mid-bottom waters. No considerable variation was observed between two feeding groups, even though bottom feeders were expected to contain much higher levels of TBT from sediments. Studies have shown that TBT is mainly associated with particulate matter and high concentrations are therefore liable to be found in areas of high sedimentation[24,25]. Sediments act as both a sink and a potential source of TBT to the aquatic environment[22,24–26]. In contrast to TBT, MBT has been shown to be predominant in the water column, particularly in heavily polluted or near sewage disposal sites[27,28]. As MBT was the major contributor of ∑BT in all fish species (Figure 3), water column concentration of MBT might have been the major source resulting in comparable values in water column and bottom feeders. Also, TBT

Table 3 Comparison of butyltin concentrations ranges (ng g^{-1} wet wt) in the muscle of water column feeding and bottom feeding fish collected from Australia, Papua New Guinea and the Solomon Islands.

Feeding type	*n*	*MBT*	*DBT*	*TBT*	*ΣBT*
Water column feeder	5	< 4.0–26	< 0.36–2.7	< 0.13–11	ND-40
Bottom feeder	15	< 4.0–42	< 0.36–3.1	< 0.13–13	ND-47

Samples with all the three butyltin concentrations below the detection limit were assigned ΣBT of ND (not detected).

degrades directly to MBT in surficial sediments[27,29]. MBT formed by the degradation of TBT in sediments may be redistributed to the water column until an equilibrium is reached between particulate and dissolved concentrations. These phenomena may explain why the concentrations of butyltins in both surface and bottom feeding fish were comparable. Specific experiments organised around one or two species with large sample size may be necessary to identify differences between surface and bottom feeders.

The presence of butyltins in fish may have implications for human dietary exposure. The estimated average daily intake of butyltins via consumption of fish muscle by Australians at age 15, and between the ages of 25–34 as well as the average age are presented in Table 4. These estimates are based on the daily consumption of fish per capita during 1986–1988[30]. Dietary intakes were estimated by multiplying butyltin concentration in fish by the amount consumed. The dietary intake of butyltin was in the range of 337–416 ng $person^{-1}day^{-1}$, an order of magnitude lower than the range estimated for several organochlorine pesticides[13]. A tolerable daily intake (TDI) of 15 μg TBT $person^{-1}day^{-1}$ for a person weighing 60 kg has been set out based on reduction of immune function[12]. Based on the small number of samples of fish used in this study, the estimated intake values are much lower than the tolerable limits, and butyltin contamination of fish does not appear to pose a threat to human health in Australia. Nevertheless, human exposure to butyltins via consumption of meat, dairy and farm products is not clearly understood. Further, and detailed studies are needed in this regard. It is worth mentioning that cooking is ineffective in eliminating butyltin concentrations in food[31].

It can be inferred from the present study that butyltin compounds are widely distributed in fish muscle and liver collected from Australia, Papua New Guinea and the

Table 4 Estimated average daily intake of butyltin compounds by Australians at different age groups (ng $person^{-1}day^{-1}$) via fish muscle consumption. MBT, DBT and TBT levels are shown in brackets, respectively.

Age group (yr)	*Fish consumption ($g\ day^{-1}$)*	*ΣBT intake ($ng\ person^{-1}day^{-1}$)*
15	17	337 (265, 24, 48)
25–34	18	356 (281, 25, 50)
Average	21	416 (328, 29, 59)

Solomon Islands. The concentrations were higher in liver than in muscle. The high proportion of MBT in fish muscle and liver implies degradation of TBT in fish as well as the direct input of MBT from municipal sewage treatment plant effluents. Domestic sewage may be considered as one of the main sources of butyltins in recent years. Monitoring butyltins in domestic and industrial effluents may provide information on the amount of butyltins discharged into water bodies. On the basis of a preliminary assessment, the average dietary intake of butyltins in Australia was considerably lower than that of the established tolerable daily intake.

It is worth mentioning that a TBT level of 3.5 mg l^{-1} in Port Phillip Bay in Australia is the highest value reported so far for sea water[32]. Concentrations in waters within enclosed marinas in this bay were in the ranges of 26–1,250 ng l^{-1} and more open waters of the bay exhibited concentrations typical of those found in areas where boating activity occurs (1–27 ng l^{-1}). Similarly, elevated concentrations of TBT were found in sediments of enclosed marinas (750–2,360 ng g^{-1} wet wt) in Port Phillip Bay[33]. These values are extremely high considering the toxic effects exerted by TBT at few ng/l on bivalve mollusks. Analysis of butyltin residues in fish from such highly polluted areas may provide information pertaining to toxic effects of TBT on fish under field conditions.

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