Dietary Exposure To Thyroid Disrupting Chemicals: A Community-Based Study in Newfoundland (Canada)

Nicole Babichuk

Memorial University of Newfoundland

Atanu Sarkar (✉ atanu.sarkar@med.mun.ca)

Memorial University of Newfoundland Faculty of Medicine <https://orcid.org/0000-0003-1797-4971>

Shree Mulay

Memorial University of Newfoundland

John Knight

Memorial University of Newfoundland

Edward Randell

Memorial University of Newfoundland

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Abstract

The marine ecosystem around the Island of Newfoundland is contaminated by Thyroid Disrupting Chemicals (TDCs). Coastal inhabitants may be exposed to TDCs through consumption of contaminated local seafood products and affecting thyroid functions. The aim of this study was to explore: (1) consumption frequency of local seafood products consumed by rural residents, (2) thyroid hormones (THs) and TDCs concentrations in residents, (3) relationships between local seafood consumption, TDC concentrations, and THs. Participants ($n = 80$) were recruited from two rural Newfoundland communities. Seafood consumption was measured through a validated seafood consumption questionnaire. Blood samples were collected from all participants and tested for THs (thyroid stimulating hormone, free thyroxine, free triiodothyronine) and TDCs including polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), and dichlorodiphenyldichloroethylene (p,p'-DDE). Cod was the most frequently consumed local species, but there was a wide range of other local species consumed. Older participants (> 50 years) had greater plasma concentrations of PBB-153, PCBs and p,p'-DDE, and males had higher concentrations of all TDCs than females. The consumption frequency of local cod was found to be positively associated with several PCB congeners, p,p'-DDE and $\sum_{14}$ TDCs. There was no significant relationship between TDCs and THs in either simple or multivariate linear regression analyses.

Introduction

Endocrine Disrupting Chemicals (EDCs) are exogenous substances that cause adverse health effects in organisms or their offspring, consequent to changes in endocrine functions (Damstra et al., 2002). Most EDCs are manufactured intentionally or as byproducts of human industrial activities. They can be found in the household environment, shedding from consumer products into dust; they are also present in many personal care products, food items, and can shed from household items. Eventually, EDCs escape into the environment where they can disperse over long distances.

Thyroid hormones (THs) include triiodothyronine (T3), thyroxine (T4) and thyroid-stimulating hormone (TSH), and these are the prime targets of several structurally diverse EDCs, also known as thyroid disrupting chemicals (TDCs). THs play crucial roles in human growth and development, contributing to the nervous system, bones, lungs, cardiovascular system, and metabolic functions. TH levels are regulated through a negative feedback system involving the hypothalamus, pituitary, and thyroid gland. Peripheral metabolism of these hormones helps maintain levels within a narrow normal range (Khetan, 2014). TDCs alter TH homeostasis through various mechanisms (e.g. altering receptor proteins, binding to transport proteins, modifying cellular uptake and metabolism of THs), thus interfering with the hypothalamic-pituitary-thyroid axis (Boas et al., 2006).

Research has pointed to polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), and dichlorodiphenyldichloroethylene (DDE) as being thyroid disruptors, and their ubiquitous presence in the environment makes them an ideal candidate to study

population-level exposure and health effects. There are five PBDE congeners (PBDE-47, -99, -100, -153, -154) used commercially that are most commonly found in the environment and are known to bioaccumulate (O'Driscoll et al., 2016). In studies measuring the accumulation of PBDEs in serum and tissues, PBDE-47, -99, and -153 predominate (Costa et al., 2008; Linares et al., 2015). All three of these chemicals are associated with thyrotoxic effects in humans. Evidence indicates that PBDEs may compete with THs for binding to the transport protein transthyretin (TTR) in the serum and suppress TH receptor transcription in tissues (Lema et al., 2008; Macaulay et al., 2015; Yang et al., 2011), while PCBs have been shown to decrease total T4 levels in rats and mice (Hallgren et al., 2001). This trend persists at the population level in humans, where PCB concentrations have been reciprocally associated with total T4 in women from the US (Turyk et al., 2007). PCBs have also been shown to upregulate microsomal enzymes (Hallgren et al., 2001), and the hydroxylated metabolites (OH-PCBs) can competitively bind to TTR (Purkey et al., 2004). PBB-153 has been associated with metabolic alterations (Walker et al., 2019). PBB levels are associated with higher free T3 (FT3) but lower FT4 (Curtis et al., 2019), and at the population level PBB-153 is associated with thyroid disease (Jacobson et al., 2017). A positive association has been found between DDE and both FT4 and total T3, while a negative association has been established between DDE and TSH (Meeker et al., 2007). Exposure to DDE is associated with a decline in serum TTR, elevated TH receptor mRNA expression and upregulated hepatic enzymes (Liu et al., 2011).

Ingestion of contaminated foods is a major route of TDC exposure in humans (Jones-Otazo et al., 2005). Seafood products frequently have high concentrations of TDCs, and studies have found TDCs in the serum of seafood consumers (Bocio et al., 2003; Schecter et al., 2006a; Voorspoels et al., 2007; Domingo et al., 2008; Byrne et al., 2018). However, many of the previous studies exploring associations between TDCs in seafood and human serum levels have not incorporated consumption surveys to investigate the type and frequency of seafood being consumed. Although Turyk et al. (2008) incorporated a dietary survey to link the congener levels of PBDEs, PCBs and DDE with THs, the study did not explore the relationship between frequency of consumption and specific congeners of TDCs.

In the present paper we address the existing knowledge gap by linking seafood ingestion (via a dietary survey) and serum levels of TDCs, as well as exploring possible associations with THs. A community-based survey was conducted on the Island of Newfoundland (NL), a Canadian province which has a rich history in fishing and local seafood consumption, as reflected in their culture and cuisine. The majority of the population (90%) live along the coasts, with easy access to local seafood (Irvine, 2012). Previous studies have shown that residents of rural communities prefer to consume local over imported seafood (Lowitt, 2013).

The island of NL shares its coastlines with the Atlantic Ocean and the Gulf of St Lawrence (GSL). The south and west coasts of the island sit directly in the GSL, while the northeast coast is situated in the open Atlantic Ocean and receives the Labrador Current (LC; Fig. 1). Further upstream from the GSL is the St. Lawrence River (SLR), the Great Lakes, and the associated drainage basin which is home to over 45 million people from the US and Canada. This is one of the most contaminated waterways in Canada, draining more than 25% of the Earth's freshwater reserves along with urban and industrial effluents

(Environment and Climate Change Canada, 2017a). Several types of TDCs have been found in the water, sediment, fish, and mammals sampled along the river and its estuary (Environment and Climate Change Canada 2017b; Houde et al. 2014; Marcogliese et al. 2015; Gentes et al. 2012; Simond et al. 2017; Pelletier and Rondeau 2013). The GSL is the drainage point for the SLR into the North Atlantic Ocean. The LC originates in the Arctic and flows along the northeast coast of NL; it is believed to be contaminated by TDCs deposited in the Arctic by long-range atmospheric transport. The presence of TDCs in higher trophic-level organisms in the Canadian Arctic indicates environmental pollution throughout the food chain (Verreault et al., 2018; Morris et al., 2018). The Arctic acts as a sink for many chemicals transported on atmospheric currents, hence, as climate change continues in these areas, older banned or legacy chemicals may undergo a resurgence in the global environment. Climate change in the Arctic is believed to melt sea-ice cover resulting in the release of more TDCs, previously locked in the ice shield, into ocean currents and marine ecosystems (Armitage et al., 2011).

A recent survey showed that cod (*Gadus morhua*) and turbot (*Scophthalmus maximus*) caught around the island of NL are contaminated with PBDEs (Babichuk et al., 2018). Thus, NL is a unique region for studying human exposure to TDCs through local seafood consumption and its effects on population health. We hypothesize that consumption of local seafood in coastal communities in NL is associated with TDCs concentration in the blood samples of the seafood consumers and affecting the levels of THs. We therefore aimed to explore: (1) consumption frequency of local seafood products consumed by rural residents, (2) THs (TSH, free T4, free T3) and TDCs (PBDEs, PCBs, PBBs, and DDE) concentrations in residents, (3) relationships between local seafood consumption, TDCs and THs.

Methods And Materials

Site selection and recruitment

Two rural communities were selected for this study; Burin on the south coast and New-Wes-Valley (NWV) on the northeast coast (Fig. 2). Study sites were selected due to their remote location, small population (2000–3000 residents), proximity to a blood collection facility, limited access to imported seafood from local grocery stores and easy availability of local seafood (i.e. self-harvested, local fish plants, and professional fish harvesters). Data collection questionnaires (seafood consumption frequency and participant demographics) were administered between February and April 2018. Participants were recruited by telephone through numbers were purchased from InfoCanada© and invited to participate in the study. Additional recruitment methods included advertisements on social media (town council Facebook page), emails circulated throughout the local healthcare and fitness centers and recruitment via hospital staff of patients attending blood collection services. Inclusion criteria encompassed age (> 18 years), non-pregnant, and capable of giving consent to participate. A total of 80 participants took part in the study of which six had a preexisting diagnosis of hypothyroidism. This sample size aligns with other peer-reviewed exploratory/pilot studies from Canada (Sandanger et al., 2007, n = 110), US (Byrne et al. 2018 n = 84) and China (Zhang et al. 2010, n = 50). Both studies examined relationships between blood TDCs and thyroid hormones.

Seafood consumption questionnaire (SCQ)

All participants completed the SCQ, which was modified and validated from the seafood consumption survey obtained from Lowitt (2013) to explore local vs imported seafood preference on the west coast of Newfoundland. The survey consisted of questions on consumption frequency of 15 types of local seafood over the past month. Other questions included frequency of local seafood consumption at different times of the year (fall, winter, spring, summer), and demographic questions about age, postal code, sex, and years of residence in the community. Surveys were coded and entered into SPSS for analysis (Supplementary file 1). For clarity, local seafood was defined in the SCQ as “*seafood harvested close to the community, whereas non-local seafood refers to seafood from other coasts (Newfoundland), countries or provinces, or seafood of which the participant is unsure of the source*”.

Blood sample collection

All participants who completed the SCQ provided two blood samples for testing TDCs and THs. Blood samples for TDCs were collected in 4x4-mL BD® Vacutainer ethylene-diamine-tetra-acetic acid (EDTA)-coated tubes. Samples were centrifuged for 10 minutes at 2500 rpm, and the supernatant plasma was transferred to a pre-cleaned 7-mL glass vial with Teflon disc screw-top (Supelco #23741) and stored at -20°C. The collected plasma samples were stored at -20°C until shipment on dry ice to the accredited laboratory (ISO/IEC 17025 and CAN-P-43, <https://www.inspq.qc.ca/en/ctq/eqas>) The Centre de Toxicologie du Québec (CTQ)/the Institute National de Santé Publique (INSPQ) in Quebec City (Quebec) to analyze TDC concentrations.

Blood samples for THs analysis were collected in 1 x 3.5ml BD Vacutainer gold top serum-separator tube. Briefly, blood was allowed to clot for 10 minutes, then centrifuged at 2500 rpm for 10 minutes. Serum was transferred to a test tube and stored at -20°C until they were transported back to the Health Sciences Centre (St. John's, NL) for analysis of THs.

Extraction and analysis of TDCs

Extraction and analysis of plasma TDCs was conducted by the Institute National de Santé Publique du Québec (INSPQ) in Quebec City. TDCs were extracted from participant plasma samples using the methods described by Fisher et al. (2016). Internal standards were added to the plasma samples; TDCs were extracted using liquid-liquid extraction with hexane and a saturated ammonium sulfate solution. The hexane layer containing the TDC extracts was removed and concentrated, then purified on activated florisil columns using a solution of dichloromethane: hexane (9 mL; 25:75). Internal labeled standards including hexachlorobenzene- $^{13}\text{C}_6$, α -HCH- $^{13}\text{C}_6$, oxychlordan- $^{13}\text{C}_{10}$, trans-nonachlore- $^{13}\text{C}_{10}$, PCB 141- $^{13}\text{C}_{12}$, PCB 153- $^{13}\text{C}_{12}$, PCB 180- $^{13}\text{C}_{12}$, PCB-194- $^{13}\text{C}_{12}$, 3,6-F2-PBDE 99, Parlar 26- $^{13}\text{C}_{10}$, Parlar 50- $^{13}\text{C}_{10}$, p,p'-DDE- $^{13}\text{C}_{12}$, PBDE-101 and PBDE-77- $^{13}\text{C}_{12}$) were used to determine percent recovery of analytes after extraction. Solvent extracts were applied to the Agilent 60 m DB-XLB column (0.25 mm i.e., 0.25 μm film thickness) and run on GC-MS instrument (Agilent 6890 Network/7890A coupled to an Agilent 5973 Network/5975C, Agilent Technologies; Mississauga, Ontario, Canada); helium was used as the carrier gas

and 3µL injections were applied (splitless mode). Methane was used as the reagent gas for mass spectrometry, which was operated in select ion monitoring mode, with negative chemical ionization. Final concentrations of the TDCs measured in serum were given in µg/L, and the method detection limits (MDL) fell between 0.005 and 0.3µg/L, depending on the compound. TDCs concentrations were lipid-normalized and SI units (pmol/g) were used for serum TDC analysis to account for the molecular weight of the different congeners. Total serum lipid level (g/L) included total cholesterol, free cholesterol, triglyceride, and phospholipid levels measured by enzymatic methods and colorimetry.

THs testing

After defrosting, samples were briefly vortexed and centrifuged at 2000 rpm. Each sample was tested for TSH, FT3, and FT4 using an Architect Chemiluminescent Micro-Particle Immunoassay kit (Abbott Laboratories) run on an Architect i2000SR immunoassay analyzer (Abbott Laboratories). Concentrations were expressed in mIU/L (TSH) and pmol/L (FT4, FT3). The lab reference ranges from the Clinical Biochemistry Laboratory at the Health Sciences Centre (HSC, St. John's) were used to determine the participants' thyroid profiles (TSH 0.35–4.94 mIU/L, FT4 9–19 pmol/L, FT3 2.6–5.7 pmol/L).

Statistical analysis

All statistical analyses were conducted using SPSS® software (version 26, IBM). For the SCQ, the differences in consumption frequencies by species between demographic groups (age, sex, community, and hypothyroidism) were analyzed by chi-square test. Some species had a limited number of responses for seafood consumption frequency more than once per month and therefore violated the chi-square test assumptions. The responses for these species were amalgamated into fewer response categories and analyzed by Fisher's exact test.

TDCs with more than 5% of samples below the method detection limit (MDL) were corrected to MDL/2 (Lubin et al., 2004; Dallaire et al., 2009). The median and interquartile range were calculated for all THs as well as for TDCs with $\geq 70\%$ detection frequency (more than 70% of our participant samples have concentrations above the MDL). Age, sex, and community were coded as categorical variables. Age was divided into two groups (< 50 years and > 50 years of age) as TSH concentrations trend towards higher concentrations with increasing age, particularly after 50 years (Surks and Hollowell, 2007). The Mann-Whitney U-test was used to compare the difference between TDCs and THs concentrations between sexes, age groups, and communities, with a significance level of $p < 0.05$.

We tested for normality in the THs and TDC data by constructing histograms to look for skew and running Shapiro-Wilkes tests for normality; all TDC and TSH data were right-skewed, so the values were log₁₀-transformed to satisfy conditions of normality (Curran-Everett, 2018; Chevrier et al., 2010) (see Supplementary file 2). FT4 and FT3 were not skewed, and therefore not transformed. Outliers were identified by determining the inter-quartile range (IQR) and creating lower ($Q1 - [1.5 \times IQR]$) and upper ($Q3 + [1.5 \times IQR]$) fences (Sjödín et al., 2018). There was one outlier outside of the IQR range ± 3 standard deviations (PBB-153), and this value was winsorized to the next highest value within 3 standard deviations of the range of the mean (Verner et al., 2015).

Relationships between local seafood consumption and plasma TDC concentrations were assessed using a Kruskal-Wallis H-test. Six participants with hypothyroidism on oral TH medication were excluded from TH analysis, as controlled TH levels are not representative of the effects of TDCs on TH concentrations. Univariate linear regression was performed to test for association between TDCs and THs in the absence of any other covariates. Multivariate linear regression was run to test for relationships between serum TDCs and THs with age and sex as covariates and community as an exposure variable.

Results

Demographics of the participants

The majority (70%) of participants were > 50 years (Table 1) and female, and 84% of participants had resided in their community for 20 + years. A further breakdown of the participant demographics showed that in the younger age group (< 50 years), more females (n = 15) than males (n = 5) participated (25% males and 75% female), however for the older (> 50 years) age group the ratios were relatively more equal (male n = 20, 37%; female n = 34, 63%). The study population distribution is similar to the provincial population, where the largest proportion of the populations is > 50 years of age (Statistics Canada, 2017). NWV has higher proportion of aging and female participants than Burin.

Table 1:Demographic profile of the study population from NWV and Burin.

Demographic	N (%)	
Characteristic		
Sex		
• Male	26 (32.5)	
• Female	54 (67.5)	
Age	Male	Female
• 20-29	0 (0)	6 (7.50)
• 30-39	2 (2.50)	5 (6.25)
• 40-49	3(3.75)	6 (7.50)
• 50-59	8 (10.00)	13 (16.25)
• 60-74	8 (10.00)	18 (22.50)
• 75+	4 (5.00)	5 (6.25)
• No Answer	1 (1.25)	1 (1.25)
Residence duration	Male	Female
• Less than 1 year	0 (0)	0 (0)
• 2-10 years	2 (2.50)	3 (3.75)
• 10-20 years	2 (2.50)	4 (5.00)
• 20+ years	21 (26.25)	46 (57.50)
• No Answer	1 (1.25)	1 (1.25)
	Burin	NWV
Age	29.1% <50 years (n=14)	25.0% <50 years (n=8)
	66.7% >50 years (n=32)	75.0% >50 years (n=24)
	4.2% N/A (n=2)	
Sex	37.5% Male (n=18)	25.0% Male (n=8)
	62.5% Female (n=30)	75.0% Female (24)

Seafood consumption

The most frequently consumed local seafood species was cod, followed by salmon, shrimp, scallops, and crabs (Table 2), which aligns with a previous study by Lowitt (2013). Participants from NWV reported consuming local cod more frequently in the past month than participants from Burin ($p = 0.010$).

Participants (male and female) > 50 years of age reported a significantly higher consumption frequency (per week) of local cod ($p = 0.000$), halibut ($p = 0.011$), and smelt ($p = 0.024$) in the past month than younger (< 50 years) participants. There was no difference in any species consumption frequencies between hypothyroid ($n = 6$) and non-hypothyroid participants ($n = 74$), however, these results must be interpreted with caution.

Table 2: Seafood consumption (%)^a of the participant from Burin and NWV.

Type of Seafood	Almost never		Once/month		2-3 times/month		1 or more times/week	
	Burin	NWV	Burin	NWV	Burin	NWV	Burin	NWV
Capelin	83.3	81.3	4.2	12.5	0	6.3	2.1	0
Cod*	14.6	0	35.4	25.0	37.5	59.4	10.4	15.6
Crab	68.8	78.1	22.9	21.9	2.1	0	0	0
Halibut	68.8	90.6	20.8	6.3	4.2	3.1	0	0
Herring	91.7	90.6	0	9.4	0	0	0	0
Lobster	79.2	87.5	12.5	9.4	0	0	0	3.1
Mackerel	89.6	87.5	2.1	6.3	0	6.3	0	0
Salmon	54.2	46.9	31.3	43.8	14.6	6.3	0	3.1
Shrimp**	52.1	78.1	35.4	15.6	10.4	6.3	0	0
Scallops**	41.7	87.5	50.0	9.4	4.2	3.1	2.1	0
Smelt*	93.8	81.3	0	18.7	0	0	0	0
Squid	87.5	93.8	2.1	6.3	2.1	0	0	0
Trout	79.2	84.4	10.4	15.6	4.2	0	2.1	0
Turbot	87.5	100	2.1	0	0	0	0	0
^a Total % may not equal to 100 due to no responses from some participants * Denotes proportions (%) that were statistically significantly different ($p < 0.05$) between Burin and NWV by Fisher's exact test. ** Denotes proportions (%) that were statistically significantly different ($p < 0.05$) between Burin and NWV by chi square test.								

Plasma TDCs and lipids

All blood samples were tested by INSPQ for PBB-153; PBDE-15, -17, -25, -28, -33, -47, -99, -100, -153); PCB-28, -52, -99, -101, -105, -118, -128, -138, -153, -156, -170, -180, -183; and p,p'-DDE. However, not all tested TDCs were prevalent in the majority of the study population, therefore we restricted our analyses to the

most relevant TDCs for this study, which included those associated with adverse health outcomes in the literature, those above MDL in > 70% of the plasma samples (Dallaire et al., 2009). We focused on 15 TDCs, and two summed TDC groups (Σ_8 PCBs and Σ_5 PBDEs), as they had the highest levels of prevalence in the study population and have also been associated with changes in THs concentrations in the literature.

The mean total plasma lipid concentration (total triglycerides and total cholesterol) was 6.48 g/L (range of 3.92–10.23 g/L). An independent t-test was used to compare plasma lipid concentrations by location, sex, and age (< 50 years vs > 50 years). We did not find any differences in plasma lipid concentrations by age, sex, or community. PCBs were found in much greater concentrations in the plasma samples than PBDEs, and the highest individual TDC concentrations were from one of the most chlorinated PCB congeners, i.e., PCB-180. The congener with the greatest concentration in plasma was also the most brominated congener tested for PBDEs, i.e., PBDE-153 (Table 3).

Table 3

Plasma concentrations (pmol/g lipid) of PBDEs, PCBs, PBBs, and p,p'-DDE tested in participants from Burin and New-Wes-Valley

TDC congeners	Range	Median (IQR)
PBB-153	<MDL-5.94	0.57 (0.00-1.14)
PBDE-28	< MDL-5.94	0.00 (0.00-0.39)
PBDE-47	1.50-66.71	4.84 (3.05–8.52)
PBDE-99	< MDL-13.19	1.63 (1.00-2.23)
PBDE-100	< MDL-39.68	0.00 (0.00-2.16)
PBDE-153	< MDL-148.20	9.03 (4.16–19.09)
$\sum_5$ PBDE	3.15-224.52	19.00 (11.28–36.12)
PCB-99	< MDL-34.34	0.00 (0.00-1.81)
PCB-105	< MDL-15.49	0.69 (0.40–1.31)
PCB-118	1.29–99.18	3.84 (2.08–7.08)
PCB-128	< MDL-5.04	0.05 (0.00-0.12)
PCB-138	1.35-124.83	9.87 (4.76–22.88)
PCB-153	1.91-289.74	20.18 (9.95–49.15)
PCB-170	0.32–88.40	7.60 (2.84–15.54)
PCB-180	0.82-289.84	24.08 (8.15–53.55)
$\sum_8$ PCB	6.52-869.21	73.73 (32.36-159.99)
p,p'-DDE	32.94-1569.07	93.63 (46.80-182.26)
Only TDCs with > 70% samples above MDL or known biological relevance were included in the table (n = 80). Concentrations presented in pmol/g lipid, IQR = interquartile range		

Plasma TDCs and demographic variables

TDC concentrations were analyzed by age, sex, and community using Mann-Whitney U-test (Table 4). There was a trend towards higher individual PBDE congeners and $\sum_5$ PBDE concentrations among participants < 50 years than those > 50 years. Concentrations of PBB-153 were statistically significantly higher in participants > 50 years than participants < 50 years ($p < 0.0001$). The PCBs that were statistically significantly higher in older participants were: PCB-105 ($p < 0.0001$), PCB-118 ($p < 0.0001$), PCB-138 ($p < 0.0001$), PCB-153 ($p < 0.0001$), PCB-170 ($p < 0.0001$), PCB-180 ($p < 0.0001$), and $\sum_8$ PCBs ($p < 0.0001$). p,p'-DDE was also higher in older participants ($p < 0.0001$). We also compared $\sum_{14}$ TDC concentrations by age

and found that older participants had statistically significantly higher concentrations than younger participants ($p < 0.0001$).

Table 4
Median TDC concentrations (pmol/g lipid) in participants by age.

Median pmol/g (IQR)		
Compound	< 50 years (n = 21)	> 50 years (n = 57)
PBB-153	0.00 (0.00–0.00)	0.90 (0.17–1.32)*
PBDE-28	0.00 (0.00–0.30)	0.00 (0.00–0.42)
PBDE-47	5.16 (3.82–8.42)	4.53 (2.68–8.49)
PBDE-99	1.70 (1.33–2.23)	1.53 (0.89–2.17)
PBDE-100	0.98 (0.00–2.57)	0.00 (0.00–2.01)
PBDE-153	11.08 (4.54–25.78)	9.00 (3.76–16.93)
$\sum_5$ PBDEs	23.53 (18.19)	18.93 (10.20–36.75)
PCB-99	0.00 (0.00–0.00)	0.00 (0.00–2.13)
PCB-105	0.33 (0.25–0.58)	0.86 (0.57–1.40)*
PCB-118	1.61 (1.12–2.82)	4.47 (3.16–8.30)*
PCB-128	0.00 (0.00–0.06)	0.07 (0.00–0.14)
PCB-138	3.60 (1.71–5.90)	15.01 (8.65–25.14)*
PCB-153	6.81 (2.97–9.95)	34.54 (18.16–61.71)*
PCB-170	1.72 (0.64–2.42)	13.33 (5.91–17.11)*
PCB-180	4.93 (1.79–7.54)	41.05 (18.41–57.82)*
$\sum_8$ PCBs	23.87 (9.16–32.48)	123.84 (60.26–192.44)*
p,p'-DDE	41.86 (27.45–71.14)	131.26 (70.41–222.72)*
$\sum_{14}$ TDCs	107.40 (55.01–136.80)	289.51 (169.63–471.49)*
* denotes plasma congener concentrations that are statistically significantly higher ($p < 0.05$) between age groupings across one type of TDC, IQR = interquartile range. N = 2 participants did not provide age and were not included in this analysis.		

Over all, men had higher TDCs than in females with statistically significant differences were found for PBB-153 ($p = 0.001$), PBDE-153 ($p = 0.006$), $\sum_5$ PBDEs ($p = 0.008$), PCB-153 ($p = 0.047$), PCB-170 ($p = 0.034$), PCB-180 ($p = 0.029$) and $\sum_8$ PCBs ($p = 0.038$) (Table 5). PBB-153 ($p = 0.023$), PBDE-28 ($p = 0.001$),

PBDE-99 ($p = 0.006$) and PCB-128 ($p < 0.0001$), were significantly higher in Burin participants than in NWV participants (Table 6).

Table 5
Median TDC concentrations (pmol/g lipid) in participants by sex.

Median pmol/g (IQR)		
Compound	Male (n = 26)	Female (n = 54)
PBB-153	1.03 (0.41–1.59)*	0.00 (0.00-0.92)
PBDE-28	0.00 (0.00-0.67)	0.00 (0.00-0.34)
PBDE-47	4.96 (3.67–13.43)	4.74 (2.74–8.11)
PBDE-99	1.84 (1.02–2.90)	1.60 (0.98–2.11)
PBDE-100	0.29 (0.00-3.59)	0.00 (0.00-1.89)
PBDE-153	16.93 (9.04–29.21)*	6.70 (3.46–13.09)
Σ_5 PBDEs	24.64 (14.20-45.45)*	15.44 (8.58–27.69)
PCB-99	0.00 (0.00-0.33)	0.00 (0.00-2.03)
PCB-105	0.87 (0.40–1.44)	0.63 (0.41–1.21)
PCB-118	4.34 (2.05–7.15)	3.61 (2.07–7.01)
PCB-128	0.08 (0.00-0.14)	0.00 (0.00-0.09)
PCB-138	14.44 (5.05–26.49)	9.57 (4.55–21.79)
PCB-153	36.55 (12.52-63.51)*	18.78 (9.40-47.09)
PCB-170	13.55 (3.51–18.89)*	6.09 (2.48–14.40)
PCB-180	42.90 (11.17–66.08)*	19.04 (7.46–46.06)
Σ_8 PCBs	117.92 (59.49-201.63)*	59.94 (29.35-150.06)
p,p'-DDE	114.92 (52.31-177.89)	79.91 (41.56-185.98)
Σ_{14} TDCs	283.33 (171.20-425.23)	179.53 (106.46-350.53)
* denotes plasma congener concentrations that are statistically significantly higher ($p < 0.05$) between males and females across one type of TDC, IQR = interquartile range.		

Table 6
Median TDC concentrations (pmol/g lipid) in participants by community.

Median pmol/g (IQR)		
Compound	Burin (n = 48)	NWV (n = 32)
PBB-153	0.81 (0.00-1.14)*	0.00 (0.00-1.61)
PBDE-28	0.24 (0.00-0.55)*	0.00 (0.00-0.00)
PBDE-47	5.11 (3.53-9.62)	4.14 (2.46-6.93)
PBDE-99	1.88 (1.20-2.46)*	1.34 (0.00-1.79)
PBDE-100	0.60 (0.00-2.63)	0.00 (0.00-1.76)
PBDE-153	9.03 (4.98-23.08)	10.86 (2.93-16.36)
Σ_5 PBDEs	21.35 (13.35-38.04)	13.88 (7.75-24.87)
PCB-99	0.00 (0.00-1.81)	0.00 (0.00-2.62)
PCB-105	0.62 (0.40-1.13)	0.89 (0.42-1.55)
PCB-118	3.40 (1.97-6.15)	4.53 (2.28-8.49)
PCB-128	0.09 (0.05-0.15)*	0.00 (0.00-0.00)
PCB-138	9.21 (4.61-16.70)	18.76 (5.34-27.51)
PCB-153	19.07 (10.07-39.06)	39.21 (9.80-64.75)
PCB-170	6.75 (2.63-14.25)	11.57 (3.25-17.14)
PCB-180	20.01 (8.15-46.67)	34.75 (8.85-58.30)
Σ_8 PCBs	61.68 (32.36-134.17)	129.42 (32.06-199.80)
p,p'-DDE	81.85 (45.11-135.48)	121.94 (54.29-244.75)
Σ_{14} TDCs	191.79 (118.35-317.53)	277.81 (124.11-469.63)
* denotes plasma congener concentrations that are statistically significantly higher (p < 0.05) between Burin and NWV across one type of TDC, IQR = interquartile range.		

PBDE-99 (p = 0.032) was the only TDC that was higher in non-hypothyroidism participants. No other TDCs were significantly different between hypothyroid and non-hypothyroid participants. However, the results of this analysis must be interpreted with caution due to a very low number of participants with hypothyroidism (n = 6).

TDCs and seafood consumption

Fish consumption responses were consolidated to 'never', 'once per month' and 'more than once per month', as there were very few participants who reported consuming any species more than once per month, and only cod consumption frequencies were used for this analysis as this was by far the most commonly consumed species. A Kruskal-Wallis H-test was used to explore whether TDC concentrations differed based on local seafood consumption (Table 7). There was a general trend towards higher plasma TDC concentrations in the highest reported consumption frequency (2+/month) of local cod. Plasma concentrations of PCBs – 118, -138, -153, -170, -180, $\sum_8$ PCBs and $\sum_{14}$ TDCs were all statistically significantly higher in participants who reported consuming local cod twice or more times in the past month, compared with participants who reported no consumption or those that reported consuming cod once in the past month. Plasma PCB-105 and p,p'-DDE were statistically significantly higher in participants who reported consuming local cod 2 + times in the past month than the participants who reported consuming local cod not at all in the past month.

Table 7

Association between local cod consumption frequencies and TDC concentrations (pmol/g lipid).

SCQ consumption frequency by TDC	Median (IQR)	Kruskal-Wallis H	P
PCB-105	0.38 (0.23–0.47)	16.53	0.0001
0/month*	0.59 (0.35–1.03)		
1/month	0.86 (0.60–1.58)		
2+/month**			
PCB-118	2.06 (1.16–2.51)	16.36	0.0001
0/month*	3.36 (1.72–4.62)		
1/month*	4.86 (3.05–9.50)		
2+/month**			
PCB-138	4.57 (1.44–6.88)	15.71	0.0001
0/month*	8.23 (3.45–16.30)		
1/month*	15.51 (8.03–25.99)		
2+/month**			
PCB-153	9.41 (2.61–15.63)	16.86	0.0001
0/month*	15.34 (5.79–33.83)		
1/month*	38.55 (18.08–61.40)		
2+/month**			
PCB-170	2.77 (0.64–4.77)	15.38	0.0001
0/month*	4.07 (1.48–12.03)		
1/month*	13.40 (5.89–17.04)		
2+/month**			
PCB-180	8.36 (1.77–14.34)	16.20	0.0001
0/month*	12.63 (4.43–36.75)		
1/month*	41.05 (17.77–56.86)		
2+/month**			

SCQ consumption frequency by TDC	Median (IQR)	Kruskal-Wallis H	P
$\sum_8$PCBs	30.06 (8.37–51.31)	17.77	0.0001
0–1/month*	47.46 (21.72-105.74)		
1–2/month*	136.87 (58.79-193.54)		
2+/month**			
p,p'-DDE	46.58 (24.21–56.02)	10.86	0.004
0/month*	73.91 (41.47-132.05)		
1/month	124.50 (66.91-232.78)		
2+/month**			
$\sum_{14}$TDCs	107.82 (56.34-136.37)	17.44	0.0001
0–1/month*	161.93 (107.66-311.88)		
1–2/month*	289.51 (175.68-474.69)		
2+/month**			
Only statistically significantly positive associations were reported ($p < 0.05$), indicated by * (lower) or ** (higher) within a congener, IQR = interquartile range.			

Kruskal-Wallis H-test was used to analyze the difference in plasma TDC concentrations by seafood consumption in different seasons (fall, winter, spring and summer). Seasonal consumption frequencies were consolidated into three categories: 'never', 'once/month' and 'more than once/month'. Higher consumption of local seafood in fall was positively associated with plasma concentrations of PCB-118 ($p = 0.033$), PCB-128 ($p = 0.034$), PCB-170 ($p = 0.021$), PCB-180 ($p = 0.011$) and $\sum_8$ PCBs ($p = 0.026$). Higher consumption of local seafood in the winter was positively associated with plasma concentrations of PCB-105 ($p = 0.021$), PCB-118 ($p = 0.016$), PCB-128 ($p = 0.007$), PCB-138 ($p = 0.027$), PCB-153 ($p = 0.023$), PCB-170 ($p = 0.031$), PCB-180 ($p = 0.018$), and $\sum_8$ PCBs ($p = 0.017$). In spring, only PBDE-100 ($p = 0.039$) was associated with greater cod consumption. There was no difference in plasma TDC concentrations for the various seafood consumption frequencies in the summer.

Consumption of cod was analyzed separately from all remaining (non-cod) seafood species because cod was the most commonly consumed seafood species. Additionally, when cod and all remaining seafood species were analyzed together, the results were the same as when cod was analyzed alone. This may be due to the high reported consumption frequencies of cod masking the association of other less commonly consumed species with the plasma TDC concentrations. Multivariate regression was run, with cod consumption as the predictor and plasma TDC concentration as the outcome variable, with age, sex, and community as covariates. Frequency of local cod consumption ('never', 'once/month', 'more than

once/month') was significantly associated with PCB-105, PCB-118, PCB-128, PCB-138, PCB-153, PCB-170, PCB-180, $\sum_8$ PCBs and p,p'-DDE concentrations in the study samples (Table 9).

Table 8
Multivariate linear regression analysis of cod consumption (categorical predictor) and plasma TDC concentrations

Predictors	Outcome	β (95%CI)	P-value
Cod	PCB-105	0.385 (0.138–0.647)	0.003
Cod	PCB-118	0.443 (0.201–0.680)	0.0001
Cod	PCB-128	0.480 (0.142–0.763)	0.006
Cod	PCB-138	0.356 (0.122–0.593)	0.003
Age		0.263 (0.051–0.480)	0.016
Cod	PCB-153	0.393 (0.164–0.621)	0.001
Age		0.277 (0.070–0.486)	0.010
Cod	PCB-170	0.370 (0.144–0.593)	0.002
Age		0.338 (0.133–0.543)	0.002
Cod	PCB-180	0.381 (0.156–0.605)	0.001
Age		0.328 (0.124–0.533)	0.002
Cod	$\sum_8$ PCBs	0.397 (0.170–0.623)	0.001
Age		0.297 (0.091–0.505)	0.005
Cod	p,p'-DDE	0.271 (0.020–0.530)	0.035
PCBs in pmol/g, significance $p < 0.05$, β = Standardized coefficient, CI = confidence interval.			

Table 9
Median and interquartile range (IQR) of thyroid hormone concentrations from participants of Burin and NWV (n = 74).

	Range			Median (IQR)		
	All	Burin	NWV	All	Burin	NWV
TSH (mIU/L)	0.44–5.20	0.65–5.20	0.44–4.67	1.90 (1.32–2.31)	1.74 (1.17–2.14)	2.02 (1.53–2.72)*
FT ₄ (pmol/L)	9.25–15.10	9.25–14.92	9.8–15.10	11.90 (11.25–13.18)	11.73 (11.01–12.72)	12.95 (11.73–13.58)*
FT ₃ (pmol/L)	2.70–4.90	2.90–4.60	2.70–4.90	3.80 (3.40–4.10)	3.85 (3.53–4.20)	3.50 (3.30–4.00)
Lab reference ranges: TSH 0.35–4.94 mIU/L, FT ₄ 9.0–19.0 pmol/L, FT ₃ 2.6–5.7 pmol/L.						
* denotes the statistically significantly higher concentration between communities (Burin [n = 48] and NWV [n = 32]) at the < 0.05 significance level						
Hypothyroid participants not included.						

Age was a statistically significant positive predictor of PCB-138, PCB-153, PCB-170, PCB-180, and Σ_8 PCBs concentrations in this multivariate analysis, and all congener concentrations were higher in older than in younger participants (Table 8). No other covariates were associated with TDC concentrations. We analyzed the association between the consumption of all other seafood species and plasma TDC concentrations. Except for cod, all other seafood species had consumption frequencies summed, and therefore each species consumption frequency was one predictor variable. There was no association between frequencies of all other seafood consumption (without cod) and TDCs.

Plasma THs and demographic variables

The median TH values (Table 9) for non-hypothyroid participants were all within the lab reference ranges. Only one participant (female > 50 years, no known history of thyroid disorders) had TSH levels above the normal reference range (TSH = 5.2 mIU/L); this is close to the prevalence of hypothyroidism in the Canadian population (2–3%, Clemens et al., 2011). There was no effect of age on TH concentrations in the study population. FT₃ was the only TH where sex had an effect on concentration, with males (n = 26) having statistically significantly higher median [IQR] FT₃ concentrations (4.00 pmol/L [3.65–4.23]) than females (n = 54; 3.70 pmol/L [3.40–3.93]; p = 0.046); however, both male and female concentrations were within the lab reference range.

TSH concentrations (median [IQR]) were higher in the serum samples from NWV (2.02 mIU/L [1.53–2.72]) than those from Burin (1.74 mIU/L [1.17–2.14]; p = 0.018), and FT₄ concentrations (median [IQR]) were also higher in participants from NWV (12.95 pmol/L [11.73–13.58]) than in those from Burin (11.73

pmol/L [11.01–12.72]; $p = 0.019$). There was no difference in FT3 concentrations between the two locations (Table 9).

Relationships between TDCs and THs

There was no relationship between TDCs and THs in either simple or multivariate linear regression analyses when continuous variables were used for TDC and TH concentrations. Multivariate regression was also performed with TDCs categorized into quartiles to account for non-monotonic dose-response curve thresholds in the relationships between TDCs and THs (Chevrier et al., 2010). Again, there was no relationship between TDCs and THs. We also tested for the association between $\sum_{14}$ TDCs (predictor) and THs (outcome) using a multivariate regression analysis with age, sex, and community as covariates, but there was no association.

Discussion

This study links local marine species' consumption with blood levels of TDCs and THs, and demographic profile of participants (age, sex and community). We found that residents from the study communities consumed local cod more than any other species, and that participants that were older (> 50 years), male and/or residents of Burin consumed more cod than other demographic groups. There were some obvious trends in TDC concentrations by age, sex and community; older participants (> 50 years) had greater plasma concentrations of PBB-153, PCBs and p,p'-DDE, and males had higher concentrations of all TDCs than females. Participants from Burin in southern NL generally had greater concentrations of PBB-153 and PBDEs, while participants from NWV in northeastern NL generally had greater concentration of PCBs and p,p'-DDE. Frequency of local cod consumption was also found to be positively associated with several PCB congeners, p,p'-DDE and $\sum_{14}$ TDCs. Seasonal effects of seafood consumption on plasma TDC concentrations were also observed; several TDCs were positively associated with higher consumption of local seafood in fall, winter and spring. We did not find any relationship between any TDCs and THs, however multivariate regression analysis revealed that local cod consumption frequency was significantly associated with several PCB congeners, $\sum_8$ PCBs, and p,p'-DDE.

For THs, we found that TSH levels were significantly higher in NWV (2.20 ± 0.19 mIU/L) than in Burin (1.99 ± 0.13 mIU/L); however, both results were still below the upper limit for the lab's normal reference range (4.94 mIU/L). An upper limit of normal (euthyroid) concentration of 2.5 mIU/L has been suggested as a guideline for diagnosing subclinical hypothyroidism, as 95% of humans with no known thyroid conditions will have TSH values below this level; individuals with values above this limit are more likely to have underlying causes artificially elevating their TSH levels (Wartofsky and Dickey, 2005). In NWV, approximately 31% ($n = 8$) of participants had TSH values that were above the subclinical hypothyroidism limit cutoff of 2.5 mIU/L, while the proportion in Burin was lower at approximately 15% ($n = 7$), excluding participants with known hypothyroidism. Therefore, it seems that being above the subclinical hypothyroidism cutoff point was more common in NWV than in Burin, even if the average TSH concentrations above the subclinical thyroid threshold were not different. This may be explained by the

higher proportion (78%) of participants > 50 years of age in the NWV group compared with the proportion that was > 50 years of age (69%) in the Burin group. The skewed age distribution of our participants in this study may likely have impacted the thyroid hormones results, as age has been shown to be an important factor in TSH levels, and age is associated with an increased proportion of individuals with serum TSH concentrations above the 2.5 mIU/L euthyroid limit (Surks and Hollowell, 2007). Our finding of higher FT3 in males than in females aligns with other studies (Strich et al., 2017).

PBDE concentrations were higher in younger participants (< 50 years) while PBB-153, PCBs, and p,p'-DDE were generally higher in older participants (> 50 years), regardless of community. This may reflect the sources of these chemicals and the length of time they have been in production. PCBs have been in production and use since the 1930s, and DDT (the parent compound of p,p'-DDE) has been widely used as an insecticide since 1939. PCB and DDT were both restricted and/or banned by the Stockholm Convention in 2004 as part of the "Dirty Dozen" initial chemicals that were entered under the treaty. In Canada, DDT was phased out in the 1970s and completely banned by 1990; PCBs were banned from import, manufacture, and sale in 1977. However, due to their bioaccumulative nature and long half-lives, they have persisted in the environment for decades and tend to be found in higher concentrations in older individuals. It is possible that older participants had had a long time to accumulate the legacy chemicals (PCBs and DDT), which peaked in use for much of their life span, but were phased out before some of the youngest participants were born. Age has also been shown to be a predictor for PCB and p,p'-DDE concentration in other studies (Laden et al., 1999). On the other hand, PBBs (in production and use from the 1970s onwards) do not follow this trend, being "newer" flame retardants. However, other studies have observed a positive association between PBB-153 and age, corroborating with our findings of higher PBB-153 in older participants (Lim et al. 2008). PBDEs were manufactured from the 1960s onwards, and reached peak production and usage in the 1990s. They were banned under the Stockholm Convention in 2009, long after PCBs and DDT, but many PBDEs are still present in various older consumer products (i.e., automobiles, furniture, carpet, and electronic goods) to this day. PBDEs have been found to be inversely related to age, with younger populations having higher concentrations of these chemicals, likely due to a high degree of TDC exposure that occurs in infants and younger children from breastmilk and hand to mouth activity (Garí and Grimalt, 2013). These results may indicate an age-related cohort effect, particularly for PBDEs, which were higher in younger participants who would have been infants or young children around the peak of PBDE production and use (1980-2000s). Since these chemicals have now been banned, this age group (20–50 years of age) would have had a unique exposure, which would be different from older (> 50 years) or younger (< 20 years) age groups. This possible age-cohort effect of plasma TDC concentrations requires a larger-scale study to explore this effect fully.

Our study found that males had higher plasma concentrations of TDCs than females, and higher consumption of seafood among males (than females) could be the reason. Research has found higher seafood consumption by males and a higher estimated daily intake of DDT and PBDEs via seafood consumption (Guo et al., 2010). Men have also been shown to have a higher intake of PCBs/kg body weight than women from consuming contaminated fish in Norway (Knutsen et al., 2008).

We found that relatively newer TDCs, such as PBBs and PBDEs, were significantly higher in Burin participants. In comparison, legacy TDCs such as PCBs and p,p'-DDE concentrations were higher in NWV participants. PBDEs and PBB were banned from production and use much more recently than PCBs and DDT. Therefore, many products containing PBBs and PBDEs may be still found in regular consumer use or are transitioning into urban effluents and landfills. Abbasi et al. (2014) found that the largest PBDEs flow from consumer products to their waste phase (deposition of EDC-containing products into landfills, recycling centers, wastewater treatment plants) was between 2005 and 2008. PBDEs and PBBs have been documented up and down the SLR in sediments (Pelletier and Rondeau, 2013), fish (Houde et al., 2014), birds (Gentes et al., 2012), and marine mammals (Simond et al., 2017). Therefore, it is plausible that higher concentrations of these TDCs in participants from Burin (south coast of NL) results from contamination from upstream in the SLR making its way into the marine food chain, where they then could be ingested by humans through local seafood consumption.

PCBs and DDT have been banned from manufacture and use for longer than PBB and PBDEs and thus there are fewer of these chemicals transitioning from consumer products into the waste phase. They have a global distribution, with PCBs being documented in seafood species such as cod in different parts of the North Atlantic Ocean (Karl et al., 2016). PCBs and DDT/p,p'-DDE have also been found in Arctic ice (Gregor et al., 1995) and high trophic level Arctic organisms such as the polar bear (Letcher et al. 1995), indicating the widespread presence of these chemicals, both geographically and by trophic level. It appears that climate change may be causing TDCs locked in snow/ice to be released upon melting; these contaminants could then travel south via the Labrador Current into marine ecosystems. Residents of NWV (northeastern NL) may be exposed to these TDCs by consuming marine species from these contaminated ecosystems, whereas residents of Burin (southern NL) are mostly exposed to contamination from the SLR.

It is difficult to discern why we did not see any association between TDCs and THs in the rural NL population while other studies have found significant associations. The major limitation of the study was small sample size (n = 80). However, many smaller-scale research projects have successfully explored associations between TDCs and THs. For example, Byrne et al. (2018), who had a sample size of n = 85, explored PBDEs and thyroid hormones in a remote Indigenous population in coastal Alaska, where they found PBDEs in this isolated population and an association with thyroid hormones (TSH, FT3, and T3). Other examples of successful pilot/exploratory studies with small sample sizes include English et al. (2017), who looked at PBDEs in feces from Australian children (n = 61 but only n = 46 were testable), Li et al. (2017), who conducted a pilot study of PCBs in the breast milk of mothers from a recycling site (n = 46), Lin et al. (2011) who looked at PBDEs and thyroid hormones in cord blood (n = 54), Zota et al., (2011) who measured PBDEs and thyroid function in pregnant women (n = 25), and Zhang et al. (2010) who investigated PCBs and PBDEs and thyroid hormone homeostasis in e-waste recycling workers (n = 50). A recent meta-analysis of correlations between PBDEs and thyroid hormones (Zhao et al., 2015) found that PBDE effects on THs (TSH and total T4) varied and were dependent on exposure and serum concentrations. Lower PBDE concentrations (< 30 ng/g lipid) were negatively correlated with THs and higher PBDE concentrations (> 100 ng/g lipid) were positively correlated with THs, while mid-range (30–

100 ng/g lipid) PBDE levels did not correlate with a change in THs. This non-monotonic dose-response relationship between PBDEs and THs is what makes determining the health effects so difficult. Some of our PBDEs had ranges that would put them in this 30–100 ng/g lipid “no effect” range, which may be contributing to the lack of significant relationships between TDCs and THs in our study population.

Additionally, there were low TDC levels in our plasma samples compared with a past sampling of the general Canadian population. Before this study was conducted, the prevalence of TDCs in the NL population was unknown. However, the Canadian Health Measures Survey (CHMS) collects physical samples (i.e. blood) for biomonitoring of contaminant exposure in the Canadian population. Data on TDCs in blood samples from CHMS collection cycle 1 (2007–2009; Haines et al., 2017) were compared with 75th percentile and 95% confidence intervals [CI] from our study (Table 10). There is a 9-year gap between the most recent blood contaminant exposure data from the CHMS (2009) and the current study (2018), and TDC concentrations in the Canadian population may have changed during this time. Additionally, CHMS data are from a much wider age range of participants (3–79 years) than the current study (19–75+ years), which may affect the comparison between the two studies as higher PBDE and PCB serum concentrations have been reported in toddlers and children than in adults (Toms et al., 2009; Marek et al., 2013). Cycle 1 CHMS blood samples were analyzed recently (2016 to 2017) and therefore reflect current instrumentation/analytic capabilities. There is a clear trend in the present study of finding much lower plasma PCB, PBDE, PBB, and p,p'-DDE concentrations than the Canada-wide CHMS from the previous decade.

Table 10
Comparison of TDC concentrations (75th percentile [95% CI]) between the present study and the Canadian Health Measures Survey (CHMS 2007–2009) from Haines et al. (2017).

EDC congener	NL concentrations P75 (95% CI)	CHMS concentrations P75 (95% CI)
PBB-153	0.73 (0.35–0.67)	<MDL
PBDE-47	5.49 (3.81–6.30)	120 (100–140)
PBDE-99	1.43 (0.99–1.51)	21 (< MDL-26)
PBDE-100	1.39 (0.71–2.36)	21 (< MDL-27)
PBDE-153	12.28 (7.35–14.05)	33 (27–39)
PCB-99	1.43 (0.99–1.51)	<MDL
PCB-105	0.84 (0.50–0.81)	<MDL
PCB-118	4.56 (2.77–4.69)	50 (41–58)
PCB-128	0.08 (0.02–0.11)	<MDL
PCB-138	14.73 (7.63–11.32)	110 (97–130)
PCB-153	31.63 (17.09–25.99)	210 (180–240)
PCB-156	4.40 (2.29–3.45)	32 (29–36)
PCB-170	10.00 (5.39–8.21)	59 (52–65)
PCB-180	34.46 (17.55–27.56)	190 (170–220)
PCB-183	2.36 (1.25–1.86)	19 (15–22)
p,p'-DDE	117.30 (69.55-106.65)	1700 (1300–2100)
MDL = Method detection limit, concentrations in ng/g lipid		

In conclusion, the residents of Newfoundland showed exposure to environmental TDCs via local seafood consumption. A larger population based study is required for total exposure analysis and adverse outcomes.

Declarations

Ethics approval and consent to participate: The research was approved by the Newfoundland and Labrador Health Research Ethics Board (#2016:038). The research received consent from all the participants and the consent form was duly approved by the ethics board.

The statement: ... This correspondence has been reviewed by the Chair under the direction of the Health Research Ethics Board (HREB). Full board approval of this research study is granted for one year effective March 3, 2016...

Consent for publication: Not applicable

Availability of data and materials: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests: Nil

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Authors' contributions: NB collected data and analyzed and interpreted data, AS led in study design, data analysis and manuscript writing, SM in study design, data interpretation, JK in data analysis and ER in lab support and data interpretation.

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Authors' information (optional)

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Figures

Figure 1

Map of Newfoundland showing currents around the Island of Newfoundland (Base map: Source - Map base © OpenStreetMap contributors)

Figure 2

Study locations and participant residence by postal code

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