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Article

Decreased Growth Rate Associated with Tissue Contaminants in Juvenile Chinook Salmon Out-Migrating through an Industrial Waterway

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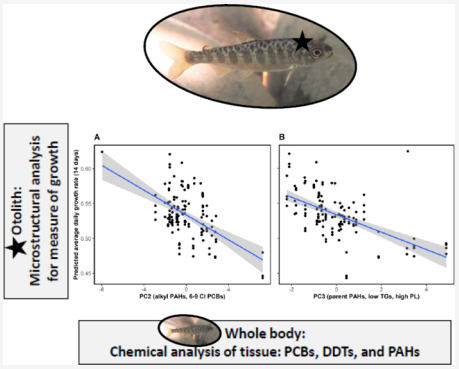
ABSTRACT: The industrial waterway in Portland Harbor, Oregon, is a migration corridor for a distinct population segment of Chinook Salmon (Upper Willamette River) currently protected by the U.S. Endangered Species Act. Juveniles are exposed to a suite of contaminants during outmigration including polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and dichlorodiphenyltrichloroethanes. We collected natural origin subyearling Chinook salmon from sites in and around the industrial harbor to evaluate growth (otolith microstructural analysis) in relation to measured chemical concentrations in tissue. A reduced growth rate was associated with higher tissue contaminant concentrations, particularly mixtures represented by PAHs and certain PCBs, which were elevated in juvenile Chinook collected throughout sites within Portland Harbor relative to those captured upstream. First-year growth is an established predictor of individual survival and eventual reproductive success in Chinook salmon. Therefore, our results indicate that legacy pollution may be limiting the population abundance of threatened Willamette River Chinook salmon, and future habitat remediation or restoration actions may benefit ongoing species recovery efforts.



KEYWORDS: *persistent organic pollutants, polycyclic aromatic hydrocarbons, growth, Chinook salmon*

INTRODUCTION

Historical industrial activities have contributed to the degradation of aquatic ecosystems through, in part, chemical pollution.[1] This is exemplified by the Willamette River in the State of Oregon (USA), which flows through highly industrialized Portland Harbor prior to its confluence with the lower Columbia River (Figure 1). For more than a century, the harbor has functioned as a commercial shipping port and working waterfront. Over the decades, numerous industries have released persistent organic pollutants (POPs) into the river including polychlorinated biphenyls (PCBs), and dichlorodiphenyltrichloroethanes (DDTs), as well as polycyclic aromatic hydrocarbons (PAHs) and butyltins [e.g., tributyltin (TBT)]. The U.S. Environmental Protection Agency (EPA) designated Portland Harbor as a Superfund site on the National Priorities List in December 2000.[2] Legacy pollution remains a concern for aquatic and aquatic-dependent organisms that rely on the lower Willamette River for habitat, including Chinook salmon (*Oncorhynchus tshawytscha*) stocks currently protected under the U.S. Endangered Species Act (ESA).

The present study focused on Chinook salmon that spawn in the upper reaches of the Willamette basin [Upper Willamette River (UWR) stock] and subsequently migrate as subyearling juveniles seaward through Portland Harbor and out to the Columbia River Estuary. The UWR Chinook population segment is considered at-risk following a threatened species designation under the ESA in 1999.[3] The McKenzie River produces the majority of natural origin Chinook salmon in the UWR evolutionarily significant unit. A portion of the juveniles from each year class, spring subyearlings, emigrate in their first spring soon after hatch. Migration timing is uncertain due to the small size of these fish, which precludes conventional in-stream tracking via telemetry and similar methods. However, this life history sub-type is expected to reside in the lower river habitat of Portland Harbor for weeks or longer to feed and grow before transitioning to the lower Columbia River Estuary.[4,5] This extended residency time would increase the likelihood of consuming contaminated prey and consequent

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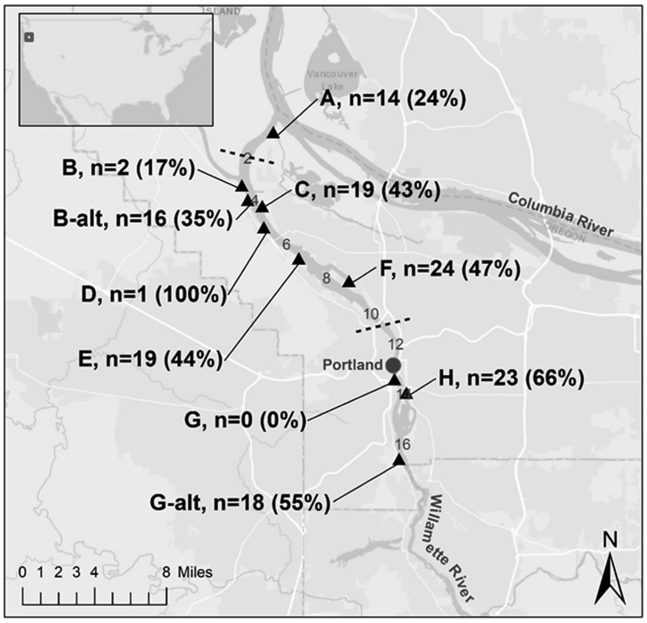

Figure 1. Map of the study area showing collection sites of 136 UWR Chinook salmon. Black triangles denote sites, parentheses show percent of Chinook salmon sampled from each site that were UWR stock (denoted by *n*). The gray numbers along the Willamette River show river miles. The circle shows the location of downtown Portland, Oregon (USA). The dashed lines show the approximate boundaries of the Portland Harbor Superfund site. The inset shows the proximity of the sample location in the USA. Service Layer Credits: Esri, HERE, DeLorme, MapmyIndia, copyright OpenStreetMap contributors, and the GIS user community.

accumulation of toxics in their tissues, as shown previously in juvenile salmon collected from industrial areas[6,7] where ingestion of contaminated prey is a dominant exposure pathway.[8,9] For example, chemical analyses of juvenile Chinook in the lower Columbia River Estuary showed an association between contaminated diet and elevated tissue concentrations of PCBs and DDTs[10] as well as PAH metabolites in bile.[11]

Chinook salmon migrating through contaminated habitats are vulnerable to both the near-term and delayed effects of toxic exposure.[12] Adverse health outcomes are likely to be influenced by interactions between chemicals in complex mixtures as well as the convergent impacts of chemical and non-chemical stressors such as surface water temperatures and pathogens.[13,14] Certain PCBs, DDTs, and PAHs that persist in Portland Harbor have previously been demonstrated to impair the growth of juvenile salmon.[15−17] First-year growth, in particular, is a key determinant of individual fitness (i.e., lifetime survival and reproduction).[18] Smaller fish are more susceptible to predation and less effective competitors for prey.[19] Therefore, population-scale effects of historical industrial pollution on the UWR Chinook population segment are likely to be mediated by reduced individual survival and specifically delayed mortality as a consequence of sublethal impairments to outmigrant growth.

The exposure−response relationship between tissue contaminant concentrations and growth has not been explored for ESA-listed UWR Chinook or evaluated for mixtures reflective of real-world exposure conditions in the lower Willamette River. Here, we used microstructural otolith analysis to assess growth in wild subyearlings captured from Portland Harbor at locations upstream, downstream, and within the designated Superfund site. Focal contaminants (PCBs, DDTs, and PAHs) were measured in whole body tissue and stomach content composite samples as a dose metric.[20] Lipids (percent and composition by class) in whole body tissue samples were also evaluated as a measure of energy availability.[21,22] Both site of collection and empirical tissue concentration of contaminant mixtures were evaluated as predictors of variability in somatic growth rate.

METHODS

Study Area and Fish Collection. The Portland Harbor Superfund site extends from river mile 2 to 11 of the lower Willamette River (Figure 1). Field operations to collect fish were conducted between river mile 0 (confluence of the Willamette and Columbia Rivers) upstream to river mile 17 (Figure 1, Table S1). Sampling site selection was guided by previously reported sediment contamination[2] and suitability for beach seining. Juvenile Chinook salmon (<100 mm fork length) were collected during April 17−22, 2018 under Scientific Research Permit 20713 from the National Marine Fisheries Service and Scientific Taking Permit (fish) 21914 from the Oregon Department of Fish and Wildlife. A total of 10 sites were sampled in a random sequence across days: one downstream (A), six within the designated Superfund site boundary (sites B, B-alt, C, D, E, and F), and three upstream sites (G, G-alt, and H). At each site, fish were collected using a 37 m × 2.4 m (10 mm mesh size) floating beach seine deployed from a 17 ft (5.2 m) Boston Whaler. Sites B, G, and D were unsuitable for beach seining and were replaced by sites B-alt and G-alt; an alternate site was not available for site D.

Juvenile Chinook salmon from each location were placed in an aerated bucket filled with river water and transported to shore for processing within 5−6 h of capture (median, ∼2.5 h). Fish were euthanized with a blunt force blow to the cranium and immediately measured for length (mm) and weight (g). Fin clips, otoliths, livers, and stomach contents were isolated and removed, weighed (livers and stomach contents), and placed in their respective containers. Individual fish and livers, and stomach contents composited by site during field dissections due to small mass, were immediately placed on dry ice and remained frozen until chemical analyses. Liver tissues were stored for potential future analysis and are not further discussed here. Fin clips were preserved in ethanol for genotyping (stock identification), and otoliths were removed and placed in dry containers for microstructural analysis (somatic growth determination).

Genetic Analysis for Stock Assignment. Juvenile Chinook salmon from diverse geographic origins rely on habitats in the lower Willamette River.[23] This study focused on UWR juvenile Chinook salmon that hatch upstream of, and migrate through, Portland Harbor. Non-target genetic stocks are presumed to originate in other Columbia River Basin watersheds and occupy areas of the Willamette/Columbia River confluence during their outmigration to the ocean. To confirm a UWR origin, genetic stock identification was performed using single nucleotide polymorphic genotypes.[24] Individuals assigned to the UWR reporting group (assignment probability of 0.8 or greater, as recommended by Moran et al.)[25] were included in subsequent analyses. Further details on the genetic analysis and stock assignments can be found in the Supporting Information.

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Environ. Sci. Technol. 2021, 55, 9968−9978

Table 1. Metrics of 135 Individual UWR Chinook Salmon Collected by Site; Mean Value (Minimum−Maximum); *p < 0.05, Upstream Site G-alt as the Reference; †p < 0.05, Upstream Site H as the Reference

site	river bank[a]	# fish	# total composites	fork length (mm)	fish mass (g)	condition factor (K)	hepatosomatic index (HSI)	% lipids[b]	TG[b,c]
A	E	14	3	43† (37−57)	0.8† (0.4−1.9)	0.9 (0.7−1.1)	1.0† (0.5−2.8)	1.4 (1.2−1.6)	17.2 (11.8−24.1)
B	W	2	1	57 (56−57)	2.4 (1.7−3.0)	1.3 (1.0−1.6)	1.1 (0.9−1.4)	2.0 (NA)	55.7 (NA)
B-alt	W	16	4	50 (36−83)	1.5 (0.3−6.3)	1.0 (0.7−1.2)	1.1 (0.8−1.5)	1.1 (1.0−1.5)	23.6 (11.6−33.3)
C	E	19	5	51 (38−75)	1.4 (0.4−4.0)	0.9 (0.8−1.0)	1.1 (0.7−2.2)	0.9* (0.8−1.1)	19.8 (0.0−44.3)
D	W	1	1	68	3.2	1	0.9	0.9	20.8
E	W	19	6	50 (38−70)	1.3 (0.5−3.6)	1.0 (0.8−1.0)	1.0† (0.7−1.5)	1.2 (0.8−1.6)	25.2 (10.4−35.2)
F	E	23	8	57* (40−89)	2.4* (0.6−6.8)	1.1*,† (0.9−1.3)	1.2* (0.8−2.2)	1.3 (0.8−3.0)	30.9 (7.8−66.3)
H[d]	Ref E	23	6	54* (40−79)	1.6 (0.4−5.2)	0.9 (0.4−1.2)	1.3* (0.3−2.2)	1.2 (0.9−1.9)	35.1 (9.0−54.7)
G-alt[d]	Ref W	18	3	45† (39−72)	1.0 (0.5−5.1)	0.9 (0.7−1.4)	1.0† (0.6−1.8)	1.2 (1.1−1.3)	20.7 (13.2−30.9)

[a]E = East bank, W=West bank, Ref E = East bank reference site upstream of Portland Harbor, Ref W = West bank reference site upstream of Portland Harbor. [b]Average fork length of individual fish per composites was included in all analyses and percent lipids was included in analyses of lipid class. [c]TG = proportion of triglycerides composing the total lipid in whole body composite samples. [d]A single tissue composite was created by combining two fish collected at site H and one fish collected at G-alt to reach the requisite mass requirements for TBT analysis in addition to POPs and PAHs; % lipids = 1.7, TG = 50.5.

Otolith Microstructure. Otolith microstructure was examined on all genetically confirmed UWR Chinook salmon to estimate recent somatic growth using methods described previously.[26] Individual otoliths were analyzed by a single observer; to control for potential observer bias, a random subset of images (10% of otoliths) were reanalyzed without source attribution (i.e., blind) to confirm consistent and repeatable measurements. Otoliths were polished to visualize the core and associated daily growth increments under a light microscope. Otolith measurements included radius at the time of capture (distance from core to edge, O_c) and radius measured at n days before capture (distance from core to daily increments from the edge, O_a). One of the 136 UWR Chinook salmon had a visible otolith annulus, indicating this fish hatched the previous year, and was excluded as a presumed yearling fish (site F), and one of the 135 subyearling otoliths was overpolished (site F) and not included in the final growth data set. For each otolith, fork length at n days prior to capture (L_a) was estimated using the Fraser−Lee equation: $L_a = d + ((L_c - d)/O_c) \times O_a$, where d is the intercept (−6.37 mm) of the regression between fish length and otolith radius (R^2 = 0.89, n = 134) and where L_c represents fork length (mm) at capture. For each individual fish, average daily growth rate (mm fork length per day) was back-calculated for the 2-week (14 d) interval prior to capture (a) using the following equation: $(L_c - L_a)/a$.

Tissue Composites. Whole body tissue composites comprised individual juvenile Chinook salmon minus fin clips, liver tissue, stomach contents, and otoliths. The number of composites for POP and PAH contaminant and lipid analyses was determined by the amount of fish mass available at each site. When sufficient mass was available (multiple fish >3.0 g from the same site), a composite was assigned for TBT analysis in addition to POPs, PAHs, and lipids (n = 6 TBT composites; analysis and results in the Supporting Information). All remaining fish for each site were divided so that the maximum number of composites for POP and PAH contaminants and lipid analysis per site could be achieved with a similar number and mass of fish per composite. A single composite was obtained from sites B (n = 2 fish) and D (n = 1 fish); sites A, B-alt, C, E, F, H, and G-alt had 3−8 total composites for POP/PAH chemistry and lipid analysis (Table 1). Sufficient mass was available for a single stomach contents composite sample from sites A, B-alt, C, E, and F. A single stomach contents composite and single TBT tissue composite was created by combining fish collected at sites H and G-alt (H + G-alt) to reach the requisite mass requirements and ensure that measures of TBT tissue concentration and stomach content POP and PAH contaminant levels upstream of Portland Harbor were available.

Analytical Chemistry. Fish tissue and stomach content composites were analyzed at NOAA's Northwest Fisheries Science Center (NWFSC) (Seattle, WA) for levels of PCBs, DDTs, and PAHs using a gas chromatography/mass spectrometry (GC/MS) method.[27] In brief, Chinook salmon bodies were homogenized and extracted with dichloromethane using an accelerated solvent extractor. The sample extracts were precleaned on an alumina−silica column, further cleaned using size-exclusion liquid chromatography, and then analyzed by low-resolution GC/MS. The target analyte list included 45 PCB congeners and 6 DDT isomers. Measured concentrations also included parent PAHs and corresponding alkylated homologues, comprising a total of 42 PAHs. Sum "low molecular weight aromatic hydrocarbons (PAHs)" (sum LMWAHs) and sum "high molecular weight aromatic hydrocarbons (PAHs)" (sum HMWAHs) were calculated using summation of PAHs based on number of aromatic rings, less than four and four or greater, respectively. Total PAH concentrations were calculated by summing the low and high molecular weight PAHs. The full list of PCB, DDT, and PAH compounds and details of the quality assurance program can be found in the Supporting Information.

Percent lipids in the fish tissue composites were measured gravimetrically at the NWFSC following extraction in dichloromethane. Lipids were not analyzed in stomach content samples due to mass restrictions. Lipid class determination (triglycerides, TGs; free fatty acids, FFAs; polar lipids, PLs; and cholesterol, Chol) was conducted using thin-layer chromatography/flame ionization detection.[27] All samples analyzed for contaminants and lipid content met established quality assurance criteria.

Statistics. To evaluate a site as a predictor of variability in somatic growth rate, two models were used. First, a generalized linear model (GLM) approach using a gamma family distribution with a log link to account for non-normally distributed data was used to evaluate the average daily increase in fork length over the 14 days prior to fish capture.[28] Second, a generalized estimating equation (GEE) using a gamma family

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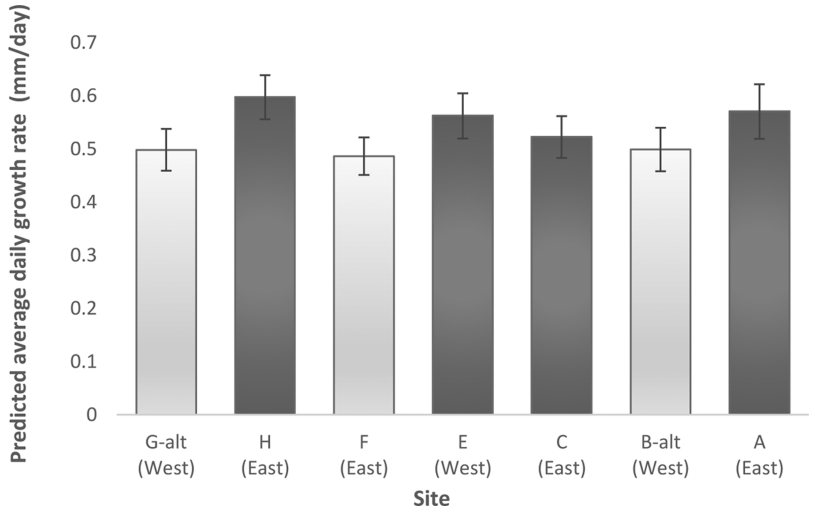

Figure 2. Predicted average daily growth rate (GLM) from otolith microanalysis of UWR Chinook salmon across 14 days prior to collection by site with 95% CI. River bank in parentheses (east or west). Dark gray bars correspond to significant differences ($p < 0.05$) from site G-alt (west bank reference site upstream of Portland Harbor). Light gray bars correspond to significant differences ($p < 0.05$) from site H (east bank reference site upstream of Portland Harbor).

distribution with a log link and an autoregressive correlation structure was used to estimate daily increments of growth, as determined from repeat measures from individual fish across the same 14 d interval.[28] Sites with two or fewer individual fish or tissue composites were not included in the statistical evaluations. Separate analyses were performed using the individual upstream sites as reference. Sites H (east bank) and G-alt (west bank) were used to represent each bank of the river to account for a presumed bank fidelity in subyearling fish that are unlikely to cross the deep channel of the lower Willamette River.[29] Fork length at time of dissection was included in all analyses to account for the linear relationship observed between growth rate and fish length ($F_{(1,132)} = 8.3$, $p = 0.005$). Models were run using individual sites and also as two groups with sites categorized by river bank (east vs west). Additional covariates considered included hepatosomatic index [HSI; (liver mass (wet-weight, g)/fish mass (wet-weight, g)) × 100] and gut fullness (based on weight of individual stomach contents). To compare models, we calculated Akaike's information criterion (AIC) values for each model and the difference in AIC values between models.[30,31] This allowed us to investigate whether the addition of, for example, HSI, explained the variability in somatic growth rate.

A contaminants model was also evaluated to analyze mixture composition as a predictor of variability in somatic growth rate, using the same GLM and GEE models outlined above. A principal component analysis (PCA) with a correlation matrix was used to account for collinearity between whole body contaminant concentrations. The inputs to the PCA model included tissue composite concentrations [ng/g wet weight (ww)] of PCB homologues (trichlorinated through non-achlorinated), DDT isomers, and PAH parent and alkyl groups separated by LMWAHs and HMWAHs as well as percent lipids and proportion of lipid class (TG, FFA, PL, and Chol). Percent lipids was included to account for lipophilic properties of the contaminants (normalization of concentrations); proportion lipid class was included as a metric of energy resources.[21,22] Components with an eigenvalue greater than 1.0 were retained, with the total variance accounted for in the retained components confirmed by a broken stick model.[32]

Fish measurements (length, mass, K, and HSI), contaminant values, and lipid measures for UWR juvenile Chinook were evaluated using a GLM as described above with the most upstream sites, H (east bank) and G-alt (west bank), serving as the reference in separate analyses. Models were run using individual locations and also as two groups of sites corresponding to the east and west banks of the river. Average fork length of individual fish in each composite sample was included in the models. Sites with two or fewer individual fish or tissue composites were not included in the statistical evaluations. Evaluations of the tissue contaminant values by site included percent lipid to account for the lipophilic chemical properties of the compounds.

All analyses were conducted in R (version 3.2.2).[33]

RESULTS

Chinook Salmon Subyearlings Collected from the Lower Willamette River. A total of 320 unmarked juvenile Chinook salmon (<100 mm fork length) were collected across the 10 lower Willamette River sites (Figure 1, Table S1), representing several distinct genetic stocks (Table S2). In total, 43% ($n = 136$) from nine sites were assigned to the UWR genetic stock group; of these, 135 were subyearlings (one fish with a visible otolith annulus was excluded from site F, as a presumed yearling). The mean fork length of the 135 UWR Chinook salmon subyearlings across all sites was 51 mm (median, minimum−maximum; 46, 36−89 mm) and mean mass was 1.5 g (1.0, 0.3−6.8 g), with 14−23 fish collected per site (excluding sites replaced with alternate sites). Length and mass were highly correlated ($p < 0.001$), and both were lower at east bank site A (43 and 0.8) relative to east bank upstream site H (54 and 1.6) (Table 1). Whole body percent lipids averaged 1.2% (median, minimum−maximum; 1.1, 0.8−3.0) across all collection locations. Percent lipids in fish from east bank site C (0.9%), within Portland Harbor, were significantly lower relative to the upstream reference fish (west bank site G-alt, 1.2%; Table 1). This general reduction in total lipids at site C corresponded to differences in the lipid class profile (Table S3), with significantly lower FFAs (17% of total lipids) relative to both upstream reference sites (east bank site H, 23%; west

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Table 2. Predicted Daily Growth Rate (mm Fork Length per Day) from Otolith Microanalysis of UWR Chinook Salmon; Slope Coefficient[a] (Standard Error)

		average daily growth rate[b] (mm/day), 14 days	*p*-values	incremental daily growth rate[c] (mm/day), 14 days	*p*-values
contaminant model	intercept	−0.804 (0.080)	<0.001	−0.644 (0.080)	<0.001
	PC1 (DDTs)	0.002 (0.006)	0.694	−0.003 (0.005)	0.526
	PC2 (alkyl PAHs, 6−9 Cl PCBs)	−0.017 (0.008)	0.040	−0.018 (0.007)	0.010
	PC3 (parent PAHs, low TGs, high PL)	−0.017 (0.011)	0.108	−0.022 (0.010)	0.022
	PC4 (3−5 Cl PCBs)	0.005 (0.010)	0.675	0.007 (0.010)	0.457
	PC5 (% lipids, high TGs, low FFA/Chol)	−0.004 (0.012)	0.773	−0.004 (0.011)	0.734
	days	NA	NA	−0.036 (0.010)	<0.001
	days × days	NA	NA	0.002 (0.0006)	0.001
	fork length (mm)	0.003 (0.002)	0.028	0.003 (0.001)	0.029

[a]For a 1-unit change in covariate (e.g., PC2), exp(coefficient) equals the ratio of predicted daily growth rate. [b]GLM adjusted for fork length at capture. [c]GEE with repeat measures to account for daily growth measurement for each individual, adjusted for fork length at capture, with an autoregressive correlation structure. Abbreviations: PAHs, polycyclic aromatic hydrocarbons; PCBs, polychlorinated biphenyls; TGs, triglycerides; FFAs, free fatty acids; Chol, cholesterol; 6−9 Cl, hexa- through nonachlorinated; 3−5 Cl, tri- through pentachlorinated.

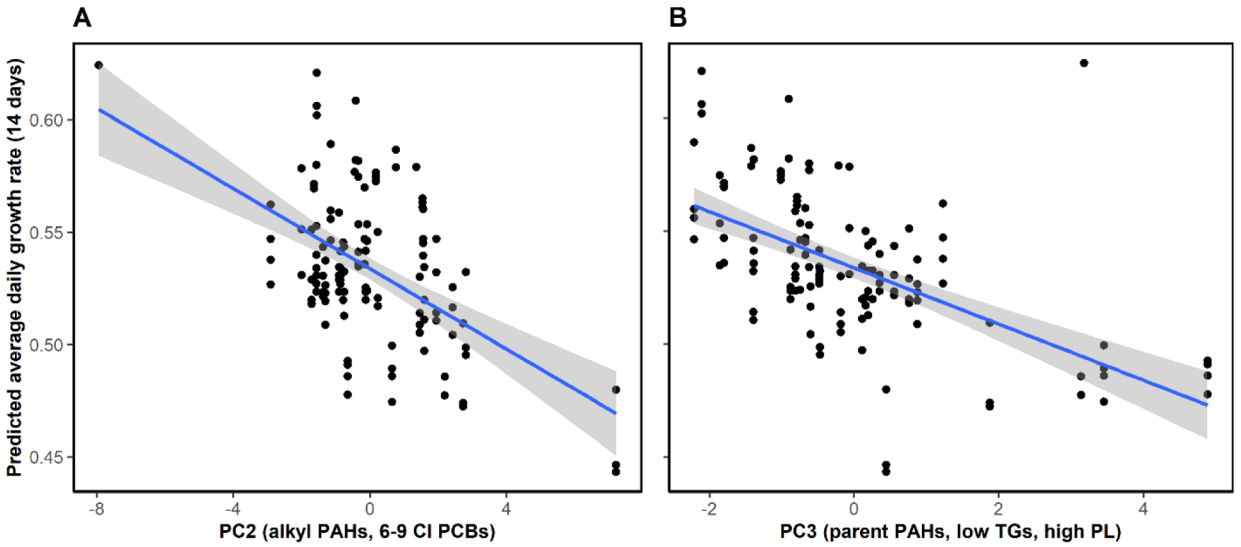

Figure 3. Predicted average daily growth from otolith microanalysis of UWR Chinook salmon across 14 days prior to collection date, as predicted by (A) PC2 [alkyl PAHs and 6−9 Cl (hexa- through nonachlorinated) PCBs] and (B) PC3 [parent PAHs, low TGs, high PLs] (see Supporting Information for PC1, PC4, and PC5).

bank site G-alt, 20%). Further details on the size metrics and measured contaminants and lipids across all juvenile Chinook salmon samples are available in the Supporting Information.

Individual Juvenile Growth Rates in Relation to River Location (Collection Site). Individual determinations of daily somatic growth were obtained from microstructural analyses of otoliths extracted from UWR juvenile Chinook salmon (n = 134 otoliths from individual fish across all study sites). Based on this approach, growth rates ranged from 0.32 to 0.87 mm/day (mean = 0.53) for the 14 days prior to collection. Predicted daily growth was variable across sites (Table S4; Figure 2). The growth rate between the two sites upstream of Portland Harbor (site H, east bank; site G-alt, west bank) was significantly different ($p < 0.05$). Fish from site H on the east bank demonstrated a rate of 0.60 (95% confidence interval (CI), 0.56−0.64), with 0.50 (95% CI, 0.46−0.54) at site G-alt on the west bank, across the 14 days prior to collection. When comparing growth at the individual collection sites to the upstream reference sites, significant differences were dependent on which upstream reference site was used. When sites within and downstream of Portland Harbor were grouped by bank, growths on the east (0.52; 95% CI, 0.50−0.55) and west (0.53; 95% CI, 0.50−0.56) bank sites were not significantly different from each other. Sampling site within Portland Harbor was not a predictor of growth in the fish collected for this study, whether considered as individual sites compared to the upstream reference sites or grouped by river bank.

Individual Juvenile Growth Rates in Relation to Tissue Contaminant Concentrations. A total of 38 whole body tissue composites collected within and around Portland Harbor were analyzed for contaminants and lipid content. Tissue contaminant concentrations in juvenile Chinook salmon reflect environmental exposures to the complex mixture of contaminants in the lower Willamette River and account for movement of fish between sites and varying residency times at a sampling location. Tissue concentrations of contaminants and lipids were evaluated using PCA to

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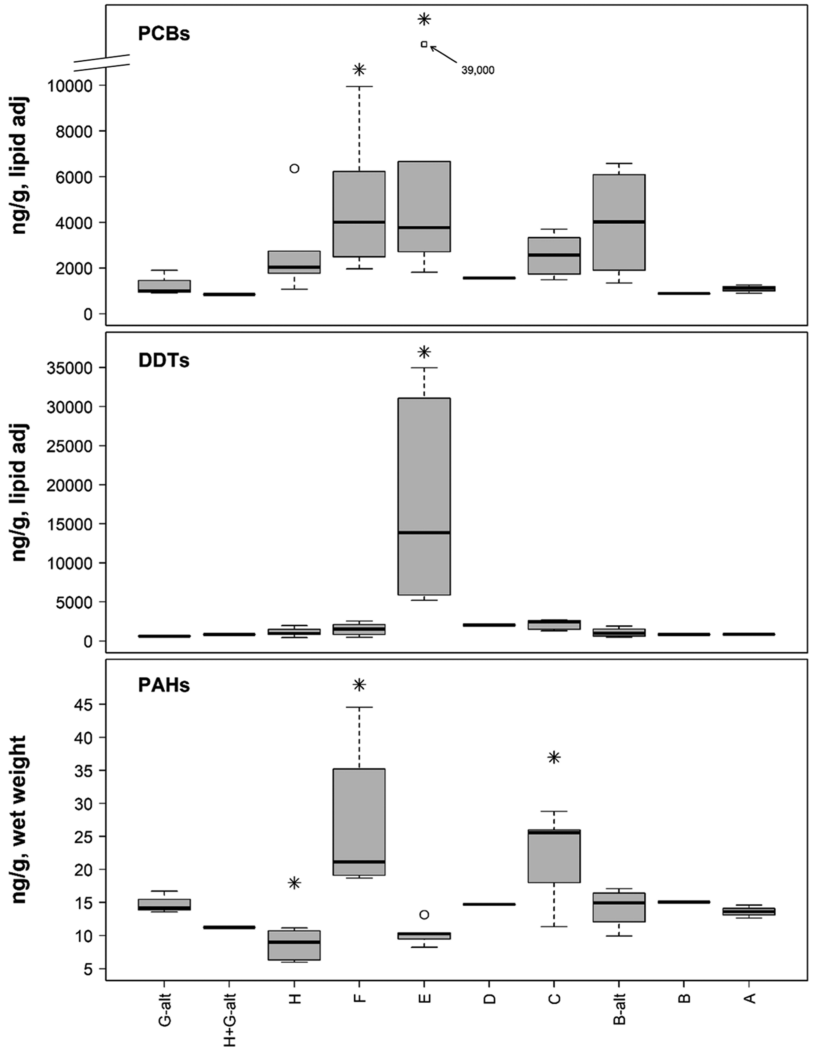

Figure 4. Measured contaminants in whole body tissue composites (minus otoliths, fin clips, livers, and stomach contents) of UWR juvenile Chinook salmon by site; sum of (top) 45 PCBs (ng/g lipid adjusted), (middle) six DDTs (ng/g lipid adjusted), and (bottom) 42 PAHs (ng/g wet weight). Open square for site E denotes outlier beyond *y*-axis. *$p < 0.05$, G-alt as the upstream reference site. (Figure S3, site H as the upstream reference site). For all box plots, the thick black line represents the median of the data. The end of the box shows the upper and lower quartiles (interquartile range; IQR). The whiskers show the highest value up to 1.5 times the IQR above the third quartile or the lowest value up to 1.5 times the IQR below the first quartile. The open symbols represent data values higher or lower than the maximum whisker values. H + G-alt = A single tissue composite created by combining fish collected at sites H and G-alt to reach the requisite mass requirements for TBT analysis in addition to POPs and PAHs.

summarize highly correlated groups of chemicals. There were five retained components (Table S5) representing DDTs (PC1), alkylated PAHs, and hexa- through nonachlorinated PCBs (PC2), parent PAHs, low TGs, and high PLs (PC3), tri- through pentachlorinated PCBs (PC4), and high percent lipids with high TGs, low FFAs, and low Chol (PC5). These components are mutually independent and therefore appropriate for inclusion as predictors in mixed effects multiple regression models.

Measured daily somatic growth was evaluated for association with mixtures of co-occurring contaminants identified through the PCA. Variability in average daily somatic growth and incremental daily growth (i.e., repeat measures) from individual fish for the 14 days before capture showed significant negative associations with the principal component represented by alkylated PAHs (LMW and HMW) and hexa- through nonachlorinated PCBs (PC2, explained 18% of the variance in the data set within the PCA) (*p*-values, 0.040 and 0.010, respectively) (Table 2; Figure 3A). This significance was maintained when the most extreme values (PC2 < −4 or PC2 > 4) were excluded; slope coefficient (standard error, SE), *p*-value: 0.030 (0.012), 0.012. There was a weak negative correlation between somatic growth rate and the component represented by parent PAHs, low TGs, and high PLs (PC3 explained 12% of the variance) when growth was evaluated as average daily growth rate (*p*-value, 0.108); the relationship was significant when evaluated as incremental daily growth rate, likely due to the increased statistical power (*p*-value, 0.022) (Table 2; Figure 3B). The DDT-specific component (PC1, 33% of the variance) and the component associated with tri- through pentachlorinated PCBs (PC4, 11% of the variance) were not significantly associated with somatic growth rate (Figure S1). Similarly, the lipid-specific principal component (PC5, 9% of the variance) was not significant. Additional condition covariates were explored (e.g., HSI), but none further explained the observed variability in somatic growth

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Environ. Sci. Technol. 2021, 55, 9968−9978

(Table S6). In consideration of PC1, PC4, and PC5 not showing an association with growth, the contaminants model was re-analyzed as a reduced GLM model that retained only components PC2 and PC3. The resulting model coefficients were similar [slope coefficient (SE), p-value: PC2, −0.017 (0.008), 0.036; PC3, −0.018 (0.010), 0.095]. In all, tissue contaminant concentration, evaluated as mixtures of correlated compounds, predicted recent growth in these natural origin juvenile Chinook salmon.

Measured Contaminants and Lipids in Juvenile Chinook Tissues and Stomach Contents. The 38 whole body tissue composites from nine sites were analyzed for toxics and lipids (Figure 1). Contaminant levels were generally elevated in whole body composites from fish collected near areas with documented sediment contamination along the east and west banks of the Portland Harbor working waterfront.[2] Specifically, concentrations of total PCB congeners were significantly higher in whole body composites collected from west bank site E (mean, 103 ng/g ww; 1.2% lipids), with a suggested increase in west bank site B-alt ($p < 0.10$) (mean, 41; 1.1% lipids), relative to upstream west bank site G-alt (15, 1.2%) (Figure 4; Table S7). The significant increase at site E was maintained after exclusion of the sample greater than 1.5 the inter-quartile range (IQR) (Figure 4). East bank site F (51; 1.3%) demonstrated a suggested increase in total PCBs ($p < 0.10$), relative to upstream east bank site H (28, 1.2%), which was significant ($p < 0.05$) when the site H sample greater than 1.5 the IQR was excluded (Figure S2). The DDT body burden at the west river bank site E (mean, 211 ng/g ww; 1.2% lipids) within Portland Harbor was more than an order of magnitude higher than all other locations, none of which exceeded 20 ng/g ww (Figure 4; Table S7). Mean whole body PAH levels were elevated at two sites within Portland Harbor (east bank sites F and C; mean, 26.8 and 21.9 ng/g ww, respectively) relative to both the upstream reference sites ($p < 0.05$; east bank site H, 8.7 ng/g ww; west bank site G-alt, 14.8) (Figure 4, Table S8 and Figure S3). Overall, tissue contaminant concentrations were elevated throughout the Harbor relative to the upstream sites, affirming that contaminant exposures in juvenile Chinook salmon occurred within the Harbor. The elevated tissue concentrations within the Harbor were maintained when specifically looking at the hexa- through nonachlorinated PCBs and PAHs represented by PC2 and PC3 and associated with a reduced growth rate (Figure S4).

The profiles of PCBs, DDTs, and PAHs in stomach contents and whole body tissue composites from the same site were similar (Figure 4; Table S9). Specifically, dietary DDTs in fish from site E (142 ng/g ww), dietary PCBs in fish from sites F, E, and B-alt (59, 46, and 53 ng/g ww, respectively), and dietary PAHs in fish from sites F and C (837 and 593 ng/g ww, respectively) were elevated relative to the stomach contents from fish upstream of the designated Superfund site locations in Portland Harbor (single composite from upstream sites H and G-alt: Sum DDTs, 17; Sum PCBs, 33; Sum PAHs, 196 ng/g ww). These results are consistent with earlier findings,[10,11] indicating a general correspondence between site-specific exposures to PCBs, DDTs, and PAHs via the diet and elevated tissue concentrations among subyearling Chinook that reside in these local habitats to feed, shelter, and grow prior to transitioning to the lower Columbia River Estuary. As noted earlier, however, restrictions intrinsic to the experimental design of the current study limited the number of samples available for stomach content analyses. Additional and more intensive sampling would likely be necessary to strengthen future analyses of localized habitat use and consequent dietary contaminant exposure.

DISCUSSION

The Portland Harbor Superfund site provides critical habitat for aquatic communities, including ESA-listed Chinook salmon. The present study collected juvenile Chinook salmon from the threatened UWR distinct population segment as they migrated seaward through the highly industrialized Portland Harbor. Clean rearing habitats (i.e., minimal toxic chemical contamination) is considered critical for adequate growth, which was evaluated as a potential effect of exposure to contaminants.[13,15] Tissue contaminant concentrations were elevated in Chinook captured throughout the Harbor relative to upstream sites, providing a biological measure of exposures that occurred at sites within Portland Harbor. Reduced somatic growth rate in the UWR juvenile Chinook salmon was correlated with increases in tissue contaminants. The ability to link exposure and sublethal response in field-caught fish resulted from using the otolith microstructure to assess growth, paired with a statistical model that accounted for the complex chemical mixture across a gradient of concentrations. Growth during the first year is an established predictor of survival in Chinook salmon.[18] Size-selective mortality in juveniles resulting from reduced growth will impact productivity and may thereby yield population-level impacts.[34] The ESA-listed Chinook salmon in the Willamette River are at historically low abundance.[3] Our results indicate that contaminants are affecting growth and may be impacting the population viability of UWR Chinook salmon.

Reduced growth in subyearlings was significantly associated with complex chemical mixtures in whole body tissues, including hexa- through nonachlorinated PCBs (PC2, Table 2). Environmentally persistent contaminants, such as PCBs and DDTs, readily accumulate in the lipid-rich tissues of fish due to their limited biotransformation capability and lipophilicity (high log K_{ow}).[8,35,36] The association of reduced growth and higher-chlorinated PCBs, relative to the lower-chlorinated PCBs, may be reflective of reduced fish metabolism of these higher-chlorinated congeners causing increased bioaccumulation. Changes in growth of fish species, including Chinook salmon, in past studies have been demonstrated with exposure to PCBs and DDTs, although at concentrations generally higher than those measured in our current field reconnaissance.[37,38] A recent meta-analysis developed a concentration−response threshold regression on PCB-related adverse effects representing contaminant-related injury to fish populations from PCB mixtures as they occur in the environment, including a concentration range that match this study.[39] Growth impairment is associated with PCB exposure, with an upper limit in induced growth reduction in exposed fish species before alternate pathways of toxicity became apparent. For the concentration range in the current study, the meta-analysis showed mortality and reproductive impairment also accounted for prediction of injury and should be similarly considered when evaluating cumulative effects of PCB-induced toxicity to fish.

The tissue concentrations of DDTs were an order of magnitude higher at a single site (site E) relative to samples collected at all other locations. The tissue concentrations in four of the six composites from site E (13 000−35 000 ng/g lipid) were more than twofold greater than an established

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Environ. Sci. Technol. 2021, 55, 9968−9978

protective DDT tissue residue concentration of 6000 ng/g lipid.[37] This finding indicates an increased possibility that these fish would have experienced an adverse biological endpoint including decreased growth or survival. The disparate DDT tissue concentrations between sites resulted in binary representation of the data versus a continuous range of tissue concentrations. This indicates the study design may have been inadequate to estimate an effect and could fail to capture a correlation between DDT concentration and growth. Other possible explanations of the absent correlation, not precluded by the description above, include the following: (1) there was no growth effect of DDT exposure on out migrating salmon, or the effect was small relative to variability in growth rates observed in our study or (2) similar to a possible outcome for PCB exposure,[38,40] fish with modified growth did not survive.

Whole body tissue PAH concentrations were significantly associated with reduced growth (PC2 and PC3, Table 2). This is consistent with a previous study specific to juvenile Chinook salmon[16] and several studies of other fish species[41] as well as a meta-analysis of PAH dose−response across nine fish species, including Chinook salmon.[42] In contrast to PCBs and DDTs, fish rapidly metabolize PAHs.[41] This occurs through the initial action of cytochrome p450, followed by a series of detoxifying steps that convert metabolites to polar complexes that can be readily eliminated by the gastrointestinal tract. Due to this rapid metabolism and elimination, PAHs do not typically accumulate in tissues.[41] Therefore, measurement of PAH exposure has traditionally relied on indirect methods, including the detection of fluorescent PAH metabolites in bile.[11,15] In our study, however, elevated PAHs were directly measured in whole bodies, indicating that exposure was recent and PAHs were bioavailable to juvenile Chinook as they moved downriver through Portland Harbor.

Decreased growth in juvenile Chinook salmon in the present study was also associated with a decrease in TGs (PC2; Table 2), consistent with an earlier experimental PAH-feeding study in juvenile Chinook.[16] Lipid allocation (by tissue and class) is an indicator of condition and energy reserves in fish.[43−45] TGs in liver, muscle, and mesenteric fat[21] are the storage form of FFAs, for release when the energy requirements of the fish exceed the energy obtained from prey consumption. They are also the preferred source of metabolic energy for growth, reproduction, and swimming.[46] Our observations may therefore reflect a diversion of energetic resources to detoxification or, possibly, behavioral factors such as reduced feeding.

Current management protections for ESA-listed UWR Chinook salmon placed constraints on our sampling effort. Accordingly, the number of fish allowed for our permitted field collection was highly restricted, and the fish were captured in a single sampling event that represented a 6-day cross-sectional analysis from a single year. Furthermore, the migratory nature of the target species leads to uncertainty regarding how long each fish had been in the sampling area or at each collection site. This is exemplified by the variability in growth rates between our two upstream reference sites, which were selected to represent the growth rate upstream of Portland Harbor. Despite practical constraints (e.g., restricted catch numbers) and variables we could not quantify (e.g., fish density), the tissue concentration data demonstrated increased levels of DDTs, PCBs, and PAHs in fish sampled from Portland Harbor, and reduced growth was associated with these increased tissue contaminant levels. Although our current study was focused on UWR Chinook, these findings have conservation implications for other salmonid populations with residence time in the lower river, including migrants from other watersheds in the upper Willamette River as well as the interior Columbia River Basin.

In conclusion, this study established a link between chemical mixture tissue concentrations and a decreased somatic growth rate among threatened UWR Chinook salmon subyearlings that rely on the historically contaminated Portland Harbor for foraging and rearing habitat, prior to transitioning seaward to the lower Columbia River Estuary. Future studies of environmentally relevant concentrations of these compounds and survival-determining biological endpoints (growth, immune function) in salmonids would offer an opportunity to substantiate the association between tissue concentrations of pollutants and adverse health outcomes. Looking ahead, newer toxicological tools in the areas of genomics and proteomics can be applied to determine contaminant effects at the molecular level and evaluate the role of chemicals in molecular variations and how those alterations impact mechanisms of toxicity and subsequent endpoints such as growth, survival, and reproduction.[47,48] As the pressure that past industrial activities place on present-day species and communities continues, these data will support ongoing efforts to protect aquatic ecosystems from chemical pollution (e.g., modeling population recovery in response to habitat improvements[49]). Our findings suggest that a reduction in exposure to toxics in Portland Harbor, through future contaminant remediation efforts, will likely increase juvenile growth, and, thereby, health and survival. Enhancing the contribution of the subyearling life history stage to the overall population abundance will support the recovery of this UWR Chinook stock.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/acs.est.1c01526.

Additional details of genetic analysis for stock assignment of individual fish, analytical chemistry target compounds and quality assurance, TBT results, and site-specific contamination profiles; plots of PC1-5, PAH concentrations with lipid adjustment, contaminant concentrations using site H as the upstream reference, contaminant loadings for PC2 and PC3, and PAH profiles for sites F and C; tables showing geographic data on fish collection, distribution of genetic stock assignments, lipid profiles across sites, chemistry data by site for whole body tissues and stomach contents, incorporation of covariates into models of growth, and PCA results showing varimax rotation. (PDF)

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