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PERSISTENT ORGANIC POLLUTANTS IN CHINOOK SALMON (*ONCORHYNCHUS TSHAWYTSCHA*): IMPLICATIONS FOR RESIDENT KILLER WHALES OF BRITISH COLUMBIA AND ADJACENT WATERS

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Abstract—We measured persistent organic pollutant (POP) concentrations in chinook salmon (*Oncorhynchus tshawytscha*) in order to characterize dietary exposure in the highly contaminated, salmon-eating northeastern Pacific resident killer whales. We estimate that 97 to 99% of polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), dichlorodiphenyltrichloroethane (DDT), and hexachlorocyclohexane (HCH) in returning adult chinook were acquired during their time at sea. Highest POP concentrations (including PCBs, PCDDs, PCDFs, and DDT) and lowest lipids were observed in the more southerly chinook sampled. While feeding by salmon as they enter some more POP-contaminated near-shore environments inevitably contribute to their contamination, relationships observed between POP patterns and both lipid content and $\delta^{13}C$ also suggest a migration-related metabolism and loss of the less-chlorinated PCB congeners. This has implications for killer whales, with the more PCB-contaminated salmon stocks in the south partly explaining the 4.0 to 6.6 times higher estimated daily intake for ΣPCBs in southern resident killer whales compared to northern residents. We hypothesize that the lower lipid content of southerly chinook stocks may cause southern resident killer whales to increase their salmon consumption by as much as 50%, which would further increase their exposure to POPs.

Keywords—Persistent organic pollutants Polychlorinated biphenyls Chinook salmon Killer whale Dietary exposure

INTRODUCTION

Two populations of resident killer whales (*Orcinus orca*) frequent the coastal waters of British Columbia, Canada, and Washington, USA. The Canadian Species at Risk Act has designated northern resident killer whales as threatened, while the Species at Risk Act and the U.S. Endangered Species Act have designated southern residents as endangered. Although both are fish-eating, polychlorinated biphenyl (PCB) concentrations in the southern residents (males: 146 mg/kg lipid wt) are almost four times that of northern residents (males: 37 mg/kg lipid wt), placing them among the most PCB-contaminated marine mammals in the world [1]. Both populations elicit a strong preference for chinook salmon (*Oncorhynchus tshawytscha*), which comprises 70% of their estimated diet [2], underscoring the need to characterize persistent organic pollutant (POP) concentrations in this salmonid.

Anadromous chinook, the largest of the Pacific salmon, spend the majority of their life in the pelagic marine environment, where they undergo the majority of their growth before returning to freshwater natal streams for spawning [3]. Fish accumulate POPs through gill uptake (bioconcentration) and dietary uptake (biomagnification) [4]. Exposure to POPs in freshwater, estuarine, and coastal environments may explain the relative contamination of some salmon stocks [5], especially in the relatively PCB-contaminated Puget Sound [6,7]. However, it is apparent that global sources acquired by salmonids during their time in the North Pacific Ocean also contribute substantially to their contamination [8,9]. This has implications for wildlife, because POPs are delivered by salmon to coastal, freshwater, and terrestrial ecosystems [8,10,11]. Salmon partly explain the POPs found in British Columbia wildlife, including resident killer whales [1] and grizzly bears [12], though questions linger about their importance relative to other prey items.

As adult salmon enter near-shore marine waters en route toward their natal streams, they undergo dramatic changes in body weight, and lipid, protein, and water content [13]. Chinook salmon can lose more than 80% of their lipid during their return migration [13], which has profound ramifications for lipid-soluble contaminant concentrations.

The extent to which chinook salmon deliver POPs to resident killer whales is unclear [1], as are the sources of POPs to salmon. In the present study we measured POPs including flame-retardants, industrial by-products, and organochlorine (OC) pesticides in ocean-migrating smolts and in returning adults from four stocks of chinook salmon from British Columbia (Canada) and Washington (USA). Our objectives were to characterize in chinook salmon the POPs acquired locally as juveniles (i.e., prior to ocean migration) and the POPs acquired during time at sea, and to estimate the contribution of chinook to POP exposure in resident killer whales.

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MATERIALS AND METHODS

Chinook salmon collections

Chinook salmon smolts ($n = 18$) and adults ($n = 24$) were collected in southern British Columbia and Washington (Fig. 1). British Columbia adult chinook salmon were collected from Johnstone Strait and near the mouth of the Fraser River in October 2000. Chinook smolts were collected from central Strait of Georgia in August 2000. Washington adult chinook salmon were collected near the mouth of the Duwamish River and from the Tumwater Falls Hatchery on the Deschutes River in September 2001. Puget Sound chinook smolts were collected from the Green/Duwamish River and the Deschutes River during the period May through June 2001. Samples were individually wrapped in aluminium foil, bagged, and frozen at −20°C for transport and subsequent analysis.

Morphometrics and stock identification

Body weight and fork length were recorded for all chinook salmon, while sex was recorded for adult chinook only (Table 1). Dorsal muscle samples (1 cm^2) were collected for the adult chinook collected in Johnstone Strait and the Lower Fraser River and smolts from the Strait of Georgia and placed in 95% ethanol for DNA analyses. Stock identification was carried out by the Molecular Genetics Lab at the Pacific Biological Station (Fisheries and Oceans Canada, Nanaimo, BC). Thirteen microsatellite loci were amplified and DNA fragments were sized and sequenced on an automated ABI 377 DNA sequencer (Applied Biosystems, Foster City, CA, USA). Data analyses and classification procedures for species and stock identification are described elsewhere [14]. Fish scales from the left side posterior of the dorsal fin above the lateral line were removed for age determination, described in detail elsewhere [15]. Age determination was carried out by the Aging Lab at the Pacific Biological Station in accordance with their procedures and criteria, also described in detail elsewhere [16].

Sample preparation

Johnstone Strait and Lower Fraser River chinook fillet (muscle) tissue homogenates were prepared for analyses for both this study and a human health hazard assessment, whereas for Duwamish River, Deschutes River, and all chinook smolts whole fish tissue homogenates were prepared. Strait of Georgia chinook smolts were prepared as individual samples. However, Puget Sound smolts were pooled due to their small body size. Additionally, pooled fillet homogenates were prepared for Lower Fraser River and Duwamish River adult chinook. For Johnstone Strait and Lower Fraser chinook, rest of fish homogenates, which included all fish tissues except for fillet, were constructed for lipid determination in order to calculate POP body burdens, described in detail later. Fillet, rest of fish, and whole fish tissues were homogenized according to procedures described in detail elsewhere [17].

Stable isotope analysis

Whole salmon homogenates (20 g) were freeze-dried for 48 to 72 h and then ground to a fine powder using a mortar and pestle. Bulk stable carbon and nitrogen isotope ratio (^{15}N:^{14}N and ^{13}C:^{12}C) measurements were carried out at the Biogeochemistry Laboratory at the University of Victoria (Victoria, BC, Canada), equipment and standards described elsewhere [17]. Isotopic composition is expressed in δ notation as the proportional deviation in parts per thousand (‰) of the isotope ratio in a sample from that of a standard as in the following equation:

$$\delta X = (R_{\text{sample}}/R_{\text{standard}} - 1) \times 1{,}000 \qquad (1)$$

where X is ^{13}C or ^{15}N, and R_{sample} and R_{standard} are the ratios of ^{13}C:^{12}C or ^{15}N:^{14}N for the sample and standard [18].

Contaminant analysis and lipid determination

Whole body ($n = 12$) and fillet ($n = 12$) adult chinook, whole body chinook smolts ($n = 6$), and one chinook smolt pool of 12 individuals (10 g) were analyzed for congener-specific polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and two pooled fillet samples for polybrominated diphenyl ethers (PBDEs), polychlorinated diphenyl ethers (PCDEs), and polybrominated biphenyls (PBBs) (reported as either individual or co-eluting congeners) using high-resolution gas chromatography–high-resolution mass spectrometry (HRGC-HRMS). For Duwamish River adults and all chinook smolts, organochlorine pesticides, including the dichlorodiphenyltrichloroethane (DDT) group [*o,p′*-DDT, dichlorodiphenyldichloroethane (DDD), dichlorodiphenyldichloroethylene (DDE), and *p,p′*-DDT, DDD, DDE], hexachlorobenzene (HCB), hexachlorocyclohexane (HCH) [α-HCH, β-HCH, γ-HCH], heptachlor, aldrin, chlordane [oxy-, γ-, α-], nonachlor [*trans-, cis-*], and mirex were measured using low-resolution gas chromatography–mass spectrometry (LRGC-MS) and gas chromatography with electron capture detection (GC-ECD).

Extraction and clean-up procedures, instrumental analysis and conditions, lipid determination, and quality assurance/quality control criteria used for PCBs, PCDDs, and PCDFs by the Regional Contaminant Laboratory (Fisheries and Oceans Canada) are described elsewhere [1,19]. Polybrominated diphenylethers, PCDEs, PBBs, and OC pesticides were analyzed by AXYS Analytical Services (Sidney, BC, Canada) according to their laboratory procedures and criteria and are described in detail elsewhere [17]; the PCDE method publication is in process. Lipid values were also determined by AXYS for samples analyzed for PBDEs, PCDEs, PBBs, and OC pesticides. Where whole fish lipid percentage data were compared, Regional Contaminant Laboratory values were reported.

Organochlorine pesticide analyses and lipid determinations for Johnstone Strait and Lower Fraser River fillet tissues were carried out by the Western Regional Laboratory, Health Canada (Burnaby, BC) using an in-house validated analytical method. The sample batch for OC pesticides included 10 samples, a reagent blank, and a replicate. Samples were spiked with ^{13}C-labeled surrogate standards and extracted with acetone:hexane (2:1 v/v) using a Polytron homogenizer (Luzern, Switzerland). The extract was centrifuged and the organic layer was further re-extracted with hexane and saturated sodium chloride. An aliquot of the organic layer was taken to dryness under vacuum with a rotary evaporator for lipid determination. The sample residue was redissolved in dichloromethane and the lipids removed by preparative gel permeation chromatography using a Waters high-pressure liquid chromatograph (Milford, MA, USA). The solvent of the gel permeation chromatography eluate was exchanged to hexane and the sample purified by eluting through a Florisil® (U.S. Silica, Berkeley Springs, WV, USA) column (2% deactivated) with dichloromethane:hexane (60:40 v/v). The purified eluate as concentrated to near dryness, dissolved quantitatively into iso-octane

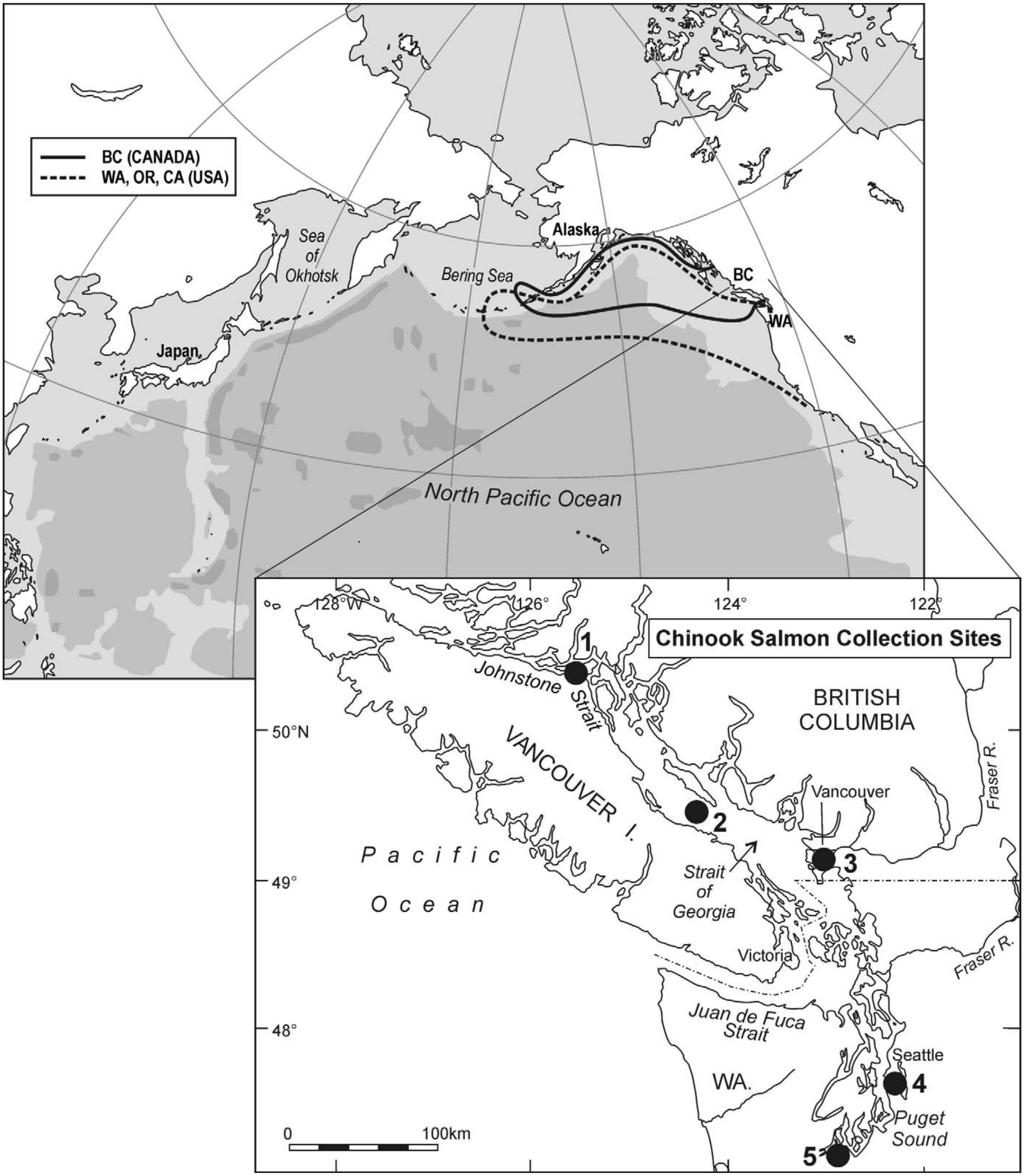

Fig. 1. Migratory routes [adapted from Fisheries and Oceans Canada, 2006 (www.pac.dfo-mpo.gc.ca/species/salmon/salmon_facts/chinook_e.htm)] and collection sites (inset map) for British Columbia (Canada) and Washington (USA) adult chinook salmon (*Oncorhynchus tshawytscha*) (n = 24). Johnstone Strait adult chinook were collected from site 1, Strait of Georgia chinook smolts were collected from site 2, Lower Fraser River adults from site 3, Duwamish River adults from site 4, and Deschutes River adults from site 5, Puget Sound smolts were collected upstream from both sites 4 and 5. Although ocean distribution for BC and Washington chinook encompasses the North Pacific Ocean and Bering Sea, greatest abundance has been observed along the North American coastal shelf waters [3]. BC = British Columbia; WA = Washington; OR = Oregon; CA = California.

Table 1. Morphometric and related data for chinook salmon (*Oncorhynchus tshawytscha*) collected from Johnstone Strait and Lower Fraser River (British Columbia, Canada), Duwamish River, and Deschutes River (Washington, USA) and chinook smolts from the Strait of Georgia (British Columbia, Canada) and Puget Sound (Washington, USA). Values represent mean ± standard error of the mean. One-way analysis of variance (ANOVA) tests were used to assess significant differences ($\alpha = 0.05$) between the four adult salmon groups ($\nu = 23$) and Tukey post hoc tests to assess which groups differed (results in italics). Student's *t* test was used to assess significant differences between fillet and rest of fish lipid percentages

Group:	Strait of Georgia smolts	Johnstone Strait adults 1	Lower Fraser River adults 2	Puget Sound smolts	Duwamish River adults 3	Deschutes River adults 4	ANOVA test (*Tukey test*)
No. of fish (*n* =)	6	6	6	12	6	6	NS[a]
Fork length (cm)	17.98 ± 1.08	88.70 ± 2.47	73.88 ± 3.13	6.90 ± 0.19	77.28 ± 2.39	78.23 ± 2.28	0.004 (*1–2; 1–3; 1–4*)
Body wt (kg)	0.08 ± 0.02	10.86 ± 1.07	6.08 ± 0.57	0.003 ± 0.0001	6.32 ± 0.57	5.95 ± 0.44	0.001 (*1–2; 1–3; 1–4*)
Age (years)	NA[b]	3.50 ± 0.22	2.50 ± 0.34	NA	2.33 ± 0.21	2.33 ± 0.21	0.010 (*1–2; 1–3; 1–4*)
Sex (male:female)	3:3	3:3	5:1	NA	3:3	3:3	0.607
Whole fish lipid (%)	0.87 ± 0.26	14.06 ± 1.37[c]	9.48 ± 0.62[c]	1.35[d]	6.38 ± 0.61	4.29 ± 0.82	0.000 (*1–2; 1–3; 1–4; 2–4*)
Fillet lipid (%)	NA	10.03 ± 1.42	5.37 ± 0.92	NA	NA	NA	0.016
Rest of fish lipid (%)		19.45 ± 1.44	12.84 ± 0.77		NA		0.002
Moisture (%)	74.93 ± 0.85	62.55 ± 1.31	66.54 ± 0.17	79.41	69.70 ± 0.65	71.66 ± 1.62	0.000 (*1–3; 1–4; 2–4*)
$\delta^{15}N$	13.68 ± 0.17	14.97 ± 0.23	15.51 ± 0.05	10.26 ± 0.42[d]	15.59 ± 0.24	15.54 ± 0.37	0.282
$\delta^{13}C$	−18.23 ± 0.24	−20.51 ± 0.35	−18.59 ± 0.27	−22.84 ± 0.26[d]	−18.94 ± 0.36	−18.11 ± 0.50	0.001 (*1–2; 1–3; 1–4*)

[a] NS = no significant difference.
[b] NA = not analyzed.
[c] Whole fish lipid percentage calculated using fillet lipid percentage and rest of fish lipid percentage.
[d] Data generated from a pooled sample of 12 individuals.

and spiked with a ^{13}C-labeled internal standard for instrumental analysis.

Instrumental analysis by HRGC-MS was carried out using a VG Autospec-Q magnetic sector mass spectrometer (Manchester, UK) coupled with a Hewlett-Packard 5890 Series II gas chromatograph (Palo Alto, CA, USA), a CTC A200S autosampler (Canboro, NC, USA), and Micromass OPUS data system. Chromatographic separation was achieved through an Agilent (Santa Clara, CA, USA) J&W DB-5 capillary chromatography column (60 m × 0.25 mm internal diameter × 0.25 μm film thickness). The mass spectrometer was operated at a resolution of 5,000 in selected ion monitoring mode using two intense ions for each analyte.

Number of congeners detected either as individual or coeluting congeners out of a theoretical possible number in 31 salmon samples were 135 out of 209 for PCBs, 3 out of 75 for PCDDs, 2 out of 135 for PCDFs, and in two muscle pools, 26 and 28 out of 44 for PBDEs, 34 and 34 out of 44 for PCDEs, and 12 and 12 out of 21 for PBBs. Detection limit substitutions were made for PCB, PCDD, PCDF, and OC pesticide analytes that were not detected when at least 70% of samples had detectable concentrations. Where more than 70% of samples did not have detectable concentrations of analytes, concentrations were not reported. For the two pooled samples analyzed for PBDE, PCDE, and PBB analytes, detection limit substitutions were made when one sample had a detectable concentration. Toxic equivalent quotients (TEQs) were calculated for PCBs, PCDDs, and PCDFs using World Health Organization International toxic equivalent factors for humans and wildlife [20].

Statistical analyses

For comparisons among adult chinook salmon groups single factor analysis of variance (ANOVA) was done. The degrees of freedom (ν) for ANOVA test were 23 (including numerator and denominator) for all analyses except for OC pesticides where ν was 17. Data met the assumptions of normality and homogeneity of variance, or were log-normalized. If significant differences ($\alpha = 0.05$) existed among the adult chinook groups, Tukey post hoc tests were done to determine which groups were significantly different from each other. For comparison between fillet and rest of fish lipid percentages student's *t* tests were used (equal variances). Since chinook smolt groups consisted of one group of six samples and one pooled sample a statistical comparison was not possible.

Body burden calculations

Estimates of POP body burdens for chinook salmon adults and smolts were determined from either concentrations from whole fish homogenates, or, in the case of Johnstone Strait and Lower Fraser River salmon, from fillet concentrations. We assumed that lipid-normalized POP concentrations would be equally partitioned between whole fish and fillet, as previously documented in salmon [21]. Lipid content was determined for Johnstone Strait and Lower Fraser River salmon using a weighted combination of fillet lipid values and rest of fish (ROF) lipid values as follows:

$$\text{mass lipid (whole fish)} = \left(\frac{\text{mass fillet}}{\text{mass whole fish}}\right)\cdot\%\text{lipid (fillet)} + \left(\frac{\text{mass ROF}}{\text{mass whole fish}}\right)\cdot\%\text{lipid (ROF)} \quad (2)$$

Body burden estimates were subsequently calculated using whole fish lipid percentages as follows:

$$\text{body burden (POPs)} = [\text{POP}]_{\text{lipid wt}} \cdot \text{mass lipid} \quad (3)$$

Principal components analysis

Principal components analysis (PCA) was used to characterize POP patterns among salmon and generate insight into the factors affecting them. Each PCB, dibenzo-*p*-dioxin, and dibenzofuran congener was evaluated for potential interferences, closeness to the limit of detection, and the percentage of undetectable (random value estimated) values. Borderline variables were tested in preliminary PCA models before inclusion in the final PCA data set, which included two dioxins (1,2,3,6,7,8-HxCDD and OCDD), two furans (2,3,7,8-TCDF and 2,3,4,7,8-PnCDF), and 130 PCBs (Appendix). Undetectable values (42 instances, or 1.31% of the data set; maximum of six undetectable values for 2,3,4,7,8-PnCDF and PCB188) were replaced by a random number between zero and the limit of detection, while the stated concentration was used for two values reported by the laboratory as not detected due to incorrect isotope ratio or NDR (peak detected but confirming-ion ratios outside of the specified range).

Samples were normalized to the concentration total to remove artifacts related to concentration differences between samples. The centered log-ratio transformation (division by the geometric mean of the concentration-normalized sample followed by log transformation) was then applied to this compositional data to produce a data set that was unaffected by negative bias or closure [7,22]. Data were then autoscaled (congeners were scaled by subtracting the variable mean and dividing by the variable standard deviation) to give every variable equal weight. Finally, a Varimax rotation was applied to the first three principal components (PCs) to simplify the physical interpretation of the PCA projections [7,23]. This rotation maximized or minimized the loading of each variable on each PC while preserving trends.

With $n = 24$ adult chinook and $p = 134$ contaminants, the PCA model provided a case where $n < p$ and the PCA model would be limited to $n - 1 = 23$ statistically valid eigenvectors [24]. The first few eigenvectors are little affected when the PCA data matrix is not of full rank and having $n < p$ does not lead to incorrect interpretations.

Linear relationships involving the PCA results were quantified using geometric mean (GM) linear regression [25,26]. The GM slope was calculated by dividing the y on x slope by the correlation coefficient for the regression, r [25]; the mean values for the x and y variables were then used to calculate the intercept for the GM regression equation. To estimate the relative shift in contaminant distribution for each sample we used the linear distance along the GM linear regression line for the fish samples, with the intersection point between the regression line and a perpendicular between the line and the sample position calculated using standard trigonometry mensuration formulae [23].

Dietary exposure calculations

As a means of characterizing health risks associated with dietary exposure we calculated estimated daily intakes (EDIs) of POPs by resident killer whales. Based on food consumption studies of captive killer whales, estimated intake as a function of body weight was calculated where food intake = 0.277 mass$^{0.663}$ [27]. We used this relationship to estimate food intake by a 2,500-kg adult killer whale and calculated EDIs for POPs with an assumption of 71.5% chinook consumption of a 96% salmonid diet [2]. Given the limited information on nonsalmon prey items in the diet of resident killer whales, we restrict our exercise here to their dominant prey item (chinook).

$$\text{Daily food intake for a 2,500 kg killer whale} = 0.277\ \text{mass}^{0.663} \quad (4)$$

$$\text{Salmonid portion of diet} = 96\% \cdot 50\ \text{kg/d} \quad (5)$$

$$\text{Chinook portion of diet} = 71.5\% \cdot 48\ \text{kg/d} \quad (6)$$

$$\text{EDI } (\mu\text{g/d}) = [\text{POP}]_{\text{wet wt}} \cdot 34\ \text{kg/d} \quad (7)$$

RESULTS AND DISCUSSION

As their primary prey item, chinook salmon provide both a source of nutrition and contaminants to northern and southern resident killer whales. The highly contaminated southern resident killer whales frequent the near-urban waters of the Strait of Georgia and Puget Sound, while northern resident killer whales ply the more remote waters of central and northern British Columbia. While logistical and ethical challenges preclude an accurate evaluation of dietary exposure to POPs by killer whales, we can estimate dietary POP exposure in these killer whales using data from chinook salmon.

Life history and feeding ecology of chinook

Since chinook salmon is the primary prey of killer whales, an understanding of their life history and feeding ecology is important to exploring issues related to exposure and bioaccumulation in the killer whale food web. Stock identification assigned at least 67% of the adults collected from Johnstone Strait to the Thompson River region and 83% of the adults collected from the mouth of the Fraser River to the Lower Fraser River region (with Harrison River being the most probable population). Harrison River stock is known to be predominantly coastal in its marine distribution, being found on the west coast of Vancouver Island, the Strait of Georgia and Washington waters, whereas Johnstone Strait (Thompson River stock) are known to migrate into the northern waters of British Columbia and the Gulf of Alaska [28] (www-comm.pac.dfo-mpo.gc.ca/publications/speciesbook/Salmon/chinook.fraser.html). All six chinook smolts sampled in the Strait of Georgia originated from eastern Vancouver Island, with five from Big Qualicum River and one from Little Qualicum River. Stock identification, $\delta^{13}C$, $\delta^{15}N$, morphometric, and meristic data provide an overview of the biology and ecology of sampled chinook salmon (Table 1).

Scale data from our chinook samples indicated that all adults migrated to marine waters during their first year of life with the exception of three British Columbia adults (two Johnstone Strait and one Lower Fraser River) that spent one year in freshwater before going to sea. Although scale data indicated that Duwamish and Deschutes river stocks migrated to marine waters during their first year of life, some fish from these populations are known to be resident stock that remain in Puget Sound waters year-round without migrating into open ocean [9].

A significant difference in $\delta^{13}C$ ratios was observed among adult stocks (one-way ANOVA, $\nu = 23$, $p = 0.001$) and lipid-normalizing the $\delta^{13}C$ ratios [29] did not statistically affect $\delta^{13}C$ ratios among adults ($r^2 = 0.24$, $\nu = 23$, $p = 0.0123$). However, no significant difference was apparent in $\delta^{15}N$ ratios among adults, suggesting similarities in trophic level (Table 1). Our

Table 2. Wet weight-based concentrations of persistent organic pollutants and toxic equivalents (TEQs) to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) for polychlorinated biphenyls (PCBs), polychlorinated dibenzo-*p*-dioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs) in returning adult chinook salmon (*Oncorhynchus tshawytscha*) from Johnstone Strait ($n = 6$) and Lower Fraser River ($n = 6$) (British Columbia, Canada); Duwamish River ($n = 6$) and Deschutes River ($n = 6$) (Washington, USA); and chinook smolts ($n = 6$) from the Strait of Georgia (British Columbia, Canada) and Puget Sound ($n = 1$ pool of 12) (Washington, USA). Values represent mean ± standard error of the mean. One-way analysis of variance (ANOVA) tests were used to assess significant differences ($\alpha = 0.05$) between the four adult salmon groups ($\nu = 23$) and Tukey post hoc tests to assess which groups differed (results in italics)

Sum congeners/isomers[a] Group:	μg/kg wet weight (except for ΣPCDD and ΣPCDF)[b]						
	SG Smolts	Johnstone Strait 1	Lower Fraser River 2	PS Smolts	Duwamish River 3	Deschutes River 4	ANOVA test *(Tukey test)*
Lipid (%)[c]	0.87 ± 0.26	10.03 ± 1.42	5.37 ± 0.92	1.35	6.38 ± 0.61	4.29 ± 0.82	*(refer to Table 1)*
ΣPCB[d]	12.03 ± 1.46	9.07 ± 1.49	46.97 ± 8.06	9.63	34.61 ± 8.09	56.09 ± 17.97	0.001 *(1-2; 1-3; 1-4)*
ΣPCB TEQ	0.30 ± 0.04	0.17 ± 0.03	0.74 ± 0.11	0.28	0.55 ± 0.12	1.09 ± 0.35	0.007 *(1-2; 1-4)*
ΣPCDD[d] (ng/kg)	1.39 ± 0.32	0.58 ± 0.05	0.81 ± 0.14	1.57	0.83 ± 0.15	1.74 ± 0.63	0.011 *(1-4)*
ΣPCDD TEQ	0.35 ± 0.10	0.03 ± 0.02	0.27 ± 0.08	0.00	0.12 ± 0.04	0.31 ± 0.05	0.00 *(1-2; 1-3; 1-4)*
ΣPCDF[d] (ng/kg)	2.03 ± 0.48	0.50 ± 0.12	1.90 ± 0.38	0.26	1.30 ± 0.24	1.92 ± 0.31	0.000 *(1-2; 1-3; 1-4)*
ΣPCDF TEQ	0.24 ± 0.06	0.06 ± 0.02	0.28 ± 0.05	0.00	0.11 ± 0.04	0.25 ± 0.06	0.007 *(1-2; 1-4)*
ΣPBDE[e]	NA[f]	NA	17.71	NA	6.43	NA	ND[g]
ΣPCDE[e]	NA	NA	0.53	NA	0.24	NA	ND
ΣPBB[e]	NA	NA	0.10	NA	0.04	NA	ND
ΣTEQs	0.89 ± 0.20	0.26 ± 0.06	1.30 ± 0.19	0.28	0.78 ± 0.18	1.65 ± 0.44	0.006 *(1-2; 1-4)*

[a] PCB = polychlorinated biphenyl; PBDE = polybrominated diphenyl ether; PCDE = polychlorinated diphenyl ether; PBB = polybrominated biphenyl; PCDD = polychlorinated dibenzo-*p*-dioxin; PCDF = polychlorinated dibenzofuran.
[b] SG = Strait of Georgia; PS = Puget Sound.
[c] Whole fish percentage lipid for SG smolts, PS smolts, Duwamish and Deschutes river adults; fillet percentage lipid for Johnstone Strait and Lower Fraser River adults.
[d] Whole fish analyzed for SG smolts, PS smolts, Duwamish and Deschutes river adults; fillet analyzed for Johnstone Strait and Lower Fraser River adults.
[e] Pooled fillet ($n = 1$ pool of 6 fish) analyzed.
[f] NA = not analyzed.
[g] ND = statistical comparison not possible.

chinook $\delta^{15}N$ ratios and $\delta^{13}C$ ratios decreased with lipid % in whole fish ($r^2 = 0.18$, $\nu = 23$, $p = 0.0404$ and $r^2 = 0.59$, $\nu = 23$, $p < 0.0001$, respectively) (data not shown). Previous studies have demonstrated that $\delta^{13}C$ and $\delta^{15}N$ ratios can be affected by the nutritional status of organisms [30–32] and that a range of at least 4 to 6‰ should be expected in both $\delta^{13}C$ and $\delta^{15}N$ values during the migration of salmon that is due to changes in lipid and protein concentrations [30]. Therefore, enrichment in $\delta^{13}C$ and $\delta^{15}N$ ratios with decreasing lipid content in our chinook salmon likely reflects the migration-associated influence of declining lipid stores on $\delta^{13}C$ and $\delta^{15}N$ values, rather than trophic level or feeding ecology. This is consistent with changes in physiological condition in chinook salmon as they near their natal streams.

Contaminant concentrations in chinook salmon

Significant differences in PCB, PCDD, and PCDF concentrations, on a wet weight basis, were observed among adult salmon ($\nu = 23$, $p = 0.001$, $p = 0.011$, $p = 0.000$, respectively), with Johnstone Strait salmon having the lowest concentrations and Deschutes River salmon having the highest concentrations (Table 2). Polychlorinated biphenyls were the dominant POP detected in all salmon sampled, including smolts. Two of the six adult chinook sampled from each of the Lower Fraser, Duwamish, and Deschutes rivers exceeded mean PCB concentrations found in a previous study where Puget Sound returning chinook, collected from either near-shore estuaries or river locations had a detected mean value of 49.1 μg/kg wet weight [9].

Significant differences in OC pesticides were observed among the adult salmon stocks (Table 3). Duwamish River salmon had the highest concentrations of all OC pesticides with the exception of HCH compounds. Total DDT dominated the OC pesticide rankings among both British Columbia and Washington smolts and three out of the four returning adult groups (Table 3), while total HCH dominated the OCs in Johnstone Strait adults. Although DDT and HCH appear to be the dominant OC pesticides detected in both British Columbia and Washington salmon, their isomeric compositions may reflect differences in distance from source or use regions. The contribution of the DDT degradation products (ΣDDE and ΣDDD) in all chinook samples was 88 to 97% of the ΣDDT, suggesting fresh input to be minimal. The high concentrations of the predominant parent α-HCH isomer, the most bioaccumulative isomer β-HCH, and lower concentrations of the insecticide γ-HCH are apparent as one moves away from source/use regions, reflecting partitioning properties which favor colder, more northerly waters [33]. The volatility of HCH ensures its ready atmospheric transport from Asia to the northeastern Pacific Ocean via prevailing winds [34].

Of the two adult chinook pools analyzed for PBDEs, the most predominant congeners detected were BDE-47 and BDE-99, respectively. The PBDE profile for Lower Fraser River chinook was 47 > 99 > 100 > 49 > 209, and for Duwamish River chinook was 47 > 99 > 100 > 49 > 120. Similar congener profiles have been observed in chinook from Oregon (BDE-47 > 99 > 100 > 49 > 154) [35] and in Lake Michigan salmonids (BDE-47 > 99 > 100 > 154 > 153) [36]. The ratio of PBDE to PCB concentrations were 0.4:1 for the Lower Fraser River adults and 0.2:1 for the Duwamish River adults, highlighting the emergence of PBDEs as a significant environmental contaminant.

Significant differences in ΣPCB TEQs, ΣPCDD TEQs, ΣPCDF TEQs, and ΣTEQs were observed among the four adult

Table 3. Wet weight-based concentrations of organochlorine pesticides in returning adult chinook salmon (*Oncorhynchus tshawytscha*) from Johnstone Strait (n = 6) and Lower Fraser River (n = 6) (British Columbia, Canada); Duwamish River (n = 6) and chinook smolts (n = 6) from the Strait of Georgia (British Columbia, Canada) and Puget Sound (n = 1 pool of 12) (Washington, USA). Values represent mean ± standard error of the mean. One-way analysis of variance (ANOVA) tests were used to assess significant differences (α = 0.05) between the three adult salmon groups (ν = 17) and Tukey post hoc tests to assess which groups differed (results in italics)

	μg/kg wet weight[a]						
Sum congeners/ isomers[b] Group:	SG Smolts[c]	Johnstone Strait[d] 1	Lower Fraser River[d] 2	PS Smolts[c]	Duwamish River[c] 3	Deschutes River[c]	ANOVA test *(Tukey test)*
Lipid (%)	0.87 ± 0.26	10.03 ± 1.42	5.37 ± 0.92	1.35	6.38 ± 0.61	4.29 ± 0.82	*(refer to Table 1)*
ΣDDT	4.38 ± 0.55	1.46 ± 0.27	4.29 ± 0.50	2.68	18.31 ± 3.94	NA[e]	(0.000) *(1-2; 1-3; 2-3)*
o,p′-DDT	*0.05 ± 0.01*	*0.07 ± 0.02*	*0.04 ± 0.00*	*0.02*	*0.12 ± 0.01*	NA	(0.014) *(2-3)*
p,p′-DDT	*0.27 ± 0.04*	*0.10 ± 0.02*	*0.22 ± 0.03*	*0.10*	*0.40 ± 0.08*	NA	(0.000) *(1-2; 1-3)*
o,p′-DDD	*0.06 ± 0.01*	*0.07 ± 0.01*	*0.06 ± 0.01*	*0.01*	*0.21 ± 0.02*	NA	(0.001) *(1-3; 2-3)*
p,p′-DDD	*0.40 ± 0.08*	*0.25 ± 0.03*	*0.60 ± 0.05*	*0.13*	*2.88 ± 0.50*	NA	(0.000) *(1-2; 1-3; 2-3)*
o,p′-DDE	*0.08 ± 0.02*	*0.07 ± 0.01*	*0.04 ± 0.00*	*0.02*	*0.12 ± 0.02*	NA	(0.000) *(1-2; 1-3; 2-3)*
p,p′-DDE	*3.52 ± 0.43*	*0.90 ± 0.21*	*3.34 ± 0.42*	*2.40*	*14.58 ± 3.39*	NA	(0.000) *(1-2; 1-3; 2-3)*
(ΣDDE + ΣDDD) ÷ ΣDDT	0.93 ± 0.00	0.88 ± 0.01	0.94 ± 0.00	0.95	0.97 ± 0.01	NA	(0.002) *(1-2; 1-3)*
HCB	0.36 ± 0.06	1.50 ± 0.16	0.85 ± 0.09	0.29	2.15 ± 0.12	NA	0.000 *(1-2; 1-3; 2-3)*
ΣHCH (α-, β-, γ-)	1.09 ± 0.25	2.28 ± 0.23	0.68 ± 0.14	0.27	1.60 ± 0.20	NA	0.000 *(1-2; 1-3; 2-3)*
alpha (α-)	*0.32 ± 0.08*	*0.98 ± 0.10*	*0.25 ± 0.05*	*0.08*	*0.84 ± 0.10*	NA	0.000 *(1-2; 2-3)*
beta (β-)	*0.34 ± 0.08*	*1.09 ± 0.11*	*0.37 ± 0.08*	*0.10*	*0.63 ± 0.08*	NA	0.000 *(1-2; 1-3)*
gamma (γ-)	*0.43 ± 0.10*	*0.21 ± 0.02*	*0.06 ± 0.01*	*0.08*	*0.12 ± 0.03*	NA	0.005 *(1-2; 1-3)*
Heptachlor	<DL[f]	<DL	<DL	<DL	<DL	NA	ND
Aldrin	<DL	<DL	<DL	<DL	<DL	NA	ND
Chlordane (*oxy-*, γ-, α-)	0.78 ± 0.14	0.68 ± 0.04	0.62 ± 0.06	0.43	1.90 ± 0.14	NA	0.000 *(1-3; 2-3)*
oxy-	*0.44 ± 0.11*	*0.14 ± 0.00*	*0.14 ± 0.01*	*0.31*	*0.49 ± 0.08*	NA	0.018 *(1-3; 2-3)*
gamma (γ-)*trans*	*0.13 ± 0.02*	*0.10 ± 0.01*	*0.07 ± 0.01*	*0.05*	*0.22 ± 0.02*	NA	0.000 *(1-2; 1-3; 2-3)*
alpha (α-)*cis*	*0.21 ± 0.04*	*0.43 ± 0.03*	*0.42 ± 0.04*	*0.07*	*1.18 ± 0.11*	NA	0.003 *(1-3; 2-3)*
ΣChlordanes[g]	1.41 ± 0.20	1.47 ± 0.07	1.6 ± 0.16	0.84	4.75 ± 0.38	NA	0.003 *(1-3; 2-3)*
ΣNonachlor (*trans-*, *cis-*)	0.58 ± 0.10	0.64 ± 0.03	0.87 ± 0.10	0.27	2.53 ± 0.25	NA	0.002 *(1-3; 2-3)*
trans-	*0.41 ± 0.07*	*0.46 ± 0.02*	*0.63 ± 0.07*	*0.22*	*1.87 ± 0.17*	NA	0.002 *(1-3; 2-3)*
cis-	*0.17 ± 0.03*	*0.18 ± 0.01*	*0.25 ± 0.03*	*0.05*	*0.67 ± 0.09*	NA	0.002 *(1-3; 2-3)*
Mirex	0.05 ± 0.01	0.02 ± 0.00	0.02 ± 0.00	0.03	0.06 ± 0.01	NA	0.003 *(1-3; 2-3)*
Heptachlor epoxide	0.05 ± 0.02	0.14 ± 0.01	0.09 ± 0.01	0.02	0.28 ± 0.04	NA	0.000 *(1-2; 1-3; 2-3)*
Endosulphan, *alpha* (α-)	<DL	<DL	<DL	<DL	<DL	NA	ND[h]
Dieldrin	0.16 ± 0.04	0.45 ± 0.02	0.64 ± 0.06	0.03	0.75 ± 0.11	NA	0.012 *(1-3)*
Endrin	0.02 ± 0.01	0.08 ± 0.01	0.06 ± 0.01	0.06	0.38 ± 0.07	NA	0.002 *(1-3; 2-3)*
Methoxychlor	<DL	<DL	<DL	<DL	<DL	NA	ND

[a] SG = Strait of Georgia; PS = Puget Sound.
[b] DDT = dichlorodiphenyltrichloroethane; DDD = dichlorodiphenyldichloroethane; DDE = dichlorodiphenyldichloroethylene; HCB = hexachlorobenzene; HCH = hexachlorocyclohexane.
[c] Whole fish analyzed.
[d] Fillet analyzed.
[e] ΣChlordanes = heptachlor + heptachlor epoxide + oxychlordane + *cis-* and *trans*-chlordane + *cis-* and *tran*s-nonachlor.
[f] NA = not analyzed.
[g] <DL = less than detection limit.
[h] ND = statistical comparison not possible.

salmon stocks (Table 2). Although PCBs explained the majority of ΣTEQs in the adult salmon groups, Lower Fraser River adults had a significantly lower Σplanar PCB TEQ contribution to the ΣTEQ compared with the Johnstone Strait and Duwamish River adults (ν = 23; p = 0.007 and p = 0.011, respectively). This in part is due to the higher ΣPCDD contribution to the ΣTEQ in the latter samples. While this may in part reflect differences in dietary exposure for the different stocks, metabolic removal or preferential elimination of the planar PCBs may also explain this observation. Polychlorinated biphenyls made up 100% of the ΣTEQs in the Puget Sound smolts, whereas in the Strait of Georgia the ΣPCDD and ΣPCDF TEQs made up a greater proportion of the ΣTEQs. These results are consistent with those found in our Puget Sound and Strait of Georgia harbor seal food baskets, likely reflecting differences in regional source inputs between pulp mills in the Strait of Georgia and more industries in Puget Sound [17].

POP origin in returning adult salmon

By comparing body burdens of POPs in returning adult chinook to out-migrating smolts and juveniles, we estimate that 97 to 99% of the body burden of PCBs, PCDDs, PCDFs, DDT, and HCH in all stocks originated during their time at sea (Table 4). Field sampling provided us with salmon that could be identified only after genetic analysis. As a result of differences in stock identification between some smolts and adults, stock-specific assignment of the POPs in adults was not directly possible. Our estimation that the majority of POPs in chinook salmon can be ascribed to their growth stage in coastal and marine waters is consistent with other studies. A study of chinook from Washington ascribed 99% of PCBs in returning Duwamish River adults to the waters of Puget Sound and the Pacific Ocean [9]. The concentrations of POPs detected in our smolts are comparable to values previously reported in outmigrating chinook salmon smolts from a number of stocks

Table 4. Estimated body burdens of persistent organic pollutants in returning adult chinook salmon (*Oncorhynchus tshawytscha*) from Johnstone Strait (n = 6) and Lower Fraser River (n = 6) (British Columbia, Canada); Duwamish River (n = 6) and Deschutes River (n = 6) (Washington, USA); and chinook smolts (n = 6) from the Strait of Georgia (British Columbia, Canada) and Puget Sound (n = 1 pool of 12) (Washington, USA). Body burden estimates calculated using POP lipid weight concentrations and whole fish lipid mass. Values represent mean ± standard error of the mean. One-way analysis of variance (ANOVA) tests were used to assess significant differences (α = 0.05) between the four adult salmon groups (ν = 23) and three adult groups for total dichlorodiphenyltrichloroethane (ΣDDT) and total hexachlorocyclohexane (ΣHCH) (ν = 17). Tukey post hoc tests were used to assess which groups differed (results in italics)

Sum congeners/ isomers[a]	Body burden (μg) (except for ΣPCDD and ΣPCDF)						
Group:	Strait of Georgia smolts	Johnstone Strait adults 1	Lower Fraser River adults 2	Puget Sound smolts	Duwamish River adults 3	Deschutes River adults 4	ANOVA test *(Tukey test)*
ΣPCBs as % returning burden	1.12 ± 0.40	141.54 ± 30.48 99.21%	537.58 ± 99.01 99.79%	0.03	216.32 ± 56.88 99.99%	339.62 ± 108.82 99.99%	0.017 *(1-2)*
ΣPCDDs (ng) as % returning burden	0.13 ± 0.04	8.99 ± 1.44 98.56%	10.14 ± 3.18 98.72%	0.005	5.50 ± 1.34 99.91%	9.76 ± 3.07 99.95%	0.194
ΣPCDFs (ng) as % returning burden	0.21 ± 0.10	8.07 ± 2.30 97.40%	23.94 ± 8.05 99.12%	0.001	8.21 ± 1.87 99.99%	11.48 ± 2.01 99.99%	0.018 *(1-2; 2-3)*
ΣPBDEs	NA[b]	NA	165.75	NA	54.34	NA	ND[c]
ΣDDT[d] as % returning burden	0.15 ± 0.08	24.11 ± 6.30 99.38%	44.46 ± 8.00 99.66%	0.01	107.96 ± 28.08 99.99%	NA	0.003 *(1-3)*
ΣHCH[e] as % returning burden	0.04 ± 0.02	35.01 ± 3.74 99.88%	6.68 ± 1.21 99.40%	0.001	9.32 ± 1.55 99.99%	NA	0.000 *(1-2; 1-3)*

[a] PCB = polychlorinated biphenyl; PCDD = polychlorinated dibenzo-*p*-dioxin; PCDF = polychlorinated dibenzofuran; PBDE = polybrominated diphenyl ether; DDT = dichlorodiphenyltrichloroethane; DDD = dichlorodiphenyldichloroethane; DDE = dichlorodiphenyldichloroethylene; HCH = hexachlorocyclohexane.

[b] NA = not analyzed.

[c] ND = statistical comparison not possible.

[d] ΣDDT includes DDT (*o,p′*-DDT, *p,p′*-DDT), DDD (*o,p′*-DDD, *p,p′*-DDD), and DDE (*o,p′*-DDE, *p,p′*-DDE).

[e] ΣHCH includes (α-, β-, γ-) HCH.

in Washington and Oregon [37], further underscoring the limited contribution of locally acquired contaminants during the juvenile stage. It is increasingly clear that salmon acquire the majority POPs during their growth period at sea and that more research is needed on the extent of Pacific Ocean food web contamination.

Lipid-normalized ΣPCB and ΣDDT concentrations increased with $\delta^{15}N$ ratios among adult chinook ($r^2 = 0.31$, $\nu = 23$, $p = 0.0046$ and $r^2 = 0.46$, $\nu = 17$, $p = 0.0020$, respectively), as did ΣPCB and ΣDDT body burdens ($r^2 = 0.25$, $\nu = 23$, $p = 0.0306$ and $r^2 = 0.34$, $\nu = 17$, $p = 0.155$, respectively) (results not shown). While our observed relationship between these POPs and $\delta^{15}N$ could be interpreted as reflecting an influence of trophic level, it may also signal an effect of migration-associated lipid changes. Changes in tissue concentrations of lipid and protein in migrating salmon complicate this interpretation of stable isotope-defined trophic level assignment [30].

Contaminant patterns in adult chinook

In the present study the primary purpose of PCA modeling is to quantitatively compare the contaminant distributions between different adult chinook populations. Because the PCA algorithm uses the variable magnitudes when decomposing the data set into a series of orthonormal rank 1 matrices or PCs, the substantial concentration differences between populations (Table 2) have to be removed by normalizing each sample before PCA. The difficulty is that this normalization step introduces closure (spurious negative correlations in the highest variables and negative correlations in the smallest). Centered log ratio transformation removes this closure and produces a data set where the average concentration and concentration total are identical for every sample [22,23]. In the PCA model the first two PCs account for the largest percentage of total variance in the data set and, particularly when data are normalized, reflect the most discriminating compositional features. The contaminants with variable loadings near axis center have essentially no contribution to a PC, while the contribution to a PC increases as the absolute magnitude of the variable loading.

Principal component analyses differentiated adult chinook on the basis of variation in PCB, PCDD, and PCDF congener proportions (Fig. 2a). In the Varimax rotated PCA model, chinook salmon samples project along a line from the upper left to the lower right of the samples plot (Fig. 2a). Because both variables (the PCs) in this relationship between chinook samples are affected by natural variability, rather than just measurement error, the appropriate regression line to use quantify the relationship is GM linear regression [23,25,26].

Geometric mean linear regression for the sample projections of chinook samples indicates that this linear relationship in Figure 2a is highly significant ($r^2 = 0.840$, $\nu = 22$, $p = 3.1 \times 10^{-10}$). In the corresponding variables plot, most PCDD, PCDF, and lower chlorine number PCB congeners project in the upper left quadrant while the higher chlorine number PCB congeners project in the lower right quadrant (Fig. 2b). Geometric mean regression for the variables also indicates that this linear relationship is highly significant ($r^2 = 0.377$, $\nu = 132$, $p = 3.1 \times 10^{-15}$), despite the greater amount of scatter in the variables plot. Comparison of samples and variables indicates that salmon samples projecting towards the upper left of the samples plot have higher proportions of the PCDD, PCDF, and lower chlorine number and non- and mono-*ortho* PCB congeners while samples projecting on the lower right have higher proportions of the higher chlorine number, di-*ortho* PCB congeners.

The differences in contaminant composition are not obviously related to either sex or sampling location (urban vs remote, or BC vs Puget Sound) for the salmon samples (Fig. 2a). The shifts in contaminant composition along the GM regression line for the samples (Fig. 2c) correlated with lipid content ($r^2 = 0.328$, $p = 0.0034$), $\delta^{13}C$ composition ($r^2 = 0.605$, $p = 7.7 \times 10^{-6}$), and body weight ($r^2 = 0.214$, $p = 0.0227$), but are not significantly related to $\delta^{15}N$ ($r^2 = 0.152$, $p = 0.0596$, $\nu = 22$ in all cases). Accordingly, the change in contaminant composition for the salmon appears to reflect metabolism or solubilization of the PCDD and PCDF and lower chlorine number and non- and mono-*ortho*-PCB congeners as the salmon lose lipid during migration. This suggests that the migrating salmon PCB burdens will be increasingly dominated by the more heavily chlorinated congeners. Similar observations in sockeye salmon were thought to reflect a greater metabolic capacity by salmonids for PCDDs and PCDFs as compared to PCBs [11]. While our results support the notion of compositional loss associated with depleting lipid reserves during migrating salmon, a contribution of local POP sources from more contaminated areas, such as Puget Sound, cannot be ruled out [5,6]. Indeed, feeding in such an area during outward- and inward-bound migrations likely does lead to increased POP concentrations in certain salmon individuals and stocks.

While our results suggest that salmon accumulate the majority of POPs during their growth period at sea, lipid depletion and metabolism in salmon associated with migration may have profound consequences for dietary exposure to POPs in resident killer whales. While both northern and southern resident killer whales preferentially consume chinook salmon, southern residents likely intercept more chinook in relatively contaminated, near-urban areas and at points closer to their natal streams. Southern residents may therefore be consuming chinook salmon that is both more contaminated and less lipid-rich.

Health risks for killer whales

Dietary POP concentrations and patterns have profound implications for killer whale POP accumulation and consequent related health risks. High trophic level marine mammals have shown susceptibility to adverse health effects such as immunotoxicity, endocrine disruption, reproductive impairment, and developmental abnormalities with elevated exposure to POPs [1]. To characterize health risks in killer whales associated with dietary exposure to POPs, chinook ΣPCB, ΣPCDD/PCDF TEQ, and ΣDDT concentrations were compared with Canadian Council of Ministers of the Environment (CCME) tissue residue guidelines for the protection of mammalian wildlife consumers of aquatic biota [38] (www.ccme.ca/assets/pdf/trg_summary_table.pdf). The Deschutes River salmon exceeded, and the Lower Fraser River salmon were approaching (Table 2), the CCME PCB tissue residue guidelines (0.79 ng TEQ/kg diet wet wt) [38]. The Duwamish River salmon (Table 2) exceeded the ΣDDT tissue residue guidelines (14.0 μg/kg diet wet wt) [38].

The ΣPCB and ΣDDT concentrations in all salmon groups were below the less conservative U.S. guidelines (New York State Department of Environmental Conservation) for protection of fish-eating wildlife [39]. All of the chinook analyzed

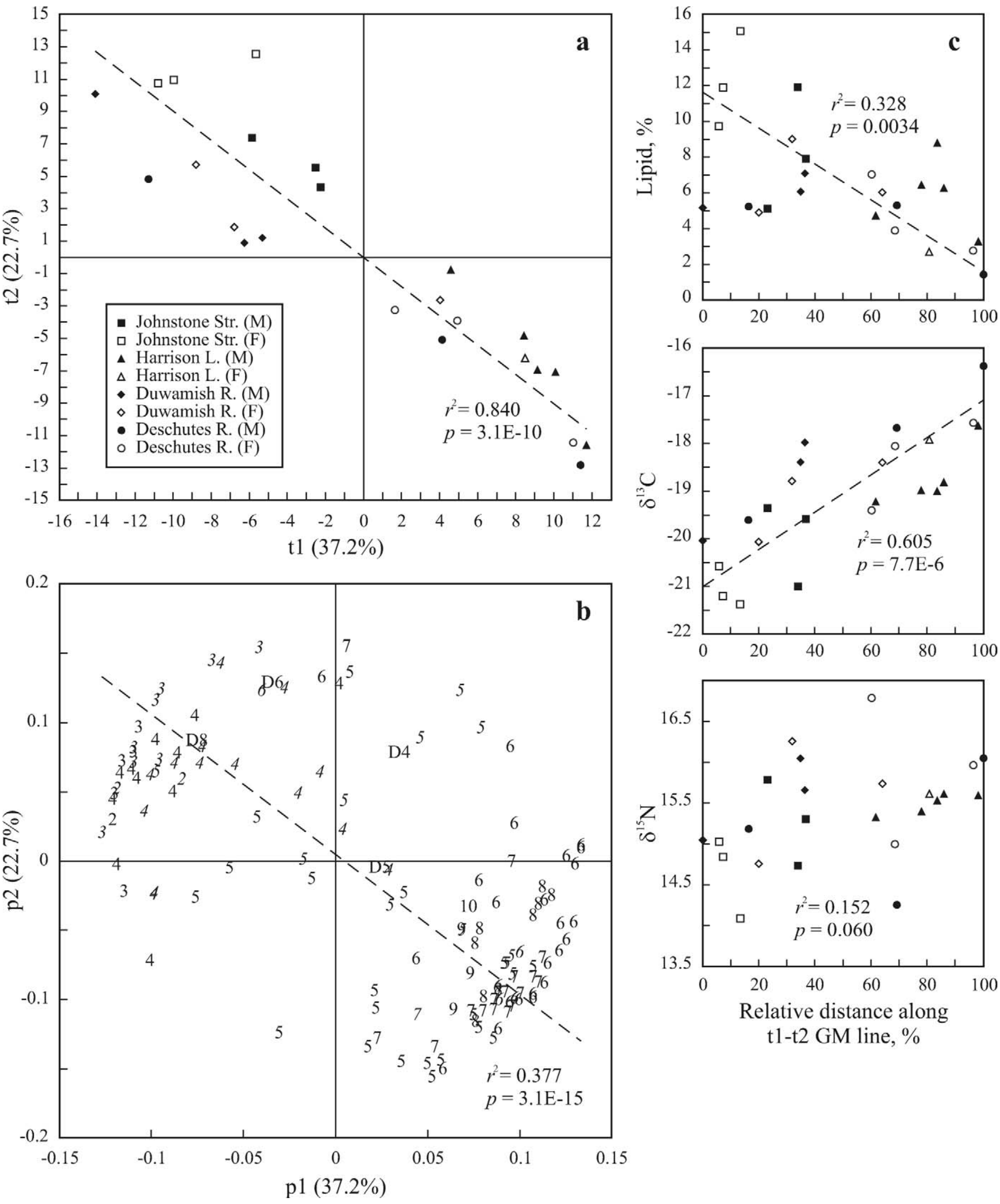

Fig. 2. Contaminant patterns in chinook salmon are relevant to assessing the influence of feeding ecology in chinook and dietary exposure of persistent organic pollutants in resident killer whales. Varimax rotated projections of the first two principal components (PCs) for a principal components analysis model based on normalized concentrations (see text) showing (**a**) chinook salmon scores (t1 and t2) by sampling location and sex (M = male; F = female), and (**b**) dibenzo-*p*-dioxin (PCDD), dibenzofuran (PCDF), and polychlorinated biphenyl (PCB) congener variable loadings (p1 and p2) by chlorine number. In (**b**) PCDDs and PCDFs have a ''D'' preceding the number, the dioxin-like non-*ortho* and mono-*ortho*-PCBs are in italics and di-*ortho*-PCBs use a regular font. In (**c**) the lipid, $\delta^{13}C$ and $\delta^{15}N$ content is plotted by sampling location and sex against the relative distance along the GM linear regression line in the first PC for the salmon samples (**a**).

Table 5. Estimated daily intake (EDI) of persistent organic pollutants (POPs) by northern and southern resident killer whales. Johnstone Strait (British Columbia, Canada) chinook POP concentrations have been used to calculate EDIs for northern residents and all four chinook stocks for southern residents. We have further calculated the EDI for southern residents if they were to consume chinook of equivalent lipid content to that of northern residents, i.e., if northern residents were to consume 34 kg chinook per day (8.5% lipid); southern residents may consume up to 85 kg chinook per day (3.4% lipid)

	Estimated daily intake μg/d (except for ΣPCDD, ΣPCDF, and ΣTEQ ng/d)		
Sum congeners/isomers[a]	Northern residents	Southern residents	Southern residents (lipid-equivalent)
ΣPCB	308.49	1,248.00	2,051.38
ΣPBDE	No data	410.62	674.95
ΣPCDD	19.75	33.67	55.34
ΣPCDF	17.17	47.82	78.60
ΣDDT (DDT, DDD, DDE)	49.72	272.88	448.55
HCH (α-, β-, γ-)	77.76	51.74	85.05
ΣTEQ (PCB, PCDD, PCDF)	9.00	36.85	60.58

[a] PCB = polychlorinated biphenyl; PBDE = polybrominated diphenyl ether; PCDE = polychlorinated biphenyl ether; PBB = polybrominated biphenyl; PCDD = polychlorinated dibenzo-*p*-dioxin; PCDF = polychlorinated dibenzofuran; TEQ = toxic equivalents; DDT = dichlorodiphenyltrichloroethane; DDD = dichlorodiphenyldichloroethane; DDE = dichlorodiphenyldichloroethylene; HCB = hexachlorobenzene; HCH = hexachlorocyclohexane.

in the present study exceeded the 8 μg/kg dietary PCBs that was estimated to protect 95% of a killer whale population, based on a 17 mg/kg PCBs adverse effects threshold for marine mammals [40].

Another means of characterizing health risks associated with dietary exposure is through the calculation of EDI. Based on food intake estimates derived from studies of captive killer whales [27], we have estimated the food intake of a 2,500-kg resident killer whale to be approximately 50 kg per day. We have further estimated the chinook portion, 71.5% of a 96% salmonid diet [2], to be 34 kg/d. Taking into account observed ranges for resident killer whales [41], POP concentrations (wet wt) for Johnstone Strait chinook were used to calculate EDIs for northern residents and all four chinook stocks for southern residents (Table 5).

Our EDIs suggest that southern residents may be consuming, on a body weight basis, 4.0 times more PCBs than their northern counterparts, consistent with the differences in PCB concentrations measured in biopsies collected from free-ranging northern and southern resident killer whales [1]. However, since studies of marine mammal energetics suggest that calorimetric content is an integral component of food needs [27,42], we have also adjusted consumption to reflect equivalent lipid content. Because of the lower lipid content of our more southerly chinook salmon, there may be a compensatory increase in consumption by southern resident killer whales. This nutritionally adjusted scenario would predict that southern residents would consume 6.6 times more PCBs than northern residents. Similarly, we previously speculated that Puget Sound harbor seals consume nearly twice as much prey as Strait of Georgia seals in order to compensate for the lower lipid content in their prey, with results explaining a near-doubling of their contaminant burden [17]. Additional studies on killer whale feeding ecology and on the behavior of POPs in salmon during different life history stages will shed more insight into the sources and fate of contaminants in killer whale food webs.

The present study underscores the global nature of contaminant dispersion with chinook salmon acquiring the majority of their POPs during their time at sea. As the two resident killer whale populations in British Columbia intercept these returning salmon, they are exposed to different dietary POP concentrations. We conclude that the endangered southern resident killer whales are exposed to much higher concentrations of POPs than their northern counterparts through the consumption of more POP-contaminated chinook salmon, and may increase their consumption of salmon in order to compensate for the reduced lipid content observed in southerly chinook. In this regard, increasing climate-related stresses on salmon abundance and lipid content raise the specter of increased contaminant exposures for resident killer whales in the future.

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APPENDIX 1. Principal components analysis variable

Variable[a]	PCB substitution	Chlorine No.	No. <	Variable	PCB substitution	Chlorine No.	No. <
Dibenzo-*p*-dioxins and dibenzofurans				PCB94	22′356′	5	1
1,2,3,6,7,8-		6	—	PCB102/93	22′456′/22′356	5	—
OCDD		8	—	PCB95	22′35′6	5	—
2,3,7,8-TCDF		4	—	PCB88	22′346	5	—
2,3,4,7,8-PnCDF		5	6	PCB91	22′34′6	5	—
Non-*ortho* PCBs				PCB92/84	22′355′/22′33′6	5	—
PCB11	33′	2	—	PCB89	22′346′	5	—
PCB15	44′	2	—	PCB101/90	22′455′/22′34′5	5	—
PCB35	33′4	3	2	PCB99	22′44′5	5	—
PCB37	344′	3	—	PCB119	23′44′6	5	—
PCB80	33′55′	4	—	PCB109/83	233′46/22′33′5	5	—
PCB79	33′45′	4	—	PCB97/86	22′3′45/22′345	5	—
PCB81	344′5	4	—	PCB115/87	2344′6/22′345′	5	—
PCB77	33′44′	4	—	PCB85	22′344′	5	—
PCB126	33′44′5	5	—	PCB110	233′4′6	5	—
PCB169	33′44′55′	6	—	PCB82	22′33′4	5	—
Mono-*ortho* PCBs				PCB155	22′44′66′	6	2
PCB26	23′5	3	—	PCB150	22′34′66′	6	4
PCB25	23′4	3	—	PCB148	22′34′56′	6	2
PCB31	24′5	3	—	PCB136	22′33′66′	6	—
PCB28	244′	3	—	PCB154	22′44′56	6	—
PCB33/20	2′34/233′	3	—	PCB151	22′355′6	6	—
PCB22	234′	3	—	PCB135/144	22′33′56′/22′345′6	6	—
PCB72	23′55′	4	—	PCB147	22′34′56	6	—
PCB68	23′45′	4	—	PCB149	22′34′5′6	6	—
PCB57	233′5	4	1	PCB139/140	22′344′6/22′344′6′	6	—
PCB67	23′45	4	—	PCB143/134	22′3456′/22′33′56	6	—
PCB58	233′5′	4	2	PCB142/131	22′3456/22′33′46	6	—
PCB63	234′5	4	—	PCB146/161	22′34′55′/233′45′6	6	—
PCB61/74	2345/244′5	4	—	PCB132/153	22′33′46′/22′44′55′	6	—
PCB70/76	23′4′5/2′345	4	—	PCB168	23′44′5′6	6	—
PCB66	23′44′	4	—	PCB141	22′3455′	6	—
PCB56/60	233′4′/2344′	4	—	PCB137	22′344′5	6	—
PCB111	233′55′	5	2	PCB130	22′33′45′	6	—
PCB120	23′455′	5	—	PCB160/163/164/138	233′456/233′4′56/233′4′5′6/22′344′5′	6	—
PCB124	2′3455′	5	—	PCB158	233′44′6	6	—
PCB108/107	233′45′/233′4′5	5	—	PCB129	22′33′45	6	—
PCB123	2′344′5	5	—	PCB166	2344′56	6	—
PCB118	23′44′5	5	—	PCB128	22′33′44′	6	—
PCB114	2344′5	5	—	PCB188	22′34′566′	7	6
PCB122	2′33′45	5	—	PCB184	22′344′66′	7	—
PCB105	233′44′	5	—	PCB179	22′33′566′	7	—
PCB159	233′455′	5	5	PCB176	22′33′466′	7	—
PCB162	233′4′55′	6	—	PCB178	22′33′55′6	7	—
PCB167	23′44′55′	6	—	PCB175	22′33′45′6	7	—
PCB156	233′44′5	6	—	PCB187/182	22′34′55′6/22′344′56′	7	—
PCB157	233′44′5′	6	—	PCB183	22′344′5′6	7	—
PCB189	233′44′55′	7	—	PCB185	22′3455′6	7	—

[a] PCB = polychlorinated biphenyl; HxCDD = hexachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran; PnCDF = pentachlorodibenzofuran.

APPENDIX 1. Continued

Variable[a]	PCB substitution	Chlorine No.	No. <	Variable	PCB substitution	Chlorine No.	No. <
Di-*ortho* PCBs				PCB174/181	22′33′456′/22′344′56	7	—
PCB10/4	26/22′	2	—	PCB177	22′33′4′56	7	—
PCB19	22′6	3	—	PCB171	22′33′44′6	7	—
PCB18	22′5	3	—	PCB192/172	233′455′6/22′33′455′	7	—
PCB17	22′4	3	—	PCB180	22′344′55′	7	—
PCB27/24	23′6/236	3	—	PCB193	233′4′55′6	7	—
PCB16/32	22′3/24′6	3	—	PCB191	233′44′5′6	7	1
PCB53	22′56′	4	—	PCB170/190	22′33′44′5/233′44′56	7	—
PCB51	22′46′	4	—	PCB202	22′33′55′66′	8	—
PCB45	22′36	4	—	PCB201	22′33′45′66′	8	—
PCB46	22′36′	4	—	PCB197	22′33′44′66′	8	—
PCB73/52	23′5′6/22′55′	4	—	PCB200	22′33′4566′	8	—
PCB49	22′45′	4	—	PCB198	22′33′455′6	8	3
PCB47/75/48	22′44′/244′6/22′45	4	—	PCB199	22′33′455′6′	8	—
PCB44	22′35′	4	—	PCB203/196	22′344′55′6/22′33′44′5′6	8	—
PCB59/42	233′6/22′34′	4	—	PCB195	22′33′44′56	8	—
PCB71/41/64	23′4′6/22′34/234′6	4	—	PCB194	22′33′44′55′	8	—
PCB40	22′33′	4	—	PCB208	22′33′455′66′	9	—
PCB96	22′366′	5	4	PCB207	22′33′44′566′	9	1
PCB103	22′45′6	5	—	PCB206	22′33′44′55′6	9	—
PCB100	22′44′6	5	—	PCB209	22′33′44′55′66′	10	—