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# Lingering Effects of Legacy Industrial Pollution on Yellow Perch of the Detroit River

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Abstract: We used yellow perch (*Perca flavescens*) captured at four sites differing in legacy industrial pollution in the Lake St. Clair–Detroit River system to evaluate the lingering sublethal effects of industrial pollution. We emphasized bioindicators of direct (toxicity) and indirect (chronic stress, impoverished food web) effects on somatic and organ-specific growth (brain, gut, liver, heart ventricle, gonad). Our results show that higher sediment levels of industrial contaminants at the most downstream Detroit River site (Trenton Channel) are associated with increased perch liver detoxification activity and liver size, reduced brain size, and reduced scale cortisol content. Trenton Channel also displayed food web disruption, where adult perch occupied lower trophic positions than forage fish. Somatic growth and relative gut size were lower in perch sampled at the reference site in Lake St. Clair (Mitchell's Bay), possibly because of increased competition for resources. Models used to determine the factors contributing to site differences in organ growth suggest that the lingering effects of industrial pollution are best explained by trophic disruption. Thus, bioindicators of fish trophic ecology may prove advantageous to assess the health of aquatic ecosystems. *Environ Toxicol Chem* 2023;42:2158–2170. © 2023 The Authors. *Environmental Toxicology and Chemistry* published by Wiley Periodicals LLC on behalf of SETAC.

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## INTRODUCTION

Mitigating the negative impacts of the anthropogenic pollution of water bodies represents a considerable challenge (Malmqvist & Rundle, 2002; Schwarzenbach et al., 2006). To evaluate risks, indicators of pollution are sought and evaluated, but this task is complicated by the fact that pollution has both direct and indirect effects on organisms, and pollutants vary greatly in their nature and mechanisms of action. In addition to direct lethal or sublethal toxic effects on living organisms, pollution can have indirect effects through contaminant-induced disruption of community structure or environmental degradation, leading to disturbed food webs that can reduce foraging opportunities or change predation pressure for some organisms (Fleeger et al., 2003; O'Callaghan et al., 2019; Saaristo et al., 2018). Of particular concern are persistent organic and metal contaminants that are the by-products of industrial activity related to manufacturing and mining (Ali et al., 2019; Ali & Sreekrishnan, 2001; Behera et al., 2018; de Almeida Rodrigues et al., 2019; Maiti et al., 2019). Such pollutants are slowly degraded and thus can exert their effects for a long period of time. They can remain in place unaltered until they are made available by disruption of stored sources or biological activity.

Organisms at higher trophic levels (i.e., their relative position in a food web based on feeding relationships) are thought to reflect summed ecological processes of ecosystems that can be influenced by water pollution and the contribution of pollution stores in sediments (Weber et al., 2008). In this respect, fish represent good candidate organisms to assess the effects of bioconcentrated levels of contaminants due to their high trophic position in aquatic food webs (van der Oost et al., 2003).

Bioindicators are responses of organisms or communities that can be used to assess the health of ecosystems. A very diverse set of bioindicators of pollution have been used in fish. These cover community bioindicators that rely on extensive taxonomic sampling, such as biodiversity and the presence of parasites (Giraudo et al., 2016; McQuatters-Gollop et al., 2019; Vidal-Martínez et al., 2010), population growth patterns that

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rely on extensive population sampling at different life stages (Trippel, 1995), as well as behavioral bioindicators (Bownik & Wlodkowic, 2021; Chen, 2020; Hong & Zha, 2019; Parker, 2016; Tierney et al., 2010), tissue and DNA damage (Blazer, 2002; Mix, 1986; Singh et al., 1988; Yancheva et al., 2016), and different molecular methods used to study the expression of detoxification molecules (Roesijadi, 1992; Schlenk et al., 2008) or oxidative stress responses (Hellou et al., 2012; Lushchak, 2011; Valavanidis et al., 2006). The search for new bioindicators of pollution has recently entered the era of exploratory analyses with very broad molecular coverage, including analyses of the proteome (López-Pedrouso et al., 2020), transcriptome (Bruneau et al., 2016; Defo et al., 2018; Houde et al., 2014; Zare et al., 2018), metabolome (Bundy et al., 2009; Cappello, 2020), and gut microbiome (Evariste et al., 2019). The continued search for new bioindicators of fish health derives from limits on the quality of information obtained from most indicators used to date or constraints on their broad implementation for ecosystem management.

An understudied aspect of growth is the allocation of resources to organ systems under energetic constraints. The growth of tissues that are more energetically expensive to maintain, such as the brain and digestive tract (Niven & Laughlin, 2008), is likely to be under intense pressure in conditions of reduced energy availability. Contaminants can contribute to reduce energy availability and growth by reducing feeding opportunities of fish, through the catabolic effect of chronic stress (Barton & Iwama, 1991), or by direct inhibition of cell proliferation or increased cell death in exposed tissues (Lijoy et al., 2006). Therefore, in the spirit of the adverse outcome pathway (AOP) framework for ecotoxicology risk assessment (Ankley et al., 2010), we assessed different levels of biological organization that may contribute to reduced growth, and thus fitness. We assessed the somatic and organ growth of wild fish exposed to different levels of pollution against measures of exposure to contaminants (liver *cyp1a1* expression), chronic stress status (scale cortisol content), and foraging behavior (trophic position and isotopic niche width) to evaluate the usefulness of bioindicators of stress status and foraging ecology in assessing the sublethal effects of pollution on aquatic wildlife. We hypothesized that legacy industrial pollution in the form of elevated levels of persistent organic chemicals and heavy metals in sediments would put local fish under energetic constraints, reducing their ability to grow and maintain energetically expensive organs to the size of counterparts inhabiting less polluted sites.

## MATERIALS AND METHODS

### Sampling sites

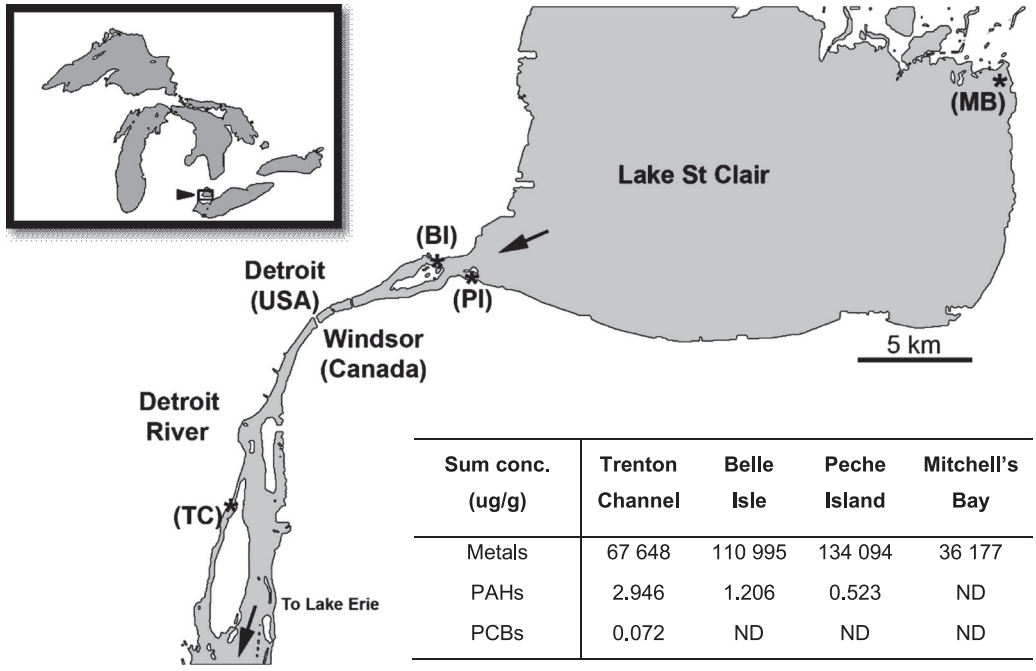
The chosen study system of the Lake St. Clair–Detroit River corridor displays a gradient of legacy industrial pollution increasing downstream along the Detroit River that can be measured in sediments (Drouillard et al., 2006; Farwell et al., 2012, 2013; Heidtke et al., 2006; Jia et al., 2010) and includes an area of concern recognized by the International Joint Commission created by the United States and Canada (International Joint Commission United States and Canada, 1987). Yellow perch (*P. flavescens*) was studied because local populations show site fidelity and little exchange of individuals between sites (Sullivan & Stepien, 2014), thereby ensuring that the contaminant exposure of specimens was limited to local conditions. Yellow perch and forage fish were caught by boat electrofishing and hoop nets from three locations along the Detroit River (Trenton Channel, Belle Isle, and Peche Island) and one location in Lake St. Clair (Mitchell's Bay). Figure 1 shows a map of the sampling sites and associated sediment contamination levels. Total sample sizes of adult yellow perch were as follows: Trenton Channel (34), Belle Isle (39), Peche Island (38), and Mitchell's Bay (30). Sampling procedures were approved by the Ontario Ministry of Natural Resources (permits LE-07-2017 and LE-07-2018) and the University of Guelph animal care committee (protocol 3682) under the guidelines of the Canadian Council on Animal Care. Mitchell's Bay in Lake St. Clair was selected as a reference site due to its location upstream of the Detroit River, with no historical record of industrial pollution. However, agricultural activity is higher near this reference site, which prompted us to evaluate the presence of agricultural pesticides at the sampling sites.

### Sediment analyses

Analyses of contaminants targeted sediments because persistent organic industrial chemicals are poorly water soluble but available to benthic organisms inhabiting sediments from where they can enter the food chain (Jaffé, 1991). Sediment samples were obtained using an Ekman grab sampler and stored in light-protected, acetone-washed Mason jars at −20 °C until analysis. Three sediment samples obtained at each site were combined to form a single composite sample representative of each site. Samples were stored less than 2 weeks before screening for polyaromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), and heavy metals by Maxxam Analytics. Analytical procedures followed standard United States Environmental Protection Agency methods (PAHs, PCBs, and heavy metals) or the Canadian Food Inspection Agency PMR 006 V1.0 method (pesticides). A more detailed description of sediment contaminant analyses is provided in Supporting Information, Section S1 (Supporting Information, Table S1).

### Liver gene expression

Quantification of *cyp1a1* liver gene expression by quantitative polymerase chain reaction (qPCR) followed the methods of Williams et al. (2017), which are detailed in Supporting Information, Section S2, along with primer nucleotide sequences (Supporting Information, Table S2). Liver cyp1a1 expression was measured in all adult yellow perch sampled, except for three individuals that showed poor RNA extraction.




| Sum conc. (ug/g) | Trenton Channel | Belle Isle | Peche Island | Mitchell's Bay |
|---|---|---|---|---|
| Metals | 67 648 | 110 995 | 134 094 | 36 177 |
| PAHs | 2.946 | 1.206 | 0.523 | ND |
| PCBs | 0.072 | ND | ND | ND |

**FIGURE 1:** Sampling sites in the Lake St. Clair–Detroit River system. Lake St. Clair and Detroit River are located between Lakes Huron and Erie of the North American Laurentian Great Lakes (arrowhead in top left inset). Water flow is from Lake St. Clair to the Detroit River and then to Lake Erie, as indicated by arrows in the main figure. The location of sampling sites is indicated by asterisks (BI, Belle Isle; MB, Mitchell's Bay; PI, Peche Island; TC, Trenton Channel). Mitchell's Bay and TC are the most upstream and downstream sampling sites, respectively. The table in the lower part of the figure lists sum concentrations of persistent industrial contaminants in the sediments at each site (ND = not detected; PAHs = polycyclic aromatic hydrocarbons; PCBs = polychlorinated biphenyls).

## Scale cortisol content

Cortisol was extracted from scales of 30 adult perch per site based on the methods of Laberge et al. (2019), and then measured in duplicate aliquots using a commercial cortisol enzyme-linked immunosorbent assay kit (product code *402710*; Neogen). To estimate the recovery of hormone through the extraction process, $^{3}$H-cortisol was added to scale samples and traced through the extraction process. The reconstituted extracts contained 84.8 ± 1.6% of the $^{3}$H label ($n = 4$). Scale cortisol content values are given as nanograms of cortisol per gram of dried scale and are corrected for efficiency of the recovery of $^{3}$H-cortisol. More details of the methods and validation of the cortisol assay by a serial dilution of scale extract displacement curve parallel to the standard curve (Supporting Information, Figure S1) are provided in Supporting Information, Section S3.

## Stable isotopes and trophic ecology

Stable isotope analyses of carbon ($^{13}$C/$^{12}$C) and nitrogen ($^{15}$N/$^{14}$N) in fish muscle were performed at the Great Lakes Institute of Environmental Research (University of Windsor, ON, Canada) as per Mumby et al. (2018). Supporting Information, Section S4, provides details of the stable isotope analyses performed to characterize the trophic ecology of individual adult yellow perch. All adult yellow perch were used for these analyses, except one individual due to muscle sample loss. A summary of stable isotope data can be seen in Supporting Information, Table S3.

## Body and organ measurements

Immediately after capture, yellow perch were terminally anesthetized using tricaine methanesulfonate (250 mg/L; Syndel International), weighed, and dissected to obtain their livers. Livers were excised, weighed, and immediately wrapped in tinfoil before freezing and storage in liquid nitrogen to allow gene expression analysis. The remaining carcasses were put on ice until further dissection later the same day. The gut and gonad were excised and weighed fresh. Gut content was expelled before weighing. Scales from the whole body surface and a sample of muscle from the dorsal musculature just caudal to the dorsal fin were also collected at this time and stored frozen for subsequent analyses of cortisol content and stable isotopes, respectively (see details below). Hearts were excised and stored in 20-mL glass vials filled with 10 mL of 10% buffered formalin until further dissection and weighing of the ventricle later in the laboratory using a high-resolution scale (Accu-124D; Fisher Scientific). The heads of fish were also stored in formalin until further dissection, trimming, and weighing of the brain later in the laboratory, therefore the masses of the heart ventricle and brain were obtained from fixed tissue. Three brains and three heart ventricles were rejected from analyses due to dissection damage. Otoliths were extracted during brain dissection and used for fish age determination by counting annuli on bisected and polished otoliths. This method is more precise than counting annuli on scales in yellow perch (Robillard & Mardsen, 1996). Annuli were counted under a dissection microscope by two observers until agreement was reached on the age of each fish.

### Statistical analysis

Correction of organ size data for individual differences in body size was required because of normal organ growth with increasing body size. Organ growth curves were obtained by graphing organ mass against body mass. The data were not log-transformed because this did not improve linear fits. Best fits between linear and quadratic relationships were assessed in Prism version 8.4.3 (GraphPad Software) for each organ growth curve. Only brain growth showed a quadratic instead of linear best fit. Residual values from these relationships, representing the distance away from the fitted curve, were used to represent individual relative organ size in all further analyses.

Between-site analyses were first conducted to confirm if characteristics of yellow perch from historically polluted sites varied compared with our reference site. Between-site data consistently violated the assumption of normality as determined by the Shapiro–Wilk test, so a Kruskal–Wallis test and, on statistical significance, Dunn's multiple comparisons test were used to determine which sites differed from one another. General linear models (or generalized linear models [GLMs] in the case of non-normal data) were then used to assess somatic growth and determine if age, *cyp1a1* liver mRNA expression levels, scale cortisol content, and relative trophic position explained observed differences in relative organ size between sites. Relative trophic position was used in the present study to account for the expected increase in trophic position with body size. The residuals obtained from the linear relationship between body mass and trophic position were used to represent relative trophic position. Several models were tested with age, *cyp1a1* liver mRNA levels, scale cortisol content, and relative trophic position as covariates, site as a fixed effect, and site*age, site*cyp1a1, site*scale cortisol, and site*relative trophic position as interaction terms. These models were followed by least significant difference pairwise comparisons to assess divergence in relationships between sites. Between-site analyses were conducted in Prism 8.4.3 (GraphPad Software), while GLMs were conducted in SPSS Statistics Ver. 26 (IBM). The statistical threshold used was 0.05.

## RESULTS AND DISCUSSION

### Legacy of industrial pollution in the Detroit River

The Detroit River was identified as a site in need of remedial action in 1978 (International Joint Commission United States and Canada, 1987). In 2010, the Detroit River Remedial Action Plan Stage 2 Report highlighted the occurrence of tumors and deformities in fish, benthos degradation, and degradation of fish, phytoplankton, and zooplankton populations due to legacy industrial pollution (Green et al., 2010). Some of these issues have been resolved in recent years, such as reduced occurrence of fish deformities and benthos degradation, but continued degradation of fish, phytoplankton, and zooplankton populations persist (Detroit River Canadian Cleanup, 2021). Of the three sites located along the Detroit River, the downstream site of Trenton Channel had the highest sum concentrations of PAHs and PCBs in sediments, while Peche Island had the highest sum concentration of heavy metals (Figure 1). Belle Isle had intermediate sum concentrations of heavy metals and PAHs among the three Detroit River sites. Mitchell's Bay, our reference site located upstream of the Detroit River in Lake St. Clair, had the lowest sum concentration of metals and nondetectable levels of PAHs and PCBs. Observed contamination levels of the persistent organic chemicals formed a gradient of increasing sum concentrations from Lake St. Clair (Mitchell's Bay) to the mouth of the Detroit River (Peche Island and Belle Isle) to the site furthest downstream on the Detroit River (Trenton Channel), consistent with past studies on these contaminants in sediment (e.g., Falk et al., 2019). A detailed breakdown of compounds detected in sediments from each site can be found in Supporting Information, Table S1. Of note, the pesticide screen of sediments only detected a low concentration of breakdown products of the banned organochlorine insecticide dichlorodiphenyltrichloroethane (DDT; p,p-dichlorodiphenyldichloroethylene and p,p-dichlorodiphenyldichloroethane) at Belle Isle. No pesticide was detected elsewhere.

The cytochrome P450 (CYP) 1A subfamily of enzymes is induced by the binding of various environmental contaminants to a cytosolic aryl hydrocarbon receptor (Ahr; Bucheli & Fent, 1995; Stegeman & Lech, 1991). The CYP1A enzyme activity is commonly used as a biomarker of contaminant exposure using the 7-ethoxyresorufin o-deethylase (EROD) assay (Whyte et al., 2000). However, EROD activity declines rapidly in frozen liver samples (Förlin & Andersson, 1985), which prompted us to choose *cyp1a1* gene expression as an alternative indicator of contaminant exposure better suited to field sampling and longer sample storage. Increased expression of *cyp1a1* contributes to increase CYP1A catalytic activity and it persists for weeks or months in fish liver after contaminant exposure (Courtenay et al., 1999; Jeon et al., 2011; Sorrentino et al., 2005). This method is increasingly used to evaluate contaminant exposure in fish (Brammell et al., 2010; Gao et al., 2011; Jönsson et al., 2010; Wong et al., 2001). The mRNA levels of *cyp1a1* measured in the liver of yellow perch significantly differed between sites ($H(138) = 22.87$, $p < 0.0001$), with fish from Mitchell's Bay showing the lowest median levels of *cyp1a1* expression and fish from Trenton Channel showing the highest (Figure 2). Dunn's multiple comparisons test found that fish from Peche Island ($p = 0.01$) and Trenton Channel ($p < 0.001$) had significantly higher levels of *cyp1a1* expression than fish from Mitchell's Bay by an average of 53% and 102%, respectively, but that the 52% average difference between Mitchell's Bay and Belle Isle *cyp1a1* expression levels was only close to the statistical threshold ($p = 0.06$). These data on gene expression of a liver detoxification enzyme generally paralleled the contamination gradient found in the sediments at the four sampling sites and imply that contaminant exposure experienced by yellow perch is higher in the Detroit River and highest on average at the most downstream site of Trenton Channel. These results are in line with observations of continued degradation of fish populations in the Detroit River due to legacy industrial pollution (Detroit River Canadian Cleanup, 2021).

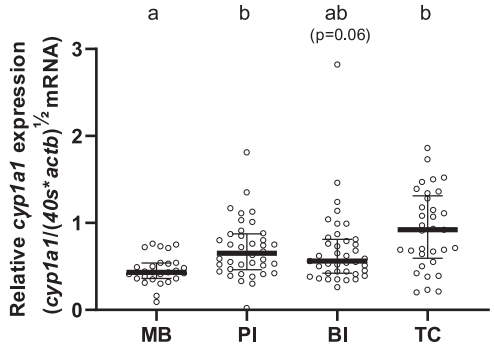



**FIGURE 2:** Liver *cyp1a1* expression of in yellow perch (*Perca flavescens)* across four test sites quantified using real-time polymerase chain reaction (qPCR). Values of *cyp1a1* expression are normalized to the geometric mean of the reference genes *40s* and *actb*. Bars represent median ± interquartile range. Letters denote differences between sites obtained by Dunn's post hoc test. The comparison between MB and BI is close to statistical significance ($p = 0.06$). BI = Belle Isle (sample size $n = 39$); MB = Mitchell's Bay ($n = 28$); PI = Peche Island ($n = 38$); TC = Trenton Channel ($n = 33$).

## Corticosteroid hormone status and pollution

We found a significant difference in scale cortisol content between sites ($H(120) = 17.8$, $p = 0.0005$; Figure 3), where Trenton Channel yellow perch had significantly lower median scale cortisol levels than perch at the other three sites ($p < 0.05$). There was no difference in perch scale cortisol content between the three other sites. Perch of Trenton Channel had on average 49% less scale cortisol content compared with perch of the other three sites.

Because scale cortisol provides an integrated measure of cortisol production (Laberge et al., 2019), the reduced scale cortisol content of yellow perch from Trenton Channel suggests that the mixture of contaminants at our most polluted site chronically reduced circulating cortisol levels and/or inhibited the capacity to respond to stressors. While acute exposure to various contaminants in captivity can stimulate the endocrine stress response and increase plasma cortisol levels (Gagnon et al., 2006; Gesto et al., 2008; Hontela et al., 1996; Lin et al., 2022; Rohonczy et al., 2021; Thomas & Rice, 1987), chronic exposure to complex mixtures of PCBs, PAHs, and heavy metals under field conditions inhibits stress responsiveness in various species, including yellow perch (Girard et al., 1998; Gravel et al., 2005; Hontela et al., 1992, 1995). In laboratory studies, contaminants that activate the Ahr and increase *cyp1a1* expression can inhibit key rate-limiting steps involved in interrenal cortisol synthesis (Aluru & Vijayan, 2006), but we did not find any relationship between yellow perch liver *cyp1a1* expression and scale cortisol content (data not shown). Also, while yellow perch from contaminated sites generally had higher levels of *cyp1a1* expression than fish from Mitchell's Bay, only the perch from Trenton Channel were characterized by lower scale cortisol content. Our results suggest that scale cortisol may be a useful bioindicator of environmental conditions that lead to adrenotoxicity in fish, a diagnosis which should be confirmed in follow-up studies using established in vitro interrenal bioassays and/or stress challenge protocols (Hontela, 2005).

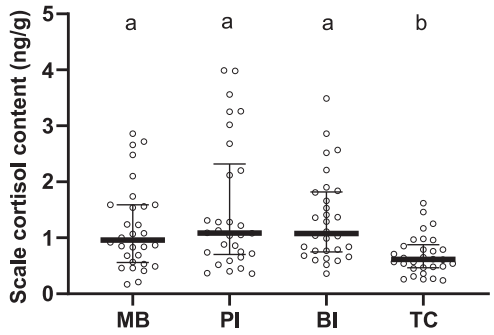



**FIGURE 3:** Scale cortisol content in yellow perch (*Perca flavescens)* across four test sites quantified using an enzyme-linked immunosorbent assay (ELISA). Values are expressed in nanogram cortisol per gram of scales. Bars represent median ± interquartile range. Letters denote differences between sites obtained by Dunn's post hoc test. BI = Belle Isle; MB = Mitchell's Bay; PI = Peche Island; TC = Trenton Channel. Sample size is 30 perch per site.

## Effects of pollution on trophic ecology

There was a significant difference in trophic position between sites ($H(140) = 93.00$, $p < 0.0001$; Figure 4A). Yellow perch from Trenton Channel had significantly lower trophic positions than perch from the other three sites (mean difference 1.2, $p < 0.0001$), while Belle Isle perch also had significantly higher trophic positions than Mitchell's Bay (mean difference 0.4, $p < 0.001$). Mitchell's Bay and Peche Island perch showed no significant difference in trophic position. Similar trends in trophic positions between these sites have been observed in other species of fish, suggesting variation in food web structure (Nawrocki et al., 2016, 2020). The $d^{15}N$ predator to forage fish ratio was also analyzed to assess if perch occupied a typical trophic level above that of small forage fish at the four sites. This measure differed between sites ($H(140) = 102.5$, $p < 0.0001$; Figure 4B) and perch from Trenton Channel had significantly lower $d^{15}N$ predator to forage fish ratios than perch at the other three sites (mean difference 0.38, $p < 0.001$). Notably, Trenton Channel was the only site to have $d^{15}N$ predator to forage fish ratios lower than 1. As with trophic position, we found that Belle Isle perch had the highest median $d^{15}N$ predator to forage fish ratio of all four sites.

Differential $\delta^{13}C$ of adult perch and forage fish also differed between sites ($H(140) = 49.1$, $p < 0.0001$; Figure 4C), but in this case it was higher in yellow perch of Belle Isle and Peche Island in comparison with the perch of Mitchell's Bay and Trenton Channel, which did not differ from each other. Similar to trophic position, variation in $\delta^{13}C$ between these sites in other species of fish has been documented (Nawrocki et al., 2016, 2020).

Finally, isotopic niche width and overlap based on trophic position and differential $\delta^{13}C$ muscle signatures of adult perch and forage fish were analyzed to assess potential differences in diet between sites. Of our four sites, Mitchell's Bay had the smallest isotopic niche width with a standard 40% ellipse area (SEA) of $0.58 \pm 0.1$, Peche Island had the largest at $1.39 \pm 0.2$, while Belle Isle (SEA = $1.04 \pm 0.2$) and Trenton Channel (SEA = $0.92 \pm 0.2$) had intermediate values (Figure 4D). The isotopic

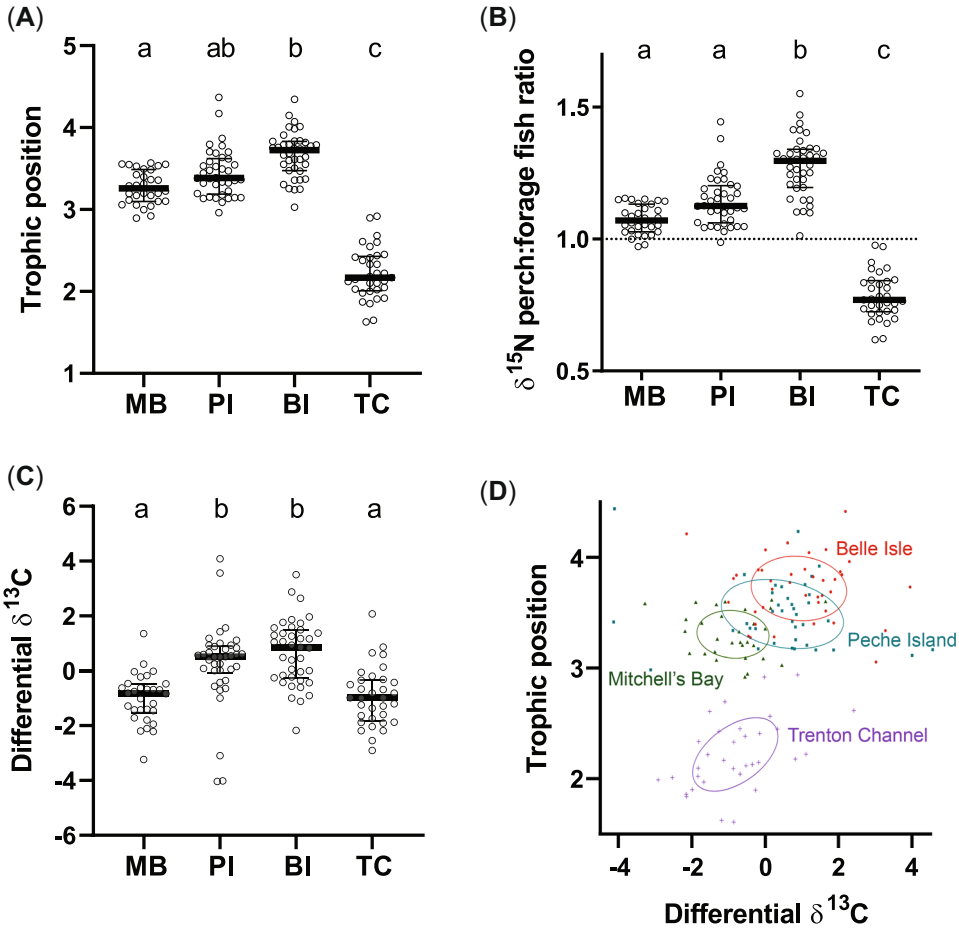



**FIGURE 4:** Yellow perch (*Perca flavescens*) trophic ecology across four test sites in the Lake St. Clair–Detroit River system. (**A**) Trophic position calculated using nitrogen stable isotope ratios of yellow perch and forage fish. (**B**) $\delta^{15}$N ratios of perch to forage fish. A $\delta^{15}$N ratio higher than 1 indicates that yellow perch are at a higher trophic position than forage fish (typical of an average food web), while a $\delta^{15}$N ratio lower than 1 indicates yellow perch are at a lower trophic position than forage fish (atypical of an average food web). (**C**) Differences in the $\delta^{13}$C of adult perch and the mean $\delta^{13}$C of forage fish at each site. Bars in (**A**)–(**C**) represent median ± interquartile range. Letters denote differences between sites obtained by Dunn's post hoc test. (**D**) Isotopic niche width of adult perch at each site based on differential $\delta^{13}$C and trophic positions were determined using 40% density Bayesian prediction intervals with the R package nicheROVER. BI = Belle Isle (sample size $n = 39$); MB = Mitchell's Bay ($n = 30$); PI = Peche Island ($n = 38$); TC = Trenton Channel ($n = 33$).

niche at Trenton Channel showed the smallest range of isotopic niche overlap with the other sites (with Mitchell's Bay 0.02%–0.08%, Peche Island 0.03%–0.1%, Belle Isle 0%–0.02%). The isotopic niches of Belle Isle and Mitchell's Bay also showed little overlap at 3%–4%. The greatest niche overlap was seen between Peche Island and Belle Isle (23%–34%) and Peche Island and Mitchell's Bay (10%–29%).

Our stable isotope results imply strong trophic disruption at Trenton Channel, providing evidence that the food web and trophic relationships are still impacted by legacy contamination at this site. Yellow perch are generalist foragers that will normally switch diet from small to large benthic invertebrates and forage fish as they grow larger (Hayward & Margraf, 1987; Iles & Rasmussen, 2005; Knight et al., 1984; Morrison et al., 1997; Parrish & Margraf, 1994; Sherwood et al., 2002). Their ability to exploit lesser value prey opportunistically allows them to occupy impacted habitats, sometimes at the cost of stunted growth (e.g., Sherwood et al., 2002). We observed that most perch sampled from Trenton Channel had digestive tracts filled with undigested remains of snail shells, a feature not seen in perch captured at other sites. Extreme reliance on snail prey may explain the low trophic position and relatively narrow isotopic niche width of adult Trenton Channel perch considering that snails have low nitrogen isotopic signatures (Post, 2002) and typically do not occur in the diet of yellow perch (Elrod et al., 1981; Keast, 1977). Disruption of yellow perch foraging patterns has been observed in metal-contaminated lakes (Iles & Rasmussen, 2005; Sherwood et al., 2002), although the concentrations in these studies were much higher (3–40 times) than those measured in our study. Thus, it is unlikely that metal contamination contributed to disruption of yellow perch trophic ecology at Trenton Channel. This contention is also supported by the observation that metal contamination at Trenton Channel was lower than the other two Detroit River sites studied. The most likely cause of trophic disruption at Trenton Channel is elevated PAH and PCB contamination; two categories of organic contaminants known for organismal bioaccumulation and trophic transfer (Honda & Suzuki, 2020; Russell et al., 1999).

## Somatic growth

We examined the possibility that environmental contamination could influence somatic growth of perch by assessing the relationships between age and body mass between sites (Figure 5). Our generalized linear model found a significant interaction effect between age and site on the mass of perch ($\chi^2(8) = 16.6$, $p = 0.03$), with post hoc pairwise comparisons indicating that fish from Peche Island attain a higher mass than fish from Mitchell's Bay at ages 2 ($p = 0.02$), 3 ($p = 0.001$), and 4 ($p < 0.001$) years. Fish from Belle Isle also have a higher mass than fish from Mitchell's Bay, but only at age 4 years ($p = 0.03$). The body mass of Trenton Channel perch did not significantly differ from the other sites at any age ($p > 0.12$). Overall, it appears that perch from Peche Island grow faster from their second year of life compared with perch sampled from our reference site and a similar trend is found later in life in perch from Belle Isle, but no difference in somatic growth is found between perch from Trenton Channel and perch from our reference site Mitchell's Bay.

The disrupted trophic relationships found at Trenton Channel did not hinder perch somatic growth. Such an absence of effect on perch somatic growth has already been noted in a study of legacy PCB contamination in the Hudson River, New York (Maceina & Sammons, 2018). The absence of effects of exposure to persistent organic pollutants at environmentally relevant concentrations on growth of adult fish seems to be the norm (Johnson et al., 2013). It is unclear why perch from Peche Island (and to some extent Belle Isle) could attain larger sizes than Mitchell's Bay perch as they aged, but perch of Peche Island had the broadest isotopic niche of all sites, suggesting more diverse prey availability, which may promote fish growth (Hayden et al., 2014).

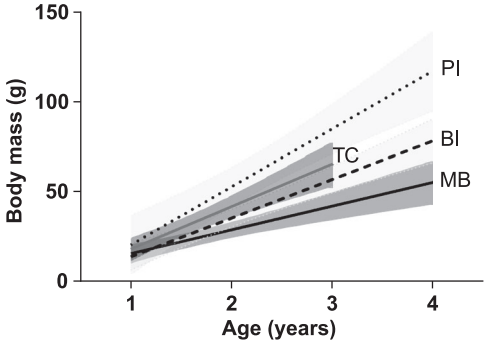



**FIGURE 5:** Growth of yellow perch (*Perca flavescens*) across four test sites in the Lake St. Clair–Detroit River system. Age was determined by counting annuli on bisected and polished otoliths. Lines of best fit and 95% confidence bands were determined by linear regression for each site. The appearance of different lines of fit and confidence bands was varied to distinguish overlapping growth curves. A significant divergence in growth for Mitchell's Bay and Peche Island perch is seen from age 2 years onwards, and divergence in growth between Belle Isle and Mitchell's Bay perch is seen at age 4 years. BI = Belle Isle (sample size $n = 39$); MB = Mitchell's Bay ($n = 30$); PI = Peche Island ($n = 38$); TC = Trenton Channel ($n = 34$).

## Organ growth

We examined the possibility that environmental contamination could influence organ growth first by assessing between-site differences in relative organ size, followed by a general linear model approach (or generalized linear model if the data did not meet assumptions) to determine which factors contributed to observed differences between sites. Preliminary models assessed age, *cyp1a1* expression and scale cortisol content due to between-site differences in these factors, but neither age, *cyp1a1* expression levels, scale cortisol content nor interaction terms including these factors (age*site, *cyp1a1**site, scale CORT*site) had any significant effects on relative organ sizes (Supporting Information, Table S4 in Section S5). As such, our final models for relative organ sizes only examined relative trophic position, site, and the interaction of relative trophic position with site.

We found a significant difference in relative brain size between sites ($H(138) = 53.1$, $p < 0.0001$; Figure 6A), with Trenton Channel perch showing significantly smaller brains than perch at the other three sites ($p < 0.001$). We assessed this between-site difference in relative brain size with a GLM including relative trophic position as a covariate due to previous observations of a positive relationship between trophic position and relative brain size in fishes (Edmunds et al., 2016; Kondoh, 2010). Our model showed that relative brain size was positively associated with relative trophic position ($\eta_p^2 = 0.25$, $F(1) = 43.9$, $p < 0.001$), site ($\eta_p^2 = 0.10$, $F(3) = 5.0$, $p = 0.003$), and the presence of a significant interaction between site and relative trophic position ($\eta_p^2 = 0.16$, $F(3) = 8.2$, $p < 0.001$). This significant interaction might be explained by Mitchell's Bay's more positive relationship ($r = 0.19$) between trophic position and brain size compared with other sites (Belle Isle $r = 0.07$, Trenton Channel $r = 0.04$), especially compared with Peche Island, which shows only a small positive correlation coefficient ($r = 0.01$) for this relationship. Although scale cortisol content was lower in perch of Trenton Channel, it was not associated with relative brain size in the GLM analysis (Supporting Information, Table S4). Given the essential roles of cortisol for the promotion of normal brain development and maturation across vertebrates (Best et al., 2017; Moisiadis & Matthews, 2014; Trejo et al., 2000), we cannot exclude the possibility that the smaller brain size of Trenton Channel perch is the result of early life programming associated with impaired corticosteroidogenesis.

We found a significant difference in relative gut size between sites ($H(141) = 40.7$, $p < 0.0001$; Figure 6B), with Mitchell's Bay perch showing significantly smaller guts than perch at the other three sites ($p < 0.01$). When assessing this between-site difference in relative gut size using GLM, we found that relative gut size was positively associated with relative trophic position ($\eta_p^2 = 0.09$, $F(1) = 12.9$, $p < 0.001$), but that there was no interaction between site and relative trophic position ($\eta_p^2 = 0.27$, $F(3) = 1.2$, $p = 0.3$). The smaller gut size of perch seen at our reference site Mitchell's Bay is puzzling. A confounding effect of competition may have influenced both somatic and gut growth patterns there. This inference is based on

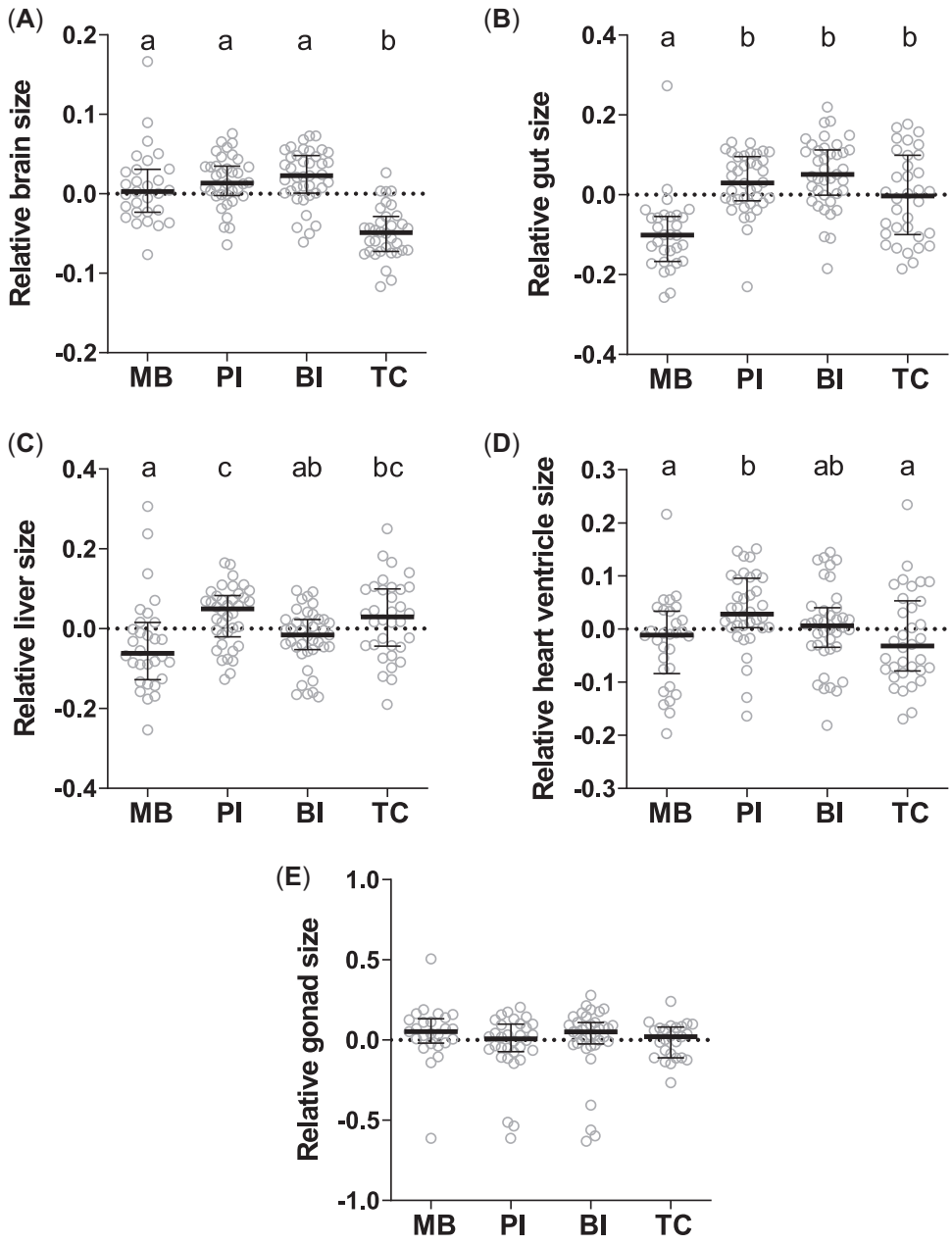



**FIGURE 6:** Site differences in relative organ size of yellow perch (*Perca flavescens*) in the Lake St. Clair–Detroit River system. (**A**) Relative brain size, (**B**) relative gut size, (**C**) relative liver size, (**D**) relative heart ventricle size, and (**E**) relative gonad size. The relative size of organs was obtained from residuals of linear (all organs, except brain) or quadratic (brain only) relationships between body mass and organ mass including perch of all sites. Bars represent median and interquartile range. Letters denote differences between sites obtained by Dunn's post hoc test. BI = Belle Isle; MB = Mitchell's Bay; PI = Peche Island; TC = Trenton Channel. Samples sizes are BI (39), MB (30), PI (38), and TC (34) everywhere, except for (**A**) (MB = 29, PI = 36) and (**D**) (BI = 38, PI = 36).

the small isotopic niche width of perch from Mitchell's Bay compared with the Detroit River sites, possibly indicating a more specialized diet. Isotopic niche width compression under intense interspecific competition has been observed in Arctic charr *Salvelinus alpinus* (Sandlund et al., 2016) and gut length is associated with diet composition and quality in fish (German & Horn, 2006; Olsson et al., 2007). Perhaps the primary prey type eaten by perch at Mitchell's Bay does not require a large gut to digest or a growth-promoting generalist diet (Hayden et al., 2014) of Detroit River perch may require increased digestion capacity supported by a larger gut.

We found a significant difference in relative liver size between sites ($H(141) = 17.8$, $p = 0.0005$; Figure 6C). Mitchell's Bay perch had significantly smaller livers than Peche Island and Trenton Channel perch ($p < 0.05$). We assessed this between-site difference in relative liver size using GLM and found that it was affected only by site ($\chi^2(3) = 9.2$, $p = 0.03$), with no significant effect of relative trophic position ($\chi^2(1) = 3.7$, $p = 0.06$) or the interaction between site and relative trophic position ($\chi^2(3) = 2.1$, $p = 0.56$). The liver hypertrophy seen at Peche Island and Trenton Channel matches previous observations of liver response to contaminant exposure in fish (Agbohessi

et al., 2015; Burlakov et al., 2021; Louiz et al., 2018; Tenji et al., 2020; Wolf & Wolfe, 2005). We suspected that this hepatic growth under energetic limitation would come at the expense of other organs, specifically energetically expensive organs like the brain or gut. However, normal brain size at Peche Island and normal gut size at both sites showing liver hypertrophy offer no support for this view.

We found a significant difference in relative heart ventricle size between sites ($H(138) = 13.8$, $p = 0.003$; Figure 6D), with Peche Island perch showing significantly larger heart ventricles than perch of Mitchell's Bay and Trenton Channel ($p < 0.05$). We assessed this between-site difference in relative heart ventricle size using GLM and found a significant effect of site ($\eta_p^2 = 0.08$, $F(3) = 3.5$, $p = 0.02$) and an interaction between site and relative trophic position ($\eta_p^2 = 0.07$, $F(3) = 3.4$, $p = 0.02$). This significant interaction might be explained by a divergent negative relationship between relative trophic position and relative heart ventricle size at Belle Isle ($r = -0.14$) compared with weak positive relationships at the other sites ($r$ between 0.03 and 0.1). Belle Isle was the only site where organochlorine DDT metabolites were detected in sediments and such contamination is known to transfer to fish tissues in food webs (Di et al., 2017; Jürgens et al., 2016). Thus, potentially cardiotoxic bioaccumulation of DDT metabolites with increasing trophic position may have reduced heart size in Belle Isle perch. Previous work in fish showed that DDT can accumulate in the heart of Atlantic salmon (Premdas & Anderson, 1963) and that exposure can slow heart rate in early-stage zebrafish (Ton et al., 2006). Mice exposed to DDT perinatally showed enlarged (not smaller) heart ventricles as adults, but most effects seen in mammals are related to hypertension and not direct effects on the heart (La Merrill et al., 2016). The relationship between trophic position and heart ventricle size warrants further investigation to verify potential deleterious effects of chronic exposure to DDT metabolites and their interaction with industrial contaminants known to be cardiotoxic (Incardona & Scholz, 2017) on fish heart growth and function.

Our sample had a strong female-biased sex ratio (80%), with only 4% males and 16% perch of undetermined sex, which were probably immature. We found no significant difference in relative gonad size between sites in mature perch ($H(119) = 4.6$, $p = 0.21$; Figure 6E). Removal of males did not change the outcome of this analysis ($H(113) = 6.9$, $p = 0.08$).

Site differences in perch growth patterns may have been influenced by competition as much as pollution. We hypothesized that legacy industrial pollution would put fish under energetic constraints and reduce their ability to grow and maintain energetically expensive organs like the brain and gut. Energetic constraints were not obvious from somatic growth patterns between sites because perch from our Lake St. Clair reference site showed the slowest growth and perch from the most polluted site—Trenton Channel—showed somatic growth on par with the fastest growing perch at Peche Island. Reduced somatic and gut growth at Mitchell's Bay may be due to increased competition for resources, as indicated by a small isotopic niche width, while reduced brain growth with normal somatic growth at Trenton Channel is associated with pollution-induced trophic disruption.

The variables measured in the present study allowed us to explore potential mechanisms underlying the effects of pollution on growth. A direct toxic effect of pollution would see an association between increased liver detoxification activity estimated by liver *cyp1a1* expression and growth inhibition, but *cyp1a1* expression was not associated with within-site variation in perch organ sizes. Baring the development of resistance to *cyp1a1*-inducing contaminants in perch (Bello et al., 2001; Courtenay et al., 1999), a plausible explanation for the mismatch between patterns of *cyp1a1* expression and organ size is that contamination did not directly influence organ growth. Unlike *cyp1a1* expression, trophic position was associated with variation in brain, gut, and heart ventricle size, suggesting that organ growth may be influenced by indirect effects on food webs. One candidate indirect effect of pollution is revealed by the co-occurrence of smaller perch brains and trophic disruption at Trenton Channel. Trophic disruption may reflect reduced prey abundance and/or quality for perch, thereby reducing energy available through foraging to invest in growth of the energetically expensive nervous system (Niven & Laughlin, 2008). Such small brains may further impact behavioral abilities related to prey capture, as seen in fish exposed to PCBs in laboratory or field conditions (Carvalho et al., 2004; Weis et al., 2001), and thus small relative brain size may represent an indicator of behavioral disruption. Alternatively, brain size may be reduced to save energy if foraging at low trophic position does not present a strong cognitive challenge requiring a large brain (Edmunds et al., 2016; Kondoh, 2010). Even though we cannot exclude the possibility that early life adrenotoxicity is responsible for irreversible reduction in perch brain growth at Trenton Channel or the direct inhibitory effect of early life PCB exposure on brain development (see King-Heiden et al., 2012), we consider these possibilities less likely because fish show indeterminate growth, conferring them the ability to grow their brains throughout life (Hariharan et al., 2016).

## SUMMARY AND CONCLUSIONS

Our study identified strong trophic disruption correlated with reduced yellow perch brain size at a Detroit River site impacted by legacy industrial pollution. Effects on perch growth were subtle and may have been influenced by competition (reduced somatic and gut growth at Mitchell's Bay) as much as pollution (reduced brain growth at Trenton Channel). As such, highlighting a potential limitation of the AOP concept under complex real-world exposure conditions, our results suggest that indirect effects such as trophic disruption can alter outcomes. Trophic disruption where competition is limited suggests that continued remediation efforts targeted at improving prey availability for fish may be beneficial. However, indicators of quality of the invertebrate community onto which fish feed are difficult to obtain (but not impossible, see Griffiths, 1991; McPhedran et al., 2016; Thornley, 1985). Thus, indicators of fish trophic ecology may be easier to implement for

monitoring efforts because trophic position and isotopic niche can be used as integrated measures of the prey community available to fish (Alp & Cucherousset, 2022; Layman et al., 2007; Yeakel et al., 2016). Ultimately, such measures of foraging ecology may prove useful indicators of fish and ecosystem health that could be used advantageously in combination with other indicators of pollution to improve environmental monitoring and assessment programs.

*Supporting Information*—The Supporting Information is available on the Wiley Online Library at https://doi.org/10.1002/etc.5701.

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## REFERENCES

Agbohessi, P., Toko, I., Ouedraogo, A., Jauniaux, T., Mandiki, S., & Kestermont, P. (2015). Assessment of the health status of wild fish inhabiting a cotton basin heavily impacted by pesticides in Benin (West Africa). *Science of the Total Environment*, *506–507*, 567–584. https://doi.org/10.1016/j.scitotenv.2014.11.047

Ali, H., Khan, E., & Ilahi, I. (2019). Environmental chemistry and ecotoxicology of hazardous heavy metals: Environmental persistence, toxicity, and bioaccumulation. *Journal of Chemistry*, *2019*, 6730305. https://doi.org/10.1155/2019/6730305

Ali, M., & Sreekrishnan, T. R. (2001). Aquatic toxicity from pulp and paper mill effluents: A review. *Advances in Environmental Research*, *5*, 175–196. https://doi.org/10.1016/S1093-0191(00)00055-1

de Almeida Rodrigues, P., Ferrari, R. G., Dos Santos, L. N., & Conte Junior, C. A. (2019). Mercury in aquatic fauna contamination: A systematic review on its dynamics and potential health risks. *Journal of Environmental Sciences*, *84*, 205–218. https://doi.org/10.1016/j.jes.2019.02.018

Alp, M., & Cucherousset, J. (2022). Food webs speak of human impact: Using stable isotope-based tools to measure ecological consequences of environmental change. *Food Webs*, *30*:e00218. https://doi.org/10.1016/j.fooweb.2021.e00218

Aluru, N., & Vijayan, M. M. (2006). Aryl hydrocarbon receptor activation impairs cortisol response to stress in rainbow trout by disrupting the rate-limiting steps in steroidogenesis. *Endocrinology*, *147*, 1895–1903. https://doi.org/10.1210/en.2005-1143

Ankley, G. T., Bennett, R. S., Erickson, R. J., Hoff, D. J., Hornung, M. W., Johnson, R. D., Mount, D. R., Nichols, J. W., Russom, C. L., Schmieder, P. K., Serrano, J. A., Tietge, J. E., & Villeneuve, D. L. (2010). Adverse outcome pathways: A conceptual framework to support ecotoxicology research and risk assessment. *Environmental Toxicology and Chemistry*, *29*, 730–741. https://doi.org/10.1002/etc.34

Barton, B. A., & Iwama, G. K. (1991). Physiological changes in fish from stress in aquaculture with emphasis on the response and effects of corticosteroids. *Annual Review of Fish Diseases*, *1*, 3–26. https://doi.org/10.1016/0959-8030(91)90019-G

Behera, B. K., Das, A., Sarkar, D. J., Weerathunge, P., Parida, P. K., Das, B. K., Thavamani, P., Ramanathan, R., & Bansal, V. (2018). Polycyclic aromatic hydrocarbons (PAHs) in inland aquatic ecosystems: Perils and remedies through biosensors and bioremediation. *Environmental Pollution*, *241*, 212–233. https://doi.org/10.1016/j.envpol.2018.05.016

Bello, S. M., Franks, D. G., Stegeman, J. J., & Hahn, M. E. (2001). Acquired resistance to Ah receptor agonists in a population of Atlantic killifish (*Fundulus heteroclitus*) inhabiting a marine superfund site: *In vivo* and *in vitro* studies on the inducibility of xenobiotic metabolizing enzymes. *Toxicological Sciences*, *60*, 77–91. https://doi.org/10.1093/toxsci/60.1.77

Best, C., Kurrasch, D. M., & Vijayan, M. M. (2017). Maternal cortisol stimulates neurogenesis and affects larval behaviour in zebrafish. *Scientific Reports*, *7*, 40905. https://doi.org/10.1038/srep40905

Blazer, V. S. (2002). Histopathological assessment of gonadal tissue in wild fishes. *Fish Physiology and Biochemistry*, *26*, 85–101. https://doi.org/10.1023/A:1023332216713

Bownik, A., & Wlodkowic, D. (2021). Applications of advanced neurobehavioral analysis strategies in aquatic ecotoxicology. *Science of the Total Environment*, *772*, 145577. https://doi.org/10.1016/j.scitotenv.2021.145577

Brammell, B. F., McClain, J. S., Oris, J. T., Price, D. J., Birge, W. J., & Elskus, A. A. (2010). CYP1A expression in caged rainbow trout discriminates among sites with various degrees of polychlorinated biphenyl contamination. *Archives of Environmental Contamination and Toxicology*, *58*, 772–782. https://doi.org/10.1007/s00244-009-9368-x

Bruneau, A., Landry, C., Giraudo, M., Douville, M., Brodeur, P., Boily, M., Gagnon, P., & Houde, M. (2016). Integrated spatial health assessment of yellow perch (*Perca flavescens*) populations from the St. Lawrence River (QC, Canada), part B: Cellular and transcriptomic effects. *Environmental Science and Pollution Research*, *23*, 18211–18221. https://doi.org/10.1007/s11356-016-7001-x

Bucheli, T. D., & Fent, K. (1995). Induction of cytochrome P450 as a biomarker for environmental contamination in aquatic ecosystems. *Critical Reviews in Environmental Science and Technology*, *25*, 201–268. https://doi.org/10.1080/10643389509388479

Bundy, J. G., Davey, M. P., & Viant, M. R. (2009). Environmental metabolomics: A critical review and future perspectives. *Metabolomics*, *5*, 3–21. https://doi.org/10.1007/s11306-008-0152-0

Burlakov, I., Volkova, I., Kryuchkov, V., Egorova, V., & Seidalieva, L. (2021). Adaptive and pathological responses of cyprinidae liver towards habitat in the Volga delta. *IOP Conference Series: Earth and Environmental Science*, *937*, 022068.

Cappello, T. (2020). NMR-based metabolomics of aquatic organisms. *eMagRes*, *9*, 81–100. https://doi.org/10.1002/9780470034590.emrstm1604

Carvalho, P. S. M., Noltie, D. B., & Tillitt, D. E. (2004). Biochemical, histological and behavioural aspects of visual function during early development of rainbow trout. *Journal of Fish Biology*, *64*, 833–850. https://doi.org/10.1111/j.1095-8649.2004.0350.x

Chen, L. (2020). Visual system: An understudied target of aquatic toxicology. *Aquatic Toxicology*, *225*, 105542. https://doi.org/10.1016/j.aquatox.2020.105542

Courtenay, S. C., Grunwald, C. M., Kreamer, G.-L., Fairchild, W. L., Arsenault, J. T., Ikonomou, M., & Wirgin, I. I. (1999). A comparison of the dose and

 

time response of CYP1A1 mRNA induction in chemically treated Atlantic tomcod from two populations. *Aquatic Toxicology*, *47*, 43–69. https://doi.org/10.1016/S0166-445X(99)00006-5

Defo, M. A., Douville, M., Giraudo, M., Brodeur, P., Boily, M., & Houde, M. (2018). RNA-sequencing to assess the health of wild yellow perch (*Perca flavescens*) populations from the St. Lawrence River, Canada. *Environmental Pollution*, *243B*, 1657–1668. https://doi.org/10.1016/j.envpol.2018.09.133

Detroit River Canadian Cleanup. (2021). *Pathway to delisting: Detroit River Canadian AOC Delisting Strategy* (Publication No. 2).

Di, S., Liu, R., Tian, Z., Cheng, C., Chen, L., Zhang, W., Zhou, Z., & Diao, J. (2017). Assessment of tissue-specific accumulation, elimination and toxic effects of dichlorodiphenyltrichloroethanes (DDTs) in carp through aquatic food web. *Scientific Reports*, *7*, 2288. https://doi.org/10.1038/s41598-017-02612-4

Drouillard, K. G., Tomczak, M., Reitsma, S., & Haffner, G. D. (2006). A River-wide survey of polychlorinated biphenyls (PCBs), polycylic aromatic hydrocarbons (PAHs), and selected organochlorine pesticide residues in sediments of the Detroit River—1999. *Journal of Great Lake Research*, *32*, 209–226. https://doi.org/10.3394/0380-1330(2006)32[209:ARSOPB]2.0.CO;2

Edmunds, N. B., Laberge, F., & McCann, K. S. (2016). A role for brain size and cognition in food webs. *Ecology Letters*, *19*, 948–955. https://doi.org/10.1111/ele.12633

Elrod, J. H., Busch, W.-D. N., Griswold, B. L., Schneider, C. P., & Wolfert, D. R. (1981). Food of white perch, rock bass and yellow perch in eastern Lake Ontario. *New York Fish and Game Journal*, *28*, 191–201.

Evariste, L., Barret, M., Mottier, A., Mouchet, F., Gauthier, L., & Pinelli, E. (2019). Gut microbiota of aquatic organisms: A key endpoint for eco-toxicological studies. *Environmental Pollution*, *248*, 989–999. https://doi.org/10.1016/j.envpol.2019.02.101

Falk, N., Reid, T., Skoyles, A., Grgicak-Mannion, A., Drouillard, K., & Weisener, C. G. (2019). Microbial metatranscriptomic investigations across contaminant gradients of the Detroit River. *Science of the Total Environment*, *690*, 121–131. https://doi.org/10.1016/j.scitotenv.2019.06.451

Farwell, M., Drouillard, K. G., Heath, D. D., & Pitcher, T. E. (2012). Acclimation of life-history traits to experimental changes in environmental contaminant concentrations in brown bullhead (*Ameiurus nebulosus*). *Environmental Toxicology and Chemistry*, *31*, 863–869. https://doi.org/10.1002/etc.1761

Farwell, M., Drouillard, K. G., Heath, D. D., & Pitcher, T. E. (2013). Associations between female reproductive traits and polychlorinated biphenyl sediment concentrations in wild populations of brown bullhead (*Ameiurus nebulosus*). *Archives of Environmental Contamination and Toxicology*, *65*, 742–752. https://doi.org/10.1007/s00244-013-9938-9

Fleeger, J. W., Carman, K. R., & Nisbet, R. M. (2003). Indirect effects of contaminants in aquatic ecosystems. *Science of the Total Environment*, *317*, 207–233. https://doi.org/10.1016/S0048-9697(03)00141-4

Förlin, L., & Andersson, T. (1985). Storage conditions of rainbow trout liver cytochrome P-450 and conjugating enzymes. *Comparative Biochemistry and Physiology B*, *80*, 569–572. https://doi.org/10.1016/0305-0491(85)90292-5

Gagnon, A., Jumarie, C., & Hontela, A. (2006). Effects of Cu on plasma cortisol and cortisol secretion by adrenocortical cells of rainbow trout (*Oncorhynchus mykiss*). *Aquatic Toxicology*, *78*, 59–65. https://doi.org/10.1016/j.aquatox.2006.02.004

Gao, K., Brandt, I., Goldstone, J. V., & Jönsson, M. E. (2011). Cytochrome P450 1A, 1B, and 1C mRNA induction patterns in three-spined stickleback exposed to a transient and a persistent inducer. *Comparative Biochemistry and Physiology C*, *154*, 42–55. https://doi.org/10.1016/j.cbpc.2011.02.005

German, D. P., & Horn, M. H. (2006). Gut length and mass in herbivorous and carnivorous prickleback fishes (Teleostei: Stichaeidae): Ontogenetic, dietary, and phylogenetic effects. *Marine Biology*, *148*, 1123–1134. https://doi.org/10.1007/s00227-005-0149-4

Gesto, M., Soengas, J. L., & Míguez, J. M. (2008). Acute and prolonged stress responses of brain monoaminergic activity and plasma cortisol levels in rainbow trout are modified by PAHs (naphthalene, β-naphthoflavone and benzo(a)pyrene) treatment. *Aquatic Toxicology*, *86*, 341–351. https://doi.org/10.1016/j.aquatox.2007.11.014

Girard, C., Brodeur, J. C., & Hontela, A. (1998). Responsiveness of the interrenal tissue of yellow perch (*Perca flavescens*) from contaminated sites to an ACTH challenge test in vivo. *Canadian Journal of Fisheries and Aquatic Sciences*, *55*, 438–450. https://doi.org/10.1139/f97-224

Giraudo, M., Bruneau, A., Gendron, A. D., Brodeur, P., Pilote, M., Marcogliese, D. J., Gagnon, C., & Houde, M. (2016). Integrated spatial health assessment of yellow perch (*Perca flavescens*) populations from the St. Lawrence River, Quebec, Canada) part A: Physiological parameters and pathogen assessment. *Environmental Science and Pollution Research*, *23*, 18073–18084. https://doi.org/10.1007/s11356-016-7002-9

Gravel, A., Campbell, P. G. C., & Hontela, A. (2005). Disruption of the hypothalamo-pituitary-interrenal axis in 1+ yellow perch (*Perca flavescens*) chronically exposed to metals in the environment. *Canadian Journal of Fisheries and Aquatic Sciences*, *62*, 982–990. https://doi.org/10.1139/f05-011

Green, N. D., Cargnelli, L., Briggs, T., Drouin, R., Child, M., Esbjerg, J., Valiante, M., Henderson, T., McGregor, D., & Munro, D. (2010). *Detroit River Canadian Remedial Action Plan: Stage 2 Report* (Publication No. 1). Detroit River Canadian Cleanup.

Griffiths, R. W. (1991). Environmental quality assessment of the St. Clair River as reflected by the distribution of benthic macroinvertebrates in 1985. *Hydrobiologia*, *219*, 143–164. https://doi.org/10.1007/BF00024752

Hariharan, I. K., Wake, D. B., & Wake, M. H. (2016). Indeterminate growth: Could it represent the ancestral condition. *Cold Spring Harbor Perspectives in Biology*, *8*, a019174. https://doi.org/10.1101/cshperspect.a019174

Hayden, B., Massa-Gallucci, A., Harrod, C., O'Grady, M., Caffrey, J., & Kelly-Quinn, M. (2014). Trophic flexibility by roach *Rutilus rutilus* in novel habitats facilitates rapid growth and invasion success. *Journal of Fish Biology*, *84*, 1099–1116. https://doi.org/10.1111/jfb.12351

Hayward, R. S., & Margraf, J. (1987). Eutrophication effects on prey size and food available to yellow perch in Lake Erie. *Transactions of the American Fisheries Society*, *116*, 210–223. https://doi.org/10.1577/1548-8659(1987)116<210:EEOPSA>2.0.CO;2

Heidtke, T., Hartig, J. H., Zarull, M. A., & Yu, B. (2006). PCB levels and trends within the Detroit River-western Lake Erie basin: A historical perspective of ecosystem monitoring. *Environmental Monitoring and Assessment*, *112*, 23–33. https://doi.org/10.1007/s10661-006-0212-8

Hellou, J., Ross, N. W., & Moon, T. W. (2012). Glutathione, glutathione *S*-transferase, and glutathione conjugates, complementary markers of oxidative stress in aquatic biota. *Environmental Science and Pollution Research*, *19*, 2007–2023. https://doi.org/10.1007/s11356-012-0909-x

Honda, M., & Suzuki, N. (2020). Toxicities of polycyclic aromatic hydrocarbons for aquatic animals. *International Journal of Environmental Research and Public Health*, *17*, 1363. https://doi.org/10.3390/ijerph17041363

Hong, X., & Zha, J. (2019). Fish behavior: A promising model for aquatic toxicology research. *Science of the Total Environment*, *686*, 311–321. https://doi.org/10.1016/j.scitotenv.2019.06.028

Hontela, A. (2005). Adrenal toxicology: Environmental pollutants and the HPI axis. In T. P. Mommsen & T. W. Moon, (Eds.), *Environmental toxicology biochemistry and molecular biology of fishes* (Vol. *6*, pp. 331–364). Elsevier. https://doi.org/10.1016/S1873-0140(05)80015-3

Hontela, A., Daniel, C., & Ricard, A. C. (1996). Effects of acute and subacute exposures to cadmium on the interrenal and thyroid function in rainbow trout, *Oncorhynchus mykiss*. *Aquatic Toxicology*, *35*, 171–182. https://doi.org/10.1016/0166-445X(96)00012-4

Hontela, A., Dumont, P., Duclos, D., & Fortin, R. (1995). Endocrine and metabolic dysfunction in yellow perch, *Perca flavescens*, exposed to organic contaminants and heavy metals in the St. Lawrence River. *Environmental Toxicology and Chemistry*, *14*, 725–731.

Hontela, A., Rasmussen, J. B., Audet, C., & Chevalier, G. (1992). Impaired cortisol stress response in fish from environments polluted by PAHs, PCBs and mercury. *Archives of Environmental Contamination and Toxicology*, *22*, 278–283. https://doi.org/10.1007/BF00212086

Houde, M., Giraudo, M., Douville, M., Bougas, B., Couture, P., De Silva, A. O., Spencer, C., Lair, S., Verreault, J., Bernatchez, L., & Gagnon, C. (2014). A multi-level biological approach to evaluate impacts of a major municipal effluent in wild St. Lawrence River yellow perch (*Perca flavescens*). *Science of the Total Environment*, *497–498*, 307–318. https://doi.org/10.1016/j.scitotenv.2014.07.059

Iles, A. C., & Rasmussen, J. B. (2005). Indirect effects of metal contamination on energetics of yellow perch (*Perca flavescens*) resulting from food web simplification. *Freshwater Biology*, *50*, 976–992. https://doi.org/10.1111/j.1365-2427.2005.01380.x

Incardona, J. P., & Scholz, N. L. (2017). Environmental pollution and the fish heart. *Fish Physiology*, *36*(Part B), 373–433. https://doi.org/10.1016/bs.fp.2017.09.006

 

International Joint Commission United States and Canada. (1987). *Revised Great Lakes Water Quality Agreement of 1978*.

Jaffé, R. (1991). Fate of hydrophobic organic pollutants in the aquatic environment: A review. *Environmental Pollution*, *69*, 235–257. https://doi.org/10.1016/0269-7491(91)90147-O

Jeon, H.-J., Park, Y.-C., Lee, W.-O., Lee, J.-H., & Kim, J.-H. (2011). cDNA cloning and expression of a cytochrome P450 1A (CYP1A) from the pale chub, *Zacco platypus*. *Korean. Journal of Limnology*, *44*, 364–372.

Jia, J., Thiessen, L., Schachtschneider, J., Waltho, J., & Marvin, C. (2010). Contaminant trends in suspended sediments in the Detroit River–Lake St. Clair–St. Clair River corridor, 2000 to 2004. *Water Quality Research Journal*, *45*, 69–80. https://doi.org/10.2166/wqrj.2010.008

Johnson, L. L., Anulacion, B. S., Arkoosh, M. R., Burrows, D. G., da Silva, D. A. M., Dietrich, J. P., Myers, M. S., Spromberg, J., & Ylitalo, G. M. (2013). Effects of legacy persistent organic pollutants (POPs) in fish—Current and future challenges. *Fish Physiology*, *33*, 53–140. https://doi.org/10.1016/B978-0-12-398254-4.00002-9

Jönsson, M. E., Gao, K., Olsson, J. A., Goldstone, J. V., & Brandt, I. (2010). Induction patterns of new CYP1 genes in environmentally exposed rainbow trout. *Aquatic Toxicology*, *98*, 311–321. https://doi.org/10.1016/j.aquatox.2010.03.003

Jürgens, M. D., Crosse, J., Hamilton, P. B., Johnson, A. C., & Jones, K. C. (2016). The long shadow of our chemical past—High DDT concentrations in fish near a former agrochemicals factory in England. *Chemosphere*, *162*, 333–344. https://doi.org/10.1016/j.chemosphere.2016.07.078

Keast, A. (1977). Diet overlaps and feeding relationships between the year classes in the yellow perch (*Perca flavescens*). *Environmental Biology of Fishes*, *2*, 53–70. https://doi.org/10.1007/BF00001416

King-Heiden, T. C., Mehta, V., Xiong, K. M., Lanham, K. A., Antkiewicz, D. S., Ganser, A., Heideman, W., & Peterson, R. E. (2012). Reproductive and developmental toxicity of dioxin in fish. *Molecular and Cellular Endocrinology*, *354*, 121–138. https://doi.org/10.1016/j.mce.2011.09.027

Kondoh, M. (2010). Linking learning adaptation to trophic interactions: A brain size-based approach. *Functional Ecology*, *24*, 35–43. https://doi.org/10.1111/j.1365-2435.2009.01631.x

Knight, R. L., Margraf, F. J., & Carline, R. F. (1984). Piscivory by walleyes and yellow perch in western Lake Erie. *Transactions of the American Fisheries Society*, *113*, 677–693. https://doi.org/10.1577/1548-8659(1984)113<677:PBWAYP>2.0.CO;2

Laberge, F., Yin-Liao, I., & Bernier, N. J. (2019). Temporal profiles of cortisol accumulation and clearance support scale cortisol content as an indicator of chronic stress in fish. *Conservation Physiology*, *7*, coz052. https://doi.org/10.1093/conphys/coz052

Layman, C. A., Arrington, D. A., Montaña, C. G., & Post, D. M. (2007). Can stable isotope ratios provide for community-wide measures of trophic structure. *Ecology*, *88*, 42–48. https://doi.org/10.1890/0012-9658(2007)88[42:CSIRPF]2.0.CO;2

Lijoy, M. K., Andreasen, E. A., & Tanguay, R. L. (2006). Aryl hydrocarbon receptor activation inhibits regenerative growth. *Molecular Pharmacology*, *69*, 257–265. https://doi.org/10.1124/mol.105.018044

Lin, F., Ni, L., & Kennedy, C. J. (2022). Diluted bitumen-induced alterations in aerobic capacity, swimming performance, and post-exercise recovery in juvenile sockeye salmon (*Oncorhynchus nerka*). *Aquatic Toxicology*, *247*:106150. https://doi.org/10.1016/j.aquatox.2022.106150

López-Pedrouso, M., Varela, Z., Franco, D., Fernández, J. A., & Aboal, J. R. (2020). Can proteomics contribute to biomonitoring of aquatic pollution? A critical review. *Environmental Pollution*, *267*, 115473. https://doi.org/10.1016/j.envpol.2020.115473

Louiz, I., Palluel, O., Ben-Attia, M., Aït-Aïssa, S., & Ben Hassine, O. K. (2018). Liver histopathology and biochemical biomarkers in *Gobius niger* and *Zosterisessor ophiocephalus* from polluted and non-polluted Tunisian lagoons (southern Mediterranean Sea). *Marine Pollution Bulletin*, *128*, 248–258. https://doi.org/10.1016/j.marpolbul.2018.01.028

Lushchak, V. I. (2011). Environmentally induced oxidative stress in aquatic animals. *Aquatic Toxicology*, *101*, 13–30. https://doi.org/10.1016/j.aquatox.2010.10.006

Maceina, M. J., & Sammons, S. M. (2018). The relation between polychlorinated biphenyls and population metrics of 4 species of fish from the upper Hudson River, New York, USA. *Environmental Toxicology and Chemistry*, *38*, 329–339. https://doi.org/10.1002/etc.4322

Maiti, D., Ansari, I., Rather, M. A., & Deepa, A. (2019). Comprehensive review on wastewater discharged from the coal-related industries—Characteristics and treatment strategies. *Water Science and Technology*, *79*, 2023–2035. https://doi.org/10.2166/wst.2019.195

Malmqvist, B., & Rundle, S. (2002). Threats to the running water ecosystems of the world. *Environmental Conservation*, *29*, 134–153. https://doi.org/10.1017/S0376892902000097

McPhedran, K. N., Grgicak-Mannion, A., Paterson, G., Briggs, T., Ciborowski, J. J. H., Haffner, G. D., & Drouillard, K. G. (2016). Assessment of hazard metrics for predicting field benthic invertebrate toxicity in the Detroit River, Ontario, Canada. *Integrated Environmental Assessment and Management*, *13*, 410–422. https://doi.org/10.1002/ieam.1785

McQuatters-Gollop, A., Mitchell, I., Vina-Herbon, C., Bedford, J., Addison, P. F. E., Lynam, C. P., Geetha, P. N., Vermeulan, E. A., Smit, K., Bayley, D. T. I., Morris-Webb, E., Niner, H. J., & Otto, S. A. (2019). From science to evidence—How biodiversity indicators can be used for effective marine conservation policy and management. *Frontiers in Marine Science*, *6*, 109. https://doi.org/10.3389/fmars.2019.00109

La Merrill, M. A., Sethi, S., Bernard, L., Moshier, E., Haraldsson, B., & Buettner, C. (2016). Perinatal DDT exposure induces hypertension and cardiac hypertrophy in adult mice. *Environmental Health Perspectives*, *124*, 1722–1727. https://doi.org/10.1289/EHP164

Mix, M. C. (1986). Cancerous diseases in aquatic animals and their association with environmental pollutants: A critical literature review. *Marine Environmental Research*, *20*, 1–141. https://doi.org/10.1016/0141-1136(86)90013-9

Moisiadis, V. G., & Matthews, S. G. (2014). Glucocorticoids and fetal programming part 2: Mechanisms. *Nature Reviews Endocrinology*, *10*, 403–411. https://doi.org/10.1038/nrendo.2014.74

Morrison, T. W., Lynch, W. E., & Dabrowski, K. (1997). Predation on zebra mussels by freshwater drum and yellow perch in western Lake Erie. *Journal of Great Lakes Research*, *23*, 177–189. https://doi.org/10.1016/S0380-1330(97)70895-5

Mumby, J. A., Jonhson, T. B., Stewart, T. J., Halfyard, E. A., Weidel, B. C., Walsh, M. G., Lantry, J. R., & Fisk, A. T. (2018). Feeding ecology and niche overlap of Lake Ontario offshore forage fish assessed with stable isotopes. *Canadian Journal of Fisheries and Aquatic Sciences*, *75*, 759–771. https://doi.org/10.1139/cjfas-2016-0150

Nawrocki, B., Colborne, S. F., Yurkowski, D. J., & Fisk, A. T. (2016). Foraging ecology of bowfin (*Amia calva*) in the Lake Huron–Erie corridor of the Laurentian Great Lakes: Individual specialists in generalist populations. *Journal of Great Lakes Research*, *42*, 1452–1460. https://doi.org/10.1016/j.jglr.2016.08.002

Nawrocki, B., McLeod, A. M., Hussey, N. E., Colborne, S. F., Del Papa, J., & Fisk, A. T. (2020). Assessing trophic position quantification methods for three piscivorous freshwater fish using stable isotopes and stomach contents. *Journal of Great Lakes Research*, *46*, 578–588. https://doi.org/10.1016/j.jglr.2020.03.017

Niven, J. E., & Laughlin, S. B. (2008). Energy limitation as a selective pressure on the evolution of sensory systems. *Journal of Experimental Biology*, *211*, 1792–1804. https://doi.org/10.1242/jeb.017574.

O'Callaghan, P., Kelly-Quinn, M., Jennings, E., Antunes, P., O'Sullivan, M., Fenton, O., & hUallacháin, D. Ó. (2019). The environmental impact of cattle access to watercourses: A review. *Journal of Environmental Quality*, *48*, 340–351. https://doi.org/10.2134/jeq.2018.04.0167

Olsson, J., Quevedo, M., Colson, C., & Svanbäck, R. (2007). Gut length plasticity in perch: Into the bowels of resources polymorphisms. *Biological Journal of the Linnean Society*, *90*, 517–523. https://doi.org/10.1111/j.1095-8312.2007.00742.x

Parker, M. O. (2016). Adult vertebrate behavioural aquatic toxicology: Reliability and validity. *Aquatic Toxicology*, *170*, 323–329. https://doi.org/10.1016/j.aquatox.2015.09.001

Parrish, D. L., & Margraf, F. J. (1994). Spatial and temporal patterns of food use by white perch and yellow perch in Lake Erie. *Journal of Freshwater Ecology*, *9*, 29–35. https://doi.org/10.1080/02705060.1994.9664424

Post, D. M. (2002). Using stable isotopes to estimate trophic position: Models, methods, and assumptions. *Ecology*, *83*, 703–718. https://doi.org/10.1890/0012-9658(2002)083[0703:USITET]2.0.CO;2

Premdas, F., & Anderson, J. (1963). The uptake and detoxification of $C^{14}$-labelled DDT in Atlantic salmon, *Salmo salar*. *Journal of the Fisheries Research Board of Canada*, *20*, 827–837. https://doi.org/10.1139/f63-053

Roesijadi, G. (1992). Metallothioneins in metal regulation and toxicity in aquatic animals. *Aquatic Toxicology*, *22*, 81–114. https://doi.org/10.1016/0166-445X(92)90026-J

 

Robillard, S. R., & Mardsen, J. E. (1996). Comparison of otolith and scale ages for yellow perch from Lake Michigan. *Journal of Great Lakes Research*, *22*, 429–435. https://doi.org/10.1016/S0380-1330(96)70967-X

Rohonczy, J., O'Dwyer, K., Rochette, A., Robinson, S. A., & Forbes, M. R. (2021). Meta-analysis shows environmental contaminants elevate cortisol levels in teleost fish—Effect sizes depend on contaminant class and duration of experimental exposure. *Science of the Total Environment*, *800*, 149402. https://doi.org/10.1016/j.scitotenv.2021.149402

Russell, R. W., Gobas, F. A. P. C., & Haffner, G. D. (1999). Role of chemical and ecological factors in trophic transfer of organic chemicals in aquatic food webs. *Environmental Toxicology and Chemistry*, *18*, 1250–1257. https://doi.org/10.1002/etc.5620180626

Saaristo, M., Brodin, T., Balshine, S., Bertram, M. G., Brooks, B. W., Ehlman, S. M., McCallum, E. S., Sih, A., Sundin, J., Wong, B. B. M., & Arnold, K. E. (2018). Direct and indirect effects of chemical contaminants on the behaviour, ecology and evolution of wildlife. *Proceedings B*, *285*, 20181297. https://doi.org/10.1098/rspb.2018.1297

Sandlund, O. T., Eloranta, A. P., Borgstrøm, R., Hesthagen, T., Johsen, S. I., Museth, J., & Rognerud, S. (2016). The trophic niche of Arctic charr in large southern Scandinavian lakes is determined by fish community and lake morphometry. *Hydrobiologia*, *783*, 117–130. https://doi.org/10.1007/s10750-016-2646-5

Schlenk, D., Celander, M., Gallagher, E., George, S., James, M., Kullman, S., van den Hurk, P., & Willett, K. (2008). Biotransformation in fishes. In R. T. Di Giulio & D. E. Hinton, (Eds.), *The Toxicology of fishes* (pp. 153–234). CRC Press.

Schwarzenbach, R. P., Escher, B. I., Fenner, K., Hofstetter, T. B., Johnson, C. A., von Gunten, U., & Wehrli, B. (2006). The challenge of micropollutants in aquatic systems. *Science*, *313*, 1072–1077. https://doi.org/10.1126/science.1127291

Sherwood, G. D., Kovecses, J., Hontela, A., & Rasmussen, J. B. (2002). Simplified food webs lead to energetic bottlenecks in polluted lakes. *Canadian Journal of Fisheries and Aquatic Sciences*, *59*, 1–5. https://doi.org/10.1139/f01-213

Singh, N. P., McCoy, M. T., Tice, R. R., & Schneider, E. L. (1988). A simple technique for quantitation of low levels of DNA damage in individual cells. *Experimental Cell Research*, *175*, 184–191. https://doi.org/10.1016/0014-4827(88)90265-0

Sorrentino, C., Roy, N. K., Courtenay, S. C., & Wirgin, I. (2005). Co-exposure to metals modulates CYP1A mRNA inducibility in Atlantic tomcod *Microgadus tomcod* from two populations. *Aquatic Toxicology*, *75*, 238–252. https://doi.org/10.1016/j.aquatox.2005.08.006

Stegeman, J. J., & Lech, J. J. (1991). Cytochrome p-450 monooxygenase systems in aquatic species: Carcinogen metabolism and biomarkers for carcinogen and pollutant exposure. *Environmental Health Perspectives*, *90*, 101–109. https://doi.org/10.1289/ehp.90-1519513

Sullivan, T. J., & Stepien, C. A. (2014). Genetic diversity and divergence of yellow perch spawning populations across the Huron–Erie corridor, from Lake Huron through western Lake Erie. *Journal of Great Lakes Research*, *40*(S2), 101–109. https://doi.org/10.1016/j.jglr.2012.12.004

Tenji, D., Micic, B., Sipos, S., Miljanovic, B., Teodorovic, I., & Kaisarevic, S. (2020). Fish biomarkers from a different perspective: Evidence of adaptive strategy of *Abramis brama* (L.) to chemical stress. *Environmental Sciences Europe*, *32*, 47. https://doi.org/10.1186/s12302-020-00316-7

Thomas, R. E., & Rice, S. D. (1987). Effect of water-soluble fraction of Cook Inlet crude oil on swimming performance and plasma cortisol in juvenile Coho salmon (*Oncorhynchus kisutch*). *Comparative Biochemistry and Physiology C*, *87*, 177–180. https://doi.org/10.1016/0742-8413(87)90200-3

Thornley, S. (1985). Macrozoobenthos of the Detroit and St. Clair Rivers with comparisons to neighbouring waters. *Journal of Great Lakes Research*, *11*, 290–296. https://doi.org/10.1016/S0380-1330(85)71771-6

Tierney, K. B., Baldwin, D. H., Hara, T. J., Ross, P. S., Scholz, N. L., & Kennedy, C. J. (2010). Olfactory toxicity in fishes. *Aquatic Toxicology*, *96*, 2–26. https://doi.org/10.1016/j.aquatox.2009.09.019

Trejo, J. L., Cuchillo, I., Machin, C., & Rua, C. (2000). Maternal adrenalectomy at the early onset of gestation impairs the postnatal development of the rat hippocampal formation: Effects on cell numbers and differentiation, connectivity and calbindin-D28K immunoreactivity. *Journal of Neuroscience Research*, *62*, 644–667. https://doi.org/10.1002/1097-4547(20001201)62:5<644::AID-JNR4>3.0.CO;2-3

Trippel, E. A. (1995). Age at maturity as a stress indicator in fisheries. *BioScience*, *45*, 759–771. https://doi.org/10.2307/1312628

Ton, C., Lin, Y., & Willett, C. (2006). Zebrafish as a model for developmental neurotoxicity testing. *Birth Defects Research, Part A*, *76*, 553–567. https://doi.org/10.1002/bdra.20281

Valavanidis, A., Vlahogianni, T., Dassenakis, M., & Scoullos, M. (2006). Molecular biomarkers of oxidative stress in aquatic organisms in relation to toxic environmental pollutants. *Ecotoxicology and Environmental Safety*, *64*, 178–189. https://doi.org/10.1016/j.ecoenv.2005.03.013

van der Oost, R., Beyer, J., & Vermeulen, N. P. E. (2003). Fish bioaccumulation and biomarkers in environmental risk assessment: A review. *Environmental Toxicology and Pharmacology*, *13*, 57–149. https://doi.org/10.1016/S1382-6689(02)00126-6

Vidal-Martínez, V. M., Pech, D., Sures, B., Purucker, S. T., & Poulin, R. (2010). Can parasites really reveal environmental impact. *Trends In Parasitology*, *26*, 44–51. https://doi.org/10.1016/j.pt.2009.11.001

Weber, R., Gaus, C., Tysklind, M., Johnston, P., Forter, M., Hollert, H., Heinisch, E., Holoubek, I., Lloyd-Smith, M., Masunaga, S., Moccarelli, P., Santillo, D., Seike, N., Symons, R., Torres, J. P. M., Verta, M., Varbelow, G., Vijgen, J., Watson, A., … Zennegg, M. (2008). Dioxin- and POP-contaminated sites—Contemporary and future relevance and challenges. *Environmental Science and Pollution Research*, *15*, 363–393. https://doi.org/10.1007/s11356-008-0024-1

Weis, J. S., Smith, G., Zhou, T., Santiago-Bass, C., & Weis, P. (2001). Effects of contaminants on behavior: Biochemical mechanisms and ecological consequences. *BioScience*, *51*, 209–217. https://doi.org/10.1641/0006-3568(2001)051[0209:EOCOBB]2.0.CO;2

Whyte, J. J., Jung, R. E., Schmitt, C. J., & Tillitt, D. E. (2000). Ethoxyresorufin-*O*-deethylase (EROD) activity in fish as a biomarker of chemical exposure. *Critical Reviews in Toxicology*, *30*, 347–570. https://doi.org/10.1080/10408440091159239

Williams, T. A., Bonham, L. A., & Bernier, N. J. (2017). High environmental ammonia exposure has developmental-stage specific and long-term consequences on the cortisol stress response in zebrafish. *General and Comparative Endocrinology*, *254*, 97–106. https://doi.org/10.1016/j.ygcen.2017.09.024

Wolf, J. C., & Wolfe, M. J. (2005). A brief overview of nonneoplastic hepatic toxicity in fish. *Toxicologic Pathology*, *33*, 75–85. https://doi.org/10.1080/01926230590890187

Wong, C. K., Yeung, H. Y., Woo, P. S., & Wong, M. H. (2001). Specific expression of cytochrome P4501A1 gene in gill, intestine and liver of tilapia exposed to coastal sediments. *Aquatic Toxicology*, *54*, 69–80. https://doi.org/10.1016/S0166-445X(00)00173-9

Yancheva, V., Velcheva, I., Stoyanova, S., & Georgieva, E. (2016). Histological biomarkers in fish as a tool in ecological risk assessment and monitoring programs: A review. *Applied Ecology and Environmental Research*, *14*, 47–75. https://doi.org/10.15666/aeer/1401_047075

Yeakel, J. D., Bhat, U., Elliott Smith, E. A., & Newsome, S. D. (2016). Exploring the isotopic niche: Isotopic variance, physiological incorporation, and the temporal dynamics of foraging. *Frontiers in Ecology and Evolution*, *4*, 1. https://doi.org/10.3389/fevo.2016.00001

Zare, A., Henry, D., Chua, G., Gordon, P., & Habibi, H. R. (2018). Differential hepatic gene expression profile of male fathead minnows exposed to daily varying dose of environmental contaminants individually and in mixture. *Frontiers in Endocrinology*, *9*, 749. https://doi.org/10.3389/fendo.2018.00749

 