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Hepatic Butyltin Concentrations in Beluga Whales (*Delphinapterus leucas*) from the St Lawrence Estuary and Northern Quebec, Canada

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Butyltin (tributyltin TBT; dibutyltin DBT and monobutyltin MBT) speciation was measured in the liver of beluga whales from the St Lawrence Estuary and Hudson Strait (northern Quebec). Using GC–MS, liver samples were analysed from 21 beluga whales found dead, stranded along the shores of the St Lawrence during the period 1995–1998. In all cases, including a neonate specimen, the liver was contaminated with butyltin compounds with concentrations in the range 0.04–2.1 mg Sn kg^{-1} on a dry weight basis. Liver samples of five beluga whales from Hudson Strait obtained in the summer of 1998 were also analysed. For these animals, hepatic butyltin concentrations were consistently below the detection limit (<0.5 ng Sn g^{-1} for MBT and <0.2 ng Sn g^{-1} for DBT and TBT). Compared with published data on the contamination by TBT of the marine mammals of the St Lawrence in 1988, these contemporary results clearly indicate that the level of contamination of the beluga whales in this coastal marine ecosystem has not decreased ten years after regulating the use of TBT-based antifoulants on small craft.

Keywords: antifouling substances; bioaccumulation; chemical speciation; mammals; pollution effects; tributyltin (TBT)

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INTRODUCTION

Widespread usage of tributyltin (TBT) as an antifouling agent in boat paint and in the plastics industry has led to marked contamination of freshwater and marine coastal ecosystems.[1,2] TBT is very toxic to aquatic organisms, having been described as the most poisonous compound ever introduced deliberately by man into the aquatic environment.[3] As is the case in many developed countries, Canada has regulated the use of organotin-based paints (since 1989); their application is prohibited on boats longer than 25 m. However, recent studies have revealed persistent TBT contamination of marine waters in Canada.[4] TBT contamination has been recorded in sediments and mussels from the St Lawrence Estuary[5] and the west coast of Canada.[6]

The bioaccumulation potential of TBT by higher aquatic trophic organisms had been considered to be low until a research group from Japan recently

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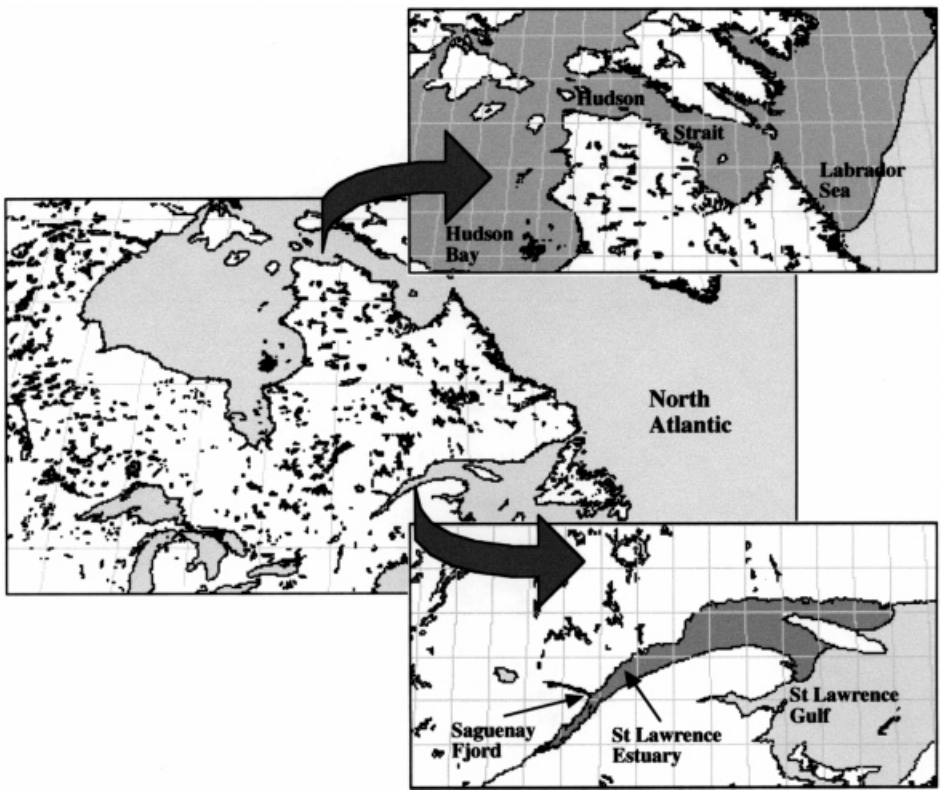

Figure 1 Sampling locations. Darkers shaded zones indicate the distribution areas of the beluga whale.

reported contamination by organotin compounds of cetaceans and pinnipeds in various regions of the world. The highest concentrations, as measured in the liver of the animals, were observed in coastal species.[7] Similar results were found in ten-year-old liver samples from beluga whales found stranded along the coasts of the St Lawrence Estuary before the use of TBT-based paints was regulated in Canada.[8] The potential adverse effects of TBT, such as immunosuppression and hepatic toxicity,[9] on marine mammals can only be inferred from toxicological studies conducted with laboratory rodents.

The unexpected finding of ubiquitous TBT contamination in marine mammals prompted this investigation of some cetaceans of the St Lawrence Estuary and Hudson Strait. The beluga whale population of the St. Lawrence estuary is considered to be permanently resident.[10] Thus, these animals are continuously exposed to contaminants derived from the heavy industrial activities along the St Lawrence River and the Great Lakes.[11] In addition, the St Lawrence River is a major shipping route between North America and the rest of the world; the commercial traffic averages 6 500 large vessels per year.[12]

There is an emerging concern about TBT contamination in remote marine coastal environments. The beluga whales living in Canadian Arctic waters are clearly less exposed to TBT originating from shipping activities than those animals in the St Lawrence Estuary. However, studies by Tanabe and co-workers have revealed TBT contamination in Dall's porpoises collected as far away as the Aleutian Chain in the North Pacific Ocean.[7] Marine mammals are an important food resource for the human inhabitants in these Arctic regions. Hence, a question arises regarding the risk posed to human consumers, particularly given that the presence of butyltin compounds in human tissues has recently been reported.[13]

The objective of this investigation of beluga whales was to evaluate the hepatic contamination of butyltin compounds, generally derived from the

antifouling biocide TBT. Two contrasting study sites were chosen. Firstly, animals from the St Lawrence Estuary were examined to evaluate current TBT contamination in resident beluga whales and consequently the efficacy of Canadian TBT regulations. Secondly, samples were collected from northern Quebec in anticipation of this being a pristine location suitable to serve as a baseline site.

MATERIALS AND METHODS

Sampling

Liver samples from 21 beluga whales found stranded along the shores of the St Lawrence Estuary (Fig. 1) between 1995 and 1998 were provided by University of Montreal's Faculté de Médecine Vétérinaire. In the summer of 1998, five adult beluga whales were collected from the Hudson Strait (Fig. 1). Liver, skin (*muktuk*) and meat samples were collected from each beluga whale (*ca* 100 g per organ). Sample collection was conducted by the local Hunting, Fishing and Trapping Association of Kangiqsujuaq. Total body length and colour have been recorded and a tooth was collected for use in estimating the age of the animal. Samples were sent frozen to the Nunavik Research Centre for storage until they were shipped to the Department of Oceanography at the Université du Québec à Rimouski for chemical analysis.

Age determination

The age of the whales was determined by counting dentine growth layers in longitudinal sections of teeth, adopting the standard of two growth layer groups per year.[14]

Chemical analysis

The analytical method was based on the derivatization of extracted organotin compounds into volatile ethylated species for their quantification and identification by GC–MS.[15] A sample of liver (5 g, wet weight) was finely divided with a surgical scalpel on an acid-cleaned watchglass. A subsample (1 g), was suspended in 5 ml of glacial acetic acid in a 20-ml Teflon tube for the extraction of the cationic organotin species. Tetrapentyltin (100 ng) was added to the suspension to serve as a surrogate standard. After a one-hour sonication with periodic agitation at room temperature, 10 ml of a hexane/tropolone (0.1%) solution was added to the acidic solution. Following a 90-min agitation on a wrist-shaker, the tube was centrifuged until phase separation was achieved.

The organic layer was removed and reduced to a volume of 5 ml under a gentle stream of nitrogen at room temperature. This step was performed to yield a small volume of hexane to elute on the LC-Si cartridge. The cationic organotin species were derivatized by adding 2 ml of an acetate buffer to the organic extract, followed by 0.5 ml of an aqueous solution of sodium tetraethylborate (2%, w/v). As the performance of this analytical protocol is greatly dependent on the quality of the sodium tetraethylborate, it was imperative to store this chemical under vacuum in a desiccator and to prepare a fresh solution of tetraethylborate just before it was used for the derivatization step.

After a 15-min agitation on the wrist-shaker, the test-tube was centrifuged for 2 min. The organic layer was removed and cleaned on a silica-gel cartridge, with an elution volume of 2 ml hexane. The cartridge was prepared as follows: a suspension of silica gel 60 (70–230-mesh; EM Separations Technology) in hexane was poured into an 8-ml acid-cleaned glass SPE column (Bakerbound; J.T. Baker). Hand-made acid-cleaned glass-wool plugs were used instead of manufactured PTFE frits, which tend to adsorb more than 50% of the standards of organotin compounds added to an organic extract.

The volume of the sample extract was reduced to 200 μl under a gentle stream of nitrogen at room temperature. The ethylated butyltin species were detected and quantified on a Varian 3400 gas chromatograph coupled to a Finnigan ion trap detector via a transfer line maintained at 250 °C. The carrier gas was helium at a flow rate of 2 ml min^{-1} and the butyltin compounds were separated on a J&W Scientific DB-5MS capillary column (0.25 mm × 30 m). The GC–ITD parameters were: sample volume, 1 μl; injection temperature, 240 °C; initial oven temperature, 70 °C for 2 min followed by a heating rate of 15 °C min^{-1} up to the final temperature of 250 °C, held for 10 min. The ion trap detector was operated in the full scan mode over the range of 100–400 Da with the automatic gain control on, and using a scan rate of 1 scan s^{-1}.

Sample preparation

Table 1 shows the results for the repeated extraction

Table 1 Standard deviation for the replicate analysis of a homogenized liver sample (no. 9509) and a non-homogenized liver sample (no. 9507)

	Concentration (ng Sn g^{-1} dry wt)		
Sample	MBT	DBT	TBT
9509-a	6	757	104
9509-b	5	737	131
9509-c	68	710	142
Mean	—	735 ± 19	126 ± 29
9507-a	2	206	122
9507-b	—	152	172
9507-c	1	39	40
Mean	—	132 ± 85	111 ± 66

of butyltin species from the liver of a beluga whale. The concentrations of butyltin species are expressed as tin (ng Sn per g of dry tissue). From these results, the analytical reproducibility was 15% for TBT and 3% for dibutyltin (DBT) when the sample was homogenized. No RSD was calculated for monobutyltin (MBT), due to the large variability in the extraction results. The detection limit was approximately 0.5 ng Sn g^{-1} for MBT and 0.2 ng Sn g^{-1} for DBT and TBT. At regular intervals, the injection of a standard of ethylated butyltin compounds was performed to ensure the validity of the calibration curves.

From Table 1, it is quite evident that sample preparation before extraction was crucial to ensure the reproducibility of the analysis. The repeated extraction of TBT and DBT from a homogenized liver sample gave satisfactory results. In contrast, poor reproducibility was achieved for replicate analyses of non-homogenized liver tissue, i.e. of different thin slices of the same liver that were finely divided and extracted. The question arises as to what extent data obtained from the analysis of small sub-samples of organs collected from large animals, such as whales, can be extrapolated to estimate the level of contamination of that animal. Obviously, it is not practical to homogenize the whole liver (15–20 kg) of a beluga whale; therefore it is necessary to define a suitable strategy of sub-sampling the liver, a key organ for TBT accumulation in mammals. This prompted a subsequent analysis of sub-samples that were carefully removed from various parts of the liver of one specimen to test for anatomical variations of the concentrations of butyltin compounds in this organ (see Results and Discussion section).

RESULTS AND DISCUSSION

Butyltin compounds in beluga whales, St Lawrence Estuary 1995–1998

The St Lawrence beluga stock is unique from an ecological point of view. Populations of free-ranging animals, in particular marine mammals, are notoriously ill-defined.[16] However, the beluga whales from the St Lawrence constitute a distinct population that is confined to a specific habitat. Moreover, the beluga whales in the St Lawrence Estuary are geographically isolated from other northern populations, being a relict population of an arctic or subarctic species since the last glaciation.[10,17]

Sharing a marine environment with numerous species of cetaceans and pinnipeds, the beluga population is at risk due to decades of exposure to toxic chemicals originating from the heavy industrialized catchment basin of the St Lawrence River. More than 20 toxic compounds have been identified in beluga from this location since 1982. Pollutants comprising organochlorinated compounds, heavy metals and polyaromatic hydrocarbons (PAHs) often occur at concentrations amongst the highest ever detected in marine mammals.[18] In contrast to many organochlorinated compounds, butyltin compounds have not generally been considered to be persistent pollutants in biota. Studies with several different types of organisms have demonstrated that debutylation occurs relatively rapidly.[2] If metabolism and elimination processes are not affected by the toxic stress due to TBT, butyltin concentrations should decline if the uptake ceases.[9] Thus, bioconcentrations should reflect recent accumulation.

Butyltin compounds were present in all the liver samples from beluga whales of the St Lawrence Estuary (Table 2). Total butyltin concentrations varied from 36 to 2085 ng Sn g^{-1} (dry wt). These concentrations are similar to those reported for cetaceans from the north Pacific, Asian coastal waters and the Atlantic coast of the USA.[7,19] There was no clear relationship between the butyltin concentrations in the liver and the body length of the specimen (Fig. 2). There was also no significant difference in butyltin concentrations between male and female whales; *t*-test calculations based on the ratio of total butyltin content ($\sum$BTs) to body length gave $t = 0.63$ with the critical value of t ($P = 0.05$) being 2.14. This finding agrees with reports for other cetaceans.[7,20]

Table 2 Concentrations of butyltin compounds in beluga liver samples

Sample[a]	Sex	Age (year)	Length (cm)	Concentration (ng Sn g^{-1} dry wt)				
				MBT	DBT	TBT	∑BTs	TBT/DBT
St Lawrence Estuary								
9507	M	0	199	1	132	111	244	0.8
9508	F	0	157	7	57	67	130	1.2
9509	M	1.5	247	26	735	116	877	0.2
9511	M	26+	419	399	282	84	764	0.3
9512	M	11	400	6	53	8	66	0.2
9601	F	21+	427	9	250	22	281	0.1
9602	F	5	325	9	45	0	54	
9604	M	16++	420	0	96	0	96	
9607	M	24+	399	11	100	0	111	
9609	M	23++	393	23	188	0	210	
9608	F	1.2	216	17	137	0	153	
9701	M	8	396	7	229	101	336	0.4
9702	F	28.5	338	7	141	58	205	0.4
9703	F	21+	363	6	1613	467	2085	0.3
9704[b]	M	0	139	9	12	15	36	1.2
9705	M	7.5	378	14	160	40	214	0.3
9706	F	31+	385	10	716	308	1034	0.4
9802	M	N/A	401	9	294	14	316	0.1
9803	F	N/A	377	27	289	27	342	0.1
9804	F	N/A	356	16	208	0	224	
9806	F	N/A	388	16	54	28	98	0.5
Hudson Strait								
98348	M	17	N/A[c]	n.d.[d]	n.d.	n.d.		
98349	F	11	N/A	n.d.	n.d.	n.d.		
98358	M	N/A	N/A	n.d.	n.d.	n.d.		
98359	F	>15	N/A	n.d.	n.d.	n.d.		
98482	F	N/A	N/A	n.d.	n.d.	n.d.		

[a] The first two digits indicate the year when the whale was found stranded.
[b] This beluga whale was a neonate.
[c] N/A, not available.
[d] n.d., not detected.

Trophic transfer of butyltin compounds to the beluga whales

Because of their known confinement to the estuary, it is possible to estimate the biotransfer of various chemical contaminants from the environment to the beluga whales in the St Lawrence Estuary. From the butyltin concentrations in the liver of beluga, an estimate of the mean concentration in the whole animal can be made. Thus, based on a mean total butyltin concentration in liver of 74 ng Sn g^{-1} (wet wt), and assuming that the mass of total butyltin in a 20-kg liver comprises 15% of the total body burden,[21] the mean butyltin concentration in a 1 000-kg whale is estimated to be 10 ng Sn g^{-1} (wet wt). This result is comparable with the mean body concentration (18 ng Sn g^{-1}) calculated using data from the 1988 St Lawrence Estuary specimens.[8]

For a population of 600 beluga whales with a normal age distribution in the St Lawrence Estuary it has been estimated that the food requirement (invertebrates, molluscs, and small fish such as capelin) would be 2000–4000 tons annually.[18] As the proposed biomagnification factor of butyltin compounds from food to a marine mammal is about 2,[21] the butyltin contamination of the food resources of the whales of the St Lawrence Estuary should be about 5 ng Sn g^{-1} (wet wt). This degree of contamination is of the same order of magnitude as the 'background' levels of butyltin contamination estimated for the molluscs inhabiting the intertidal areas of the estuary.[5] Thus, the low level

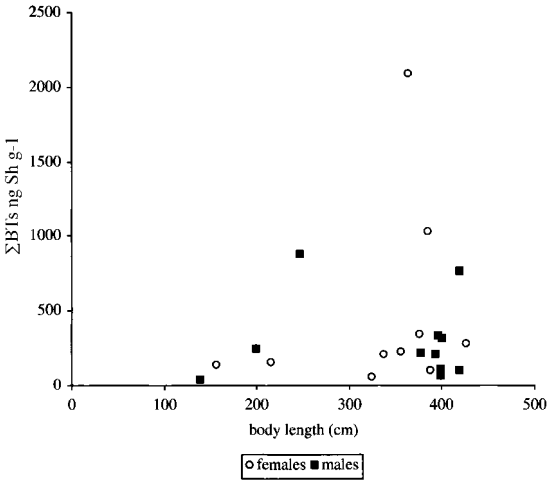

Figure 2 Relationship between butyltin contamination levels $\sum$BTs in liver of beluga whales and the body length for male and female specimens.

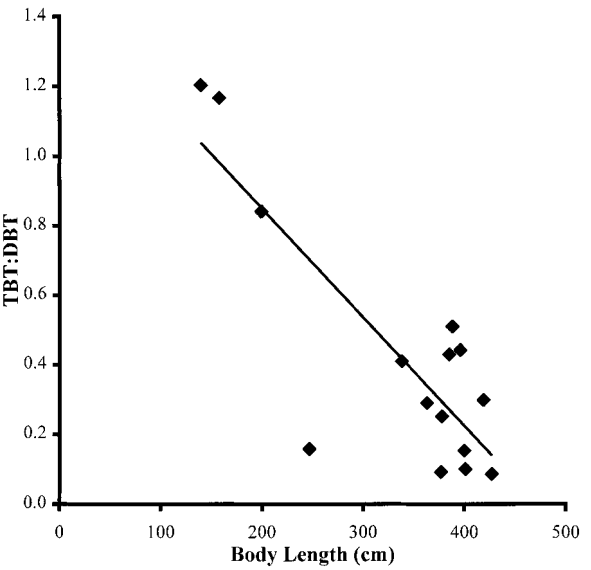

Figure 3 Relationship between the metabolic index (TBT/DBT) and the body length. The line indicates the linear regression best fit ($r^2 = 0.7226$).

of contamination of biota in the St Lawrence Estuary by butyltin compounds is adequate to explain the contemporary concentrations of butyltin species in the beluga whales.

Numerous factors, including age, health, feeding habits and habitat, may induce differences in the TBT concentrations in the liver between comparable specimens. As outlined above, the beluga whales of the St Lawrence Estuary are amongst the most contaminated whales yet studied. Other confounding processes, such as synergistic effects between toxic chemicals or the inhibition of detoxification pathways, could occur and cause higher accumulation of butyltin compounds in the most severely affected animals. For instance, it has been established that organotin compounds cause inhibitory effects on cytochrome *P*450 catalytic activity (e.g. in liver of rats, and marine and freshwater fish), which as a consequence may modulate the induction response by other organic environmental pollutants, as suggested by Fent.[9] Diet and feeding modes could be factors influencing the accumulation of butyltin compounds. Firstly, this population of beluga whales is divided into small groups, which inhabit different areas of the estuary and the gulf.[18,22] Females with their calves are found mainly in the southern part of the estuary during the summer, whereas other adults prefer to live in the St Lawrence Trough. Secondly, beluga whales can consume small fish but the size of the oesophagus limits the prey size to herrings and smaller species, such as capelin. However, a particular feeding technique used by beluga is the violent expulsion of water from the mouth to release invertebrates from the sea bottom.[23] Thus, as is rarely the case amongst toothed whales, they feed heavily on benthic organisms. Considering the known accumulation of TBT and its breakdown products in sediments, the consumption of contaminated benthic organisms could represent a notable pollutant pathway.

Potential butyltin transfer from mother to foetus

Liver sample 9704 (Table 2) was obtained from a neonate. The animal had been found alive in a bay on the south shore of the estuary and taken to the marine laboratory, but it died the same day. The necropsy revealed that the neonate had no milk in its stomach. The only explanation for the presence of butyltin compounds in the liver (with a TBT/DBT ratio of 1.2:1) is via *in-utero* transfer from maternal blood to the foetus. The transplacental transfer of butyltin compounds has been documented for a pregnant killer whale.[7] Although these authors suggested that this transfer was not as important as had been observed for methylmercury and organochlorinated compounds, the TBT/DBT concentration ratio calculated from their data is

Table 3 Concentration of butyltin compounds in different parts of the liver of a beluga whale (no. 9806)

Sample	Concentration (ng Sn g^{-1} dry wt)				
	MBT	DBT	TBT	∑BTs	TBT/DBT
Left lobe	16.0	54.0	27.5	97.5	0.5
Right lobe	—	40.5	26.0	66.5	0.6
Hepatic portal vein	18.0	51.5	74.5	144.0	1.5
Inferior vena cava	14.0	8.0	26.0	48.0	3.1

about 1.3:1 for the foetus, compared with only about 0.1:1 for the mother.

This marked difference in the TBT/DBT ratio in the liver between an adult whale and an immature specimen was also observed in results from the St Lawrence Estuary (Fig. 3). A good linear correlation ($r = 0.7226$) was obtained for the relationship between the length of the animal and the TBT/DBT ratio, which can thereby serve as a metabolism index. The highest ratios were recorded in whales younger than one year old (samples 9507, 9508 and 9704). Like most mammals, beluga neonates feed exclusively on maternal milk.[24] The high concentration of TBT relative to the concentration of its breakdown product DBT in the liver of the calves may indicate the inability of the immature liver to metabolize the triorganotin compounds coming from the mother. There are at least two alternative explanations. Firstly, maternal milk may also have a similarly high ratio. Secondly, the relatively different ratio between young and adult whales may denote recent contamination. This could be possible if the population changed feeding habits or locations.

Distribution of butyltin species within the liver

The analysis of four different parts of the liver from sample 9806 (a female) revealed disparities in concentrations and in TBT/DBT ratios within the same organ (Table 3). Hepatic tissue close to the portal vein, through which the blood normally enters the liver, had the highest butyltin concentration. Conversely, hepatic tissue near the inferior vena cava, through which the blood leaves the liver, had the lowest butyltin concentration. These two anatomical parts of the liver also exhibited higher TBT/DBT ratios than did the hepatic lobes. The higher proportion of DBT in the lobes is consistent with metabolism of triorganotin compounds occurring within the liver. These results further suggest that the blood in the whale might have a higher proportion of TBT relative to its breakdown compounds. This hypothesis is supported by Yang *et al.*,[8] who found that TBT concentrations in the bottom layers of blubber close to more highly vascularized muscle were higher than those measured in the more superficial blubber layers.

Blood may be a better indicator of contamination by butyltin compounds than the liver because both the concentrations and the TBT/∑BTs ratios vary greatly within this organ. By sampling the blood of live marine mammals (i.e. from tail for small whales such as the beluga) with a well-defined protocol, more valuable and comparable data would be obtained.

Butyltin compounds in beluga whales from northern Quebec

No butyltin compounds were detected in the liver of the five beluga whales originating from northern Quebec. This finding contrasts with data published on the butyltin contamination of whales from remote areas[7] and the notion of global TBT pollution in the marine environment as proposed by Yamada *et al.*[25] based on their observations of contaminated squid liver. In contrast to the heavy commercial traffic in the St Lawrence Estuary, the shipping activities in this part of the Canadian Arctic are very limited. In 1991, about 14 000 t of dry cargo and 36 000 t of bulk petroleum products were shipped to the northern communities. The vessels (eight cargo ships and five tankers) were supported by icebreakers of the Canadian Coastal Guard when necessary.[26] This maritime traffic is most likely insufficient to provide an appreciable input of TBT into the marine environment of northern Quebec.

Yamada *et al.*[25] have suggested, but not evaluated, the atmospheric transport of organotin to the remote marine environment. It is well documented that marine mammals inhabiting the

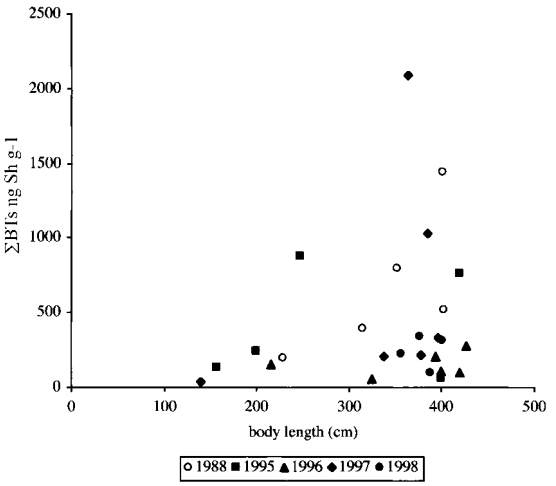

Figure 4 Yearly trend of the varation of butyltin contamination in liver of beluga whales with the body length. The organotin data for 1988 are from Ref.[8], whereas the body lengths were obtained from the database of the Centre Canadien Coopératif de la Santé de la Faune et Centre Québécois sur la Santé des Animaux Sauvages, Faculté de Médecine Vétérinaire, Université de Montréal.

Arctic are contaminated by various organochlorinated compounds, due to the long-range aerial transport of these pollutants.[27,28] Analysis of the liver samples of these beluga whales from the Hudson Strait demonstrated their contamination by organochlorinated compounds. The mean concentrations ($pg\,g^{-1}$) were ($n = 5$): $\sum$DDT $13\,202 \pm 9\,559$; $\sum$BPC $19\,540 \pm 15\,410$; and $\sum$chlordane, 242 ± 133 (Brochu, unpublished results). Thus, the beluga whales from the Hudson Strait were probably exposed to atmospheric input of organochlorinated compounds. However, since no butyltin compounds were detected in their liver, it is unlikely that aerial input could be a source of organotin compounds to the Arctic whale populations.

As the liver is a key organ for butyltin accumulation in marine mammals, and analyses failed to detect their presence in the beluga liver from Hudson Strait, the risk of human exposure to butylin compounds from the consumption of beluga meat of animals hunted in the Hudson Strait area is nil.

Yearly trend of butyltin contamination in beluga whales

The results of this study for beluga whales from the St Lawrence Estuary cover the period 1995–1998. Year-to-year differences are not evident even when the data for beluga found stranded in 1988 are included (Fig. 4).[8] ANOVA calculations based on the $\sum$BTs body length ratio gave $F = 1.44$ with the critical value of F ($P = 0.05$) being 2.84. This indicates there has been no significant variation in the contamination of this whale population since the introduction of Canadian regulations in 1989 prohibiting the use of organotin-based paints on vessels less than 25 m long. Moreover, the highest concentration was detected in a female stranded in 1997 ($2085\ ng\ Sn\ g^{-1}$ in liver sample 9703; Table 2). The major difference between the two data sets is that the concentration of MBT in the liver of the 1988 samples was higher than the concentration of TBT. One explanation could be that the 1988 samples were analysed almost ten years after collection and degradation of TBT and DBT in frozen samples after such a long storage period cannot be ruled out.

Nevertheless, these observations clearly indicate that TBT input into the marine environment of the St Lawrence Estuary is still a matter of concern ten years after restrictions were imposed. Recreational boating activities are marginal in the estuary, particularly considering that it is ice-covered during the winter months. Although other sources of organotin compounds, such as the effluents from sewage treatment plants of large cities along the St Lawrence River,[4] are possible, the predominant source of TBT is most likely to be commercial shipping. In 1992, about 7 000 large commercial vessels (94.5×10^6 t) navigated the estuary.[12] The butyltin concentrations of the beluga whales inhabiting these waters confirms the susceptibility to TBT contamination of marine organisms living in close proximity to a major shipping route.[2,29]

CONCLUSIONS

Given the similarity in butyltin concentrations in whale liver presented here and as reported for animals found 10 years ago,[8] this study has demonstrated clearly that TBT and its breakdown products are still notable contaminants for the beluga whale population of the St Lawrence Estuary. Levels of contamination in the liver of adult animals reached over $2\,000\ ng\ g^{-1}$ and were comparable with concentrations reported for other marine mammals in various parts of the world. Elevated TBT/DBT ratios in a neonate indicate that

transplacental transfer from maternal blood is a mechanism of organotin biotransfer. Similarly high ratios for suckling calves may indicate either the ongoing transfer via maternal milk, or the inefficiency of the immature liver to debutylate TBT.

The beluga whale population of the Hudson Strait, northern Quebec, had no detectable levels of organotin compounds in their liver tissues. In contrast, all individuals from the St Lawrence Estuary were contaminated with butyltin compounds, thereby confirming the susceptibility of TBT exposure for marine organisms living in the vicinity of a major shipping channel.

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