parameters were altered in rainbow trout exposed to creosote PAHs at concentrations as low as 0.6 ng·mL^{-1}. Bravo et al. (2011) noted a number of biochemical alterations in juvenile rainbow trout fed relatively low concentrations of PAHs in their diet. This study also demonstrated a reduction in survival for fish from low dose (0.66 μg·g fish^{-1}·day^{-1}) and high dose (7.8 μg·g fish^{-1}·day^{-1}) PAH treatments exposed to the pathogen *Aeromonas salmonicida*. As noted by these authors, the dietary concentrations were modeled after those observed in field-collected fish from contaminated Puget Sound estuaries. Another study on flame retardants found that dietary concentrations of PBDEs in field-collected fish compromised the immune system in juvenile Chinook, resulting in increased mortality when salmon were exposed to a common marine pathogen (Arkoosh et al. 2010).

Behavioral effects

Altered behavior in juvenile salmon can result in mortality. Many chemicals are known to affect fish behavior, including copper, cadmium, mercury, several organochlorine and current-use pesticides, organotins, and pentachlorophenol (Scott and Sloman 2004), and low levels can result in high rates of mortality (McIntyre et al. 2012). A number of mechanistic and behavioral studies support the observation of altered behavior in salmon exposed to low concentrations of copper and pesticides (Scholz et al. 2000; Sandahl et al. 2004; Laetz et al. 2009).

Field observations

There are very few field studies that have examined impacts on salmonids exposed to contaminants. One relevant study found a negative correlation between catch data for Atlantic salmon and the spray application of a pesticide to various tributaries within a river basin during smolt development for a 1-year period (Fairchild et al. 1999). Similar effects were also noted for spray events over several years and for another species (blueback herring (*Alosa aestivalis*), which is also anadromous. The authors concluded that 4-nonylphenol, a component of the spray mixture, likely affected smoltification, leading to excess mortality for this life stage. Another study confirmed the ability of the endocrine disruptors 4-nonylphenol and estradiol (E2) to impair physiological processes related to smoltification and demonstrated delayed downstream migration and increased mortality for exposed fish (Madsen et al. 2004). These results have important implications because endocrine disruptors are commonly found in contaminated estuaries.

Some of the strongest evidence supporting adverse effects in outmigrating juvenile salmon from contaminated estuaries comes from Puget Sound area field studies when viewed in light of the abovementioned laboratory studies of individual compounds or classes of compounds. One study calculated growth rates for juvenile Chinook from the Soos Creek hatchery that were captured in nearshore areas of Puget Sound within 2–4 weeks of release (Brennan et al. 2004). Of the juvenile Chinook assessed from all hatcheries (22 hatcheries, n = 86 fish captured in 2001 and n = 107 captured in 2002), fish from the Soos Creek hatchery accounted for most of the fish that exhibited negative growth (95%, 18 of 19 fish) or exhibited zero growth (100%, 4 of 4 fish) when compared with their respective release masses. Similar results were observed in 2002, with 91% of all negative growth fish (by length) coming from the Soos Creek hatchery (21 of 23 fish) (70% of all fish based on mass). Even though juvenile Chinook within the Duwamish estuary exhibit acceptable growth rates (Table 1), once they have accumulated contaminants and exit to open water they appear to be growth inhibited.

To test the observed patterns described here and the hypothesis that first-year factors are important for survival, this model was applied to the data presented in Duffy and Beauchamp (2011). In that study, juvenile Chinook were collected during their first year in open water with a mid-water tow net in three regions of Puget Sound over 4 years (1997, 1999, 2001, and 2002) during July and September. I divided the data in their table 1 by hatcheries that released into contaminated and uncontaminated estuaries (all except Hupp Springs, which release stream-type Chinook). Only hatcheries represented by more than three fish (25 of 28 possible data points) were selected, and ANOVA was performed on the groups. Survival was 2.5 times higher for fish transiting uncontaminated estuaries compared with the survival of fish migrating through contaminated estuaries (Table 6), which was consistent with the overall results of the present study. Mean release masses were higher for fish from the uncontaminated estuaries, but the differences were modest, especially when compared with the observed mass in July for these tag code groups (Table 6). Also, the specific growth rate was significantly lower in fish from contaminated estuaries. A focused examination of only the 2002 data from Duffy and Beauchamp (2011) shows the same large differences between fish from uncontaminated versus contaminated estuaries for SAR values (2.1-fold), fish mass in July (1.4-fold), and specific growth rate (1.5-fold), but with equal release masses (0.99-fold), supporting the contention that release mass was not an important factor. The results of this analysis (Table 6) are consistent with the conclusion in Duffy and Beauchamp (2011) that survival is strongly linked to fish mass in July and the conclusion of the present study that contaminated estuaries strongly affect juvenile survival. These data also support the hypothesis of this review that fish outmigrating through contaminated estuaries are likely affected by contaminants for the first phase of their marine residency. Also, if fish had experienced reduced growth in the estuaries because of prey limitations, compensatory growth (Johansen et al. 2001) would have likely allowed them to increase quickly once they encountered more favorable conditions as experienced by juveniles from other estuaries.

Table 6. Analysis of Duffy and Beauchamp (2011) data for juvenile Chinook captured in offshore waters of Puget Sound.

	SAR (%)	Release mass (g)	July mass (g)	SGR
Contaminated				
Mean	0.34	6.7	12.7	0.013
SE	0.09	0.5	1.3	0.001
n	11	11	11	11
Uncontaminated				
Mean	0.86	7.6	22.6	0.017
SE	0.12	0.5	1.9	0.001
n	14	14	14	14
p value	0.007	0.18	0.0001	0.04

Note: Data grouped by contaminated or uncontaminated rearing estuaries for juvenile Chinook. Mean and standard error (SE) are shown for smolt-to-adult return rate (SAR, %), release wet mass at hatchery, and fish wet mass in July. The specific daily growth rate (SGR) was also calculated. n is the number of hatchery–year combinations for each group over the release years 1997–2002. ANOVA p values shown for each variable. See text for details.

Laboratory studies with field-collected fish

Another strong line of evidence can be found in laboratory studies with fish from specific estuaries. One study collected juvenile Chinook from three hatcheries (Soos Creek, Kalama Creek, and Puyallup) and their respective estuary (Duwamish, Nisqually, and Puyallup). Fish were held in the lab for 40 days (1990) or 84 days (1991) (Varanasi et al. 1993). The percent survival of fish from the Duwamish River (56%) and Puyallup River (58%) estuaries was significantly less than for fish from their respective hatchery (86% and 88%). Survival for fish from the Kalama hatchery (88%) was not different from that for fish collected in the Nisqually River estuary (81%). This experiment was repeated in 1991 for the