Myriad contaminants can be found in these estuaries. There are legacy compounds such as PCBs, DDTs, and endrin, several metals (As, Cu, Cd, Hg, Pb, and Zn), PAHs, pesticides (organotins, organophosphates, carbamates, triazines, pyrethroids, and chlorophenols), a large number of industrial chemicals (e.g., phthalates, bisphenol A, flame retardants, and perfluorocarbons), pharmaceuticals and personal care products from wastewater treatment plants and septic systems, and dozens of new and emerging contaminants. We have exposure and toxicity information for a few of these chemicals; however data are lacking for most, especially with regard to fish health. Additionally, there are almost no data on the toxicity of mixtures for these contaminants. In the majority of cases, toxicants act by additive effects (see Meador 2006) and therefore should be considered the default condition. Synergism has been reported for some mixtures of pesticides (Laetz et al. 2009), which should be considered along with potential antagonist interactions. It is likely that a complex mixture of contaminants found in urban estuaries is responsible for the reduced survival experienced by juvenile Chinook that rear in these estuaries.

Over the past several decades, a number of contaminants in urban and industrial areas have increased, or remain elevated, in terms of frequency of occurrence and magnitude including polybrominated diphenyl ethers (PBDEs), bisphenol A, phthalates, organotins, PAHs, pesticides, metals, and pharmaceuticals and personal care products (Puget Sound Ambient Monitoring Program 2007; Daughton and Brooks 2011). All these contaminants are capable of causing adverse metabolic, endocrine, immune, and behavioral responses that may jeopardize the chances of juvenile fish surviving to reproductive maturity. It is worth noting that some contaminants, such as PCBs, were higher in the 1970s–1980s in Puget Sound (O'Neill and West 2009), which corresponds to many of the years exhibiting large differences in SAR values for Chinook between contaminated and uncontaminated estuaries (Table S1[1]).

Most bioaccumulative contaminants increase quickly in fish tissues. This is especially true for salmonids, because they exhibit very high rates of feeding and gill ventilation. It is not uncommon for juvenile Chinook to consume 20% of their body mass per day and to ventilate 0.5 $L \cdot kg^{-1} \cdot min^{-1}$, allowing rapid increases in toxicant tissue concentrations. Also, because the rates of uptake and elimination follow first-order kinetics, a high percentage of a steady-state concentration can occur rapidly. Even for compounds such as PCBs that may exhibit steady-state concentrations after 28 days, fish can achieve 50% of that level within 6.5 days. Rapid uptake and high rates of consumption and ventilation allow juvenile salmon to accumulate high concentrations even if their residence time in a contaminated estuary is limited. One study (Meador et al. 2010) that analyzed 111 whole-body juvenile Chinook samples (many as composites of several individuals) found that most (53%) contained high concentrations of total PCBs (>50 $ng \cdot g^{-1}$), indicating that these fish had been accumulating contaminants for an extended period of time. When considered in terms of increased growth and total amount accumulated in the estuary, the median increase for all 111 samples was 11 times over that for fish from the hatchery. Because of the low lipid content for outmigrating juvenile Chinook, almost all fish with total PCBs >50 $ng \cdot g^{-1}$ exceeded the predicted threshold concentration for toxicity (Meador et al. 2002). It is important to note that toxic effects can occur in fish without detectable increases in tissue concentrations. This has been reported for PAHs (Meador et al. 2006) and is also expected for other highly metabolized organic compounds and some metals, indicating that throughput (e.g., $\mu g \cdot g\ fish^{-1} \cdot day^{-1}$) or metabolite determination (Meador et al. 2006, 2008*a*; McElroy et al. 2011) are more appropriate metrics for exposure and toxicity for these chemicals.

Growth effects

Most of the mortality for juvenile salmon during their first year in open water is due to predation, which has been shown to be a function of size (Beamish and Mahnken 2001; Brodeur et al. 2003; Duffy and Beauchamp 2011). A dietary toxicity study of a mixture of PAHs fed to juvenile Chinook salmon found altered metabolic parameters as low as 2 $\mu g \cdot g\ fish^{-1} \cdot day^{-1}$ and severe reductions in growth and lipid content for higher doses (≈20 $\mu g \cdot g\ fish^{-1} \cdot day^{-1}$) (Meador et al. 2006). These doses are consistent with expected rates of uptake and observed PAH concentrations for stomach contents found in outmigrating Chinook in the Duwamish and Puyallup river estuaries. A large number of frequently occurring contaminants can affect organismal growth, such as tributyltin (Meador et al. 2011), copper (Marr et al. 1996), dioxin (Eisler 1986), and numerous other metabolic disruptors (phthalates, bisphenol A, perfluorooctane, and pesticides).

Physiological changes

Lipid content is also an important factor determining the probability that juvenile salmon will survive their first winter in open water (Gardiner and Geddes 1980; MacFarlane and Norton 2002; Biro et al. 2004), which is related to growth potential and having energy reserves when prey availability is reduced. One study demonstrated that juvenile Chinook experience a growth spurt once they leave the estuary, which is fueled in part by their lipid reserves (MacFarlane and Norton 2002). This rapid increase in size is advantageous for avoiding predation. Normal lipid content for juvenile Chinook can average 2%–3% (wet mass), depending on the analytical method, as they exit the estuary to open water (MacFarlane and Norton 2002; Johnson et al. 2007; Sloan et al. 2010), which can contribute to the increase in growth and serve as a reserve for the winter when prey are less abundant. Related to this, one study found that juvenile ocean-type Chinook salmon with a higher lipid content (7.9% wet mass) exhibited a SAR that was twice as high as those with a lower lipid level (4.1%) at the time of release from the hatchery (Burrows 1969).

A critical lipid content of 1% was determined in lab and field studies for rainbow trout (*Oncorhynchus mykiss*), with high mortality resulting when lipid content fell below this level (Biro et al. 2004). The concept of a critical lipid content for winter survival was also established by Finstad et al. (2004), who showed that Atlantic salmon (*Salmo salar*) needed 4400–4800 $J \cdot g^{-1}$ for winter survival, which translates to a value of approximately 0.46% wet mass for triacylglycerols (TAGs). Numerous toxicants are metabolic disruptors affecting metabolic processes, growth, and lipid homeostasis. Meador et al. (2006) reported a substantial reduction in whole-body lipid content in juvenile Chinook from 2.5% wet mass for control fish to 1.0% for high-dose fish (TAGs below 0.4% wet mass) exposed to environmentally realistic concentrations of dietary PAHs. Alterations to related physiological parameters (plasma TAGs, lipase, and albumin) were also observed at low exposure doses. Another critical aspect is the lipid-normalized tissue concentration for hydrophobic compounds. As lipid levels decline in fish, a given concentration of poorly metabolized contaminants, such as PCBs, will increase in bioavailability within the animal and result in increased toxicity (Lassiter and Hallam 1990). This is especially relevant for juvenile fish during their first winter, as lipid content declines and effective toxic concentrations rise internally.

Immunotoxicants

A number of studies demonstrate that common urban contaminants such as PAHs and PCBs are immunotoxicants in juvenile salmon at environmentally low concentrations (Arkoosh et al. 1991, 1998, 2010; Bravo et al. 2011). When the immune system is compromised by these chemicals, juvenile salmon are more susceptible to fatal infections from common pathogens found in the environment. Karrow et al. (1999) found that a number of immune