Salmon & Trout Conservation
Chemical Impacts: The link between freshwater xenobiotics and reduced juvenile salmonid marine survival

Literature Review

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I. Introduction

Many of the factors regulating salmonid populations remain poorly understood, although recent evidence suggests that environmental conditions experienced by juveniles in freshwater may have a significant effect on survival once the fish migrates into the marine environment (Fig. 1). Juvenile anadromous fish undergo a transition from parr to smolt during their downstream migrations. This transition, known as ‘smoltification’, is orchestrated by hormones and involves a plethora of physiological and behavioural changes that prepare juvenile salmonids for entry into seawater (Russell et al. 2012). The term xenobiotic denotes a substance, typically a synthetic chemical, which is foreign to an ecological system. The exposure of juvenile salmon to sublethal concentrations of xenobiotic compounds in freshwater can have adverse biological consequences on smolt quality and readiness for life at sea (Moore et al. 2003). The hypothesis is that sublethally stressed smolts will have reduced smolt-to-adult survival in the marine environment as a result of inhibited enzyme activities (important for maintaining physiological homeostasis in seawater), reduced growth, effects on migratory behaviour and impacts on predator avoidance (Finstad & Jonsson, 2001). This review explores the main categories of freshwater xenobiotics, examining their biological influences in juvenile salmonids and in turn how sublethal exposure can decrease marine survival probability.

![Diagram of freshwater perturbations and seawater performance](image)

**Figure 1:** A summary of the outcomes of freshwater environmental impacts on salmonid seawater performance (McCormick et al. 2009)

II. Pesticides

**Triazines**

Atrazine is a common agricultural herbicide often applied in spring to control undesirable plants. It has high mobility through soil, so watercourses are particularly susceptible to contamination (Waring & Moore, 2004). Due to persistent groundwater contamination it was banned in the EU in 2004 (Sass & Colangelo, 2006).
However, its safety remains controversial with atrazine being amongst the most heavily used pesticides in the United States (Jablonowski et al. 2011).

Waring & Moore (1996) found Atlantic salmon smolt mortality of ~30% when smolts were exposed to sub-lethal concentrations of atrazine before transfer to seawater. This lead to speculation that atrazine inhibits the ability of juvenile salmon to adapt to the marine environment. A study exploring this further found that salmon smolts exposed to atrazine had significantly lower gill Na+/K+ ATPase activity than a control group with no exposure (Moore & Fewings, 2003). Gill Na+/K+ ATPase activity increase is one of the major physiological processes that allows salmon to adapt to saltwater environments. Activity of the enzyme increases two to five-fold during smoltification and signifies an increase in the ability of the fish to osmoregulate in saltwater. These results were corroborated by Waring & Moore (2004), where exposure of 2, 5 and 10 µg/L atrazine caused a significant reduction in Na+/K+ ATPase activity in Atlantic salmon smolts in freshwater. Moore et al. (2008) also found that exposure of Atlantic salmon smolts to a concentration of atrazine as little as 0.1 µg/L over a 72-h period significantly reduced gill Na+/K+ ATPase activity. Plus on transfer to 33% saltwater, 100% smolt mortality occurred. Contrastingly, Matsumoto et al. (2010) did not detect a significant effect on gill Na+/K+ ATPase activity in salmon smolts after short term exposure of up to 100 µg atrazine/L. As the concentrations of atrazine tested exceeded those likely to be experienced in the natural aquatic environment, it was concluded that atrazine poses no risk to Atlantic salmon during smoltification. However, it is important to take into account that in the wild fish are likely to be exposed to atrazine for long term periods. Atrazine is relatively persistent in freshwater with a half-life up to 350 days depending on environmental conditions (Nieves-Puigdoller et al. 2007). Interestingly, Strand et al. (2011) showed that early descending smolts with low gill Na+/K+ ATPase activity may delay their final sea entry and spend some time in the lower part of the river, thereby synchronising their final sea entry with later arriving smolts. This behaviour could give smolts the opportunity to recover from contaminant exposure, although it would be dependent on contaminant concentration and exposure time.

Slight decreases in growth and elevated plasma cortisol in response to atrazine exposure have also been observed. Atrazine at concentrations at and above 6.5 µg/L caused elevated plasma cortisol (32–37ng/ml) concentrations in salmon smolts compared to controls (18ng/ml) (Waring & Moore, 2004). Elevated plasma cortisol indicates that smolts are experiencing persistent physiological stress and prolonged elevation is considered harmful due to increased susceptibility to disease and suppression of reproductive processes (Carey & McCormick, 1998). Nieves-Puigdoller et al. (2007) found that prior exposure to atrazine in freshwater caused reduced growth of Atlantic salmon smolts reared in contaminant free seawater for 1 month. As early growth in seawater is widely recognised as critical to the overall marine survival of salmon, this impact of contaminant exposure in freshwater on seawater growth indicates a detrimental effect on marine survival (Beamish et al. 2004).

As well as physiological effects, atrazine exposure has also been shown to impact migratory behaviour of Atlantic salmon smolts. Moore et al. (2007) showed a significant effect on smolt active movement following atrazine exposure in freshwater. Control fish showed a higher degree of active movement compared to groups exposed to atrazine. The effect was also dose-responsive, with the higher atrazine dose group (5µg/L) showing significantly less migratory behaviour compared to the lower dose group (0.5µg/L). This has
particular significance to the survival of smolts in the marine environment as previous telemetry studies have indicated that smolts enter the open sea during a very brief window, so inhibition or delaying of migration means smolts may miss this (Moore et al. 1995).

**Organophosphates and Carbamates**

Organophosphate and carbamate insecticides are the most widely used group of insecticides in the world. Due to their acute toxicity, concern over their use in agriculture (particularly sheep-dips) and around the home in the UK has occurred. Although these compounds offer the advantage of rapid degradation in the environment, they generally lack target specificity and have high acute toxicity towards non-target species (Fulton & Key, 2001).

The primary function of organophosphates and carbamates is to inhibit acetylcholinesterase (AChE) activity, affecting normal cholinergic nerve transmission. The inhibition of AChE causes accumulation of acetylcholine in synapses and neuromuscular junctions, resulting in overstimulation of cholinergic receptors (Fukuto 1990). The key difference is that organophosphates bind the enzyme irreversibly while carbamates bind AChE reversibly. Therefore, recovery from exposure to organophosphates requires the synthesis of new cholinesterase enzyme, a process that can last up to several weeks whereas recovery from exposure to carbamates is brief and can occur on a timescale of a few hours (Baldwin et al. 2009). Monitoring of acetylcholinesterase (AChE) inhibition has been widely used in terrestrial and freshwater aquatic systems as an indicator of exposure and effects (Fulton & Key, 2001).

Research by Sandahl et al. (2005) found a close relationship between brain AChE inhibition and behavioural impairment in juvenile coho salmon exposed to the organophosphate chlorpyrifos. Brain AChE inhibition and reductions in feeding activity and spontaneous swimming were significantly correlated. Similarly, chlorpyrifos has been shown to significantly inhibit AChE in the nervous system and muscle of juvenile steelhead trout (Sandahl & Jenkins, 2002). The carbamate carbaryl has also been shown to cause decreased brain and muscle AChE activity in juvenile cutthroat trout after exposure to environmentally relevant concentrations (750 mg/L) for 6 hours (Labenia et al. 2007). Reduced swimming and feeding means reduction in somatic growth of juvenile salmon and as previously mentioned, juvenile growth is a critical determinant of freshwater and marine survival. Additionally, as the exposure in these studies typically replicated environmentally realistic concentrations, the effects were particularly concerning.

Synergistic AChE inhibition has also been shown in juvenile coho salmon when exposure to a mixture of organophosphates occurs (Laetz et al. 2013). Brain enzyme inhibition was concentration-dependent, with a 90% reduction and a significant decrease in spontaneous swimming speed at the highest binary mixture concentrations evaluated (diazinon-malathion at 2.6 and 1.1 µg/L, respectively; ethoprop-malathion at 2.8 and 1.2 µg/L, respectively). The authors highlighted an unusually steep concentration–response relationship across a mere 2-fold increase in mixture concentration. This emphasises the importance of evaluating pesticides as mixtures rather than on individual scales.
Pyrethroids

The increased use of pyrethroid pesticides has been largely driven by their high toxicity to target organisms and their relatively low toxicity to birds, mammals and amphibians (Köprücü et al. 2006). Because of their low mammalian toxicity, synthetic pyrethroid insecticides are extensively used in place of organochlorine, organophosphates and carbamates to control pests (Aydin et al. 2005). However, application of these compounds is highly toxic to freshwater fish even at very low concentrations (Suvetha et al. 2010). Pyrethroids interact with voltage-gated sodium channels in a similar manner to organochlorine pesticides, leading to repetitive neuronal firing (Clifford et al. 2005).

Pyrethroids mostly have a lethal effect on salmonids in freshwater so research on sublethal effects and their influence on juvenile marine survival is fairly scarce. Work by Sandahl et al. (2004) did indicate sublethal excitotoxicity (where nerve cells are damaged or killed by excessive stimulation by neurotransmitters) to juvenile coho salmon central networks after exposure to 0.2 µg/L of the pyrethroid esfenvalerate. Although a loss in sensory function in response to natural odorants did not occur, the natural odorant L-Serine triggered bursts of postsynaptic activity in the olfactory bulb. Interference with olfactory function has the potential to inhibit olfactory-mediated behaviours crucial for the survival and migration of salmonids. Goulding et al. (2013) also found swimming performance affected in juvenile rainbow trout after exposure to deltamethrin at 200 and 300 ng/L. The fish had significantly lower $U_{crit}$ (the highest swimming speed that a fish can maintain for a period equal in magnitude to the time interval used in the test) values than the control fish. Neither $U_{crit}$ nor $U_{max}$ (maximum swimming speed) reduced after exposure to permethrin. Pyrethroids are divided into two classes based largely on their chemical structure and potency, with type II being the most potent. The fact deltamethrin is a type II pyrethroid may explain why it caused an effect but permethrin (type I) did not. Smolts with lower swimming speeds have reduced ability to avoid predators and more likely to become prey (Thorstad et al. 2007). Alternatively, they may miss the previously mentioned optimum window for entering the open sea. Both eventualities decrease their chances of marine survival.

### III. Hormones

**Xenoestrogens**

Effluent discharges from wastewater treatment works can be major sources of oestrogenic compounds in freshwater environments (Bangsgaard et al. 2006). Xenoestrogens are synthetic and natural compounds that imitate oestrogen. The most potent form is the pharmaceutical oestrogen ethinylestradiol, the active ingredient found in the contraceptive pill. This oestrogen is eliminated slowly from the body and degrades relatively slowly in the environment (Robinson et al. 2008). Alkylphenols varying in their side-chain lengths [ethyl-, propyl-, octyl-, and nonylphenol (EP, PP, OP, and NP, respectively)] and bisphenol A (BPA) represent a large group of structurally related xenoestrogens that have endocrine-disruptive effects. These compounds are widely used in detergents, paints, herbicides, pesticides, and plastic polymers and have the ability to accumulate in the environment (Kochukov et al. 2009).

Mechanistically, scientific evidence strongly suggests that oestrogen effects on somatic growth and osmoregulatory ability are mediated through the growth hormone (GH)/insulin-like growth factor (IGF-I)
system (Arsenault et al. 2004). Interference of environmental oestrogens in the GH/IGF-I regulation of smoltification was suspected to be a causal factor in the decline of salmon in Eastern Canada in the 1970s and 1980s. Mass forest application of the formulated pesticide Matacil 1.8D (which contains the xenoestrogen 4-nonylphenol (4NP)) coinciding with the period of smoltification of salmon was the suspected cause of the decline (Segner et al. 2013). Fairchild et al. (1999) tested this hypothesis and found significant negative relationships between pesticide spraying intensity and salmon returns. Studies by Madsen et al. (2004; 1997) found that injecting the natural oestrogen 17β-estradiol (E2) and 4NP into Atlantic salmon smolts both caused a significant decline in salinity tolerance, gill Na+/K+ ATPase activity and the number of gill chloride cells. These findings were corroborated by McCormick et al. (2005) who also found lowered salinity tolerance in injected smolts. Additionally, a decrease in plasma IGF-I levels was found, with the authors suggesting that substantial declines in smolt salinity tolerance after exposure to 4NP and E2 are caused by the suppressing effect these compounds have on plasma IGF-I levels.

Fairchild et al. (2000) looked at how water-borne concentrations of E2 and 4NP effect smoltification in Atlantic salmon. A portion of the exposed smolts experienced compromised growth and did not survive the transition from freshwater to seawater. Growth is an important factor when considering smolt survival at sea; poor growth measured close to the parr-smolt transformation has been linked to reduced survival and fewer returns of adult salmon to their native streams (Beckman et al. 1999). Bangsgaard et al. (2006) also investigated the effects of waterborne exposure to E2, but additionally looked at how the xenoestrogen 4-tert-octylphenol (OP) influenced Atlantic salmon smoltification and swimming behaviour. Two experiments were conducted, one with early exposure in November at the parr-stage and another with exposure during the spring (March) at the pre-smolt stage. Exposure to OP at 100µg/L and E2 at 500ng/L compromised smolting through reduced gill Na+/K+ ATPase activity and impairment in the ability of smolts to regulate muscle water content and plasma osmolality when subjected to 24h seawater challenge tests during the smolting period. The treated fish also migrated at a lower frequency than the control fish, indicating a reduction in migratory drive. As physiological and behavioural inhibitory effects occurred in pre-smolts and smolts, this study showed that xenoestrogen exposure has the capability to reduce marine survival even when parr are exposed significantly prior to the smolting period.

IV. Persistent Organic Pollutants (typically halogenated organic compounds)

Persistent Organic Pollutants (POPs) are highly toxic man-made organic compounds that enter aquatic systems largely from industrial and domestic origins. They exhibit high lipid solubility so are capable of bioaccumulation in fatty tissues of various organisms, increasing in concentration as they are processed and metabolised. Their tendency to degrade very slowly means they can travel long distances around the globe and persist in the environment for long periods of time (Hurwich & Chary, 2000). Well-documented examples of POPs include polychlorinated biphenyls (PCBs) and brominated flame-retardants (BFRs).

*Polychlorinated biphenyls (PCBs)*

Lerner et al. (2007) found that juvenile Atlantic salmon exposed to both 1 µg/L and 10 µg/L of a PCB mixture during smolting exhibited a dose-dependent reduction in preference for seawater. Fish treated with the
higher dose also exhibited a 50% decrease in gill Na+/K+ ATPase activity and a 10% decrease in plasma chloride levels in freshwater. Disruption of seawater preference by the PCB mixture appeared to be related to negative impacts on the endocrine parameters involved in smolt development, particularly the thyroid hormone triiodothyronine (T$_3$) and cortisol. Thyroid hormones have been proposed to play an integral part in controlling downstream migratory behaviour and seawater preference in juvenile salmonids (Iwata 1995). Olfactory imprinting is also facilitated by increases in levels of thyroid hormones during smoltification; disruption to this process by contaminants means returning adult salmon will struggle to find their native streams and could fail to complete their life cycle (Lema & Nevitt 2004). It was concluded from the study that freshwater exposure to PCBs has the potential to inhibit preparatory adaptations that occur during smoltification, thereby reducing marine survival and sustainability of salmon populations. PCBs were also shown to impair smoltification and seawater performance in the anadromous Arctic char (Salvelinus alpinus) by Jørgensen et al. (2004). Juvenile char exposed to 100 mg/Kg of PCB had either a transient or a permanent reduction in plasma GH and IGF-I, alongside reduced titres of thyroxin and T$_3$ during the period of smoltification. These hormonal alterations corresponded with impaired hyposmoregulatory ability, plus reduced growth rate and survival after transference to seawater. Consequently, fewer juveniles matured compared to the control and lower concentration PCB treatment, indicating that if PCB levels are high enough impacts may occur at the level of the population by reducing the number of spawning adults.

**Brominated flame-retardants**

Brominated flame-retardants have similar properties to many classic organochlorine compounds and are structurally similar to PCBs. Within the general BFR category, there are three main divisions; tetrabromobisphenol-A (TBBPA), polybrominated diphenyl ethers (PBDEs), and hexabromocyclododecane (HBCD) (Palace et al. 2008). HBCD has a half-life of 2–25 days in water and studies around UK waters have shown evidence of bioaccumulation and biomagnification in the aquatic food chain (Law et al. 2006; Morris et al. 2004). Lower & Moore (2007) demonstrated that exposure to HBCD did not affect seawater adaptability in juvenile Atlantic salmon. However, disruption of plasma thyroid hormone levels and reduced olfactory function did occur. Over a 30-day period of HBCD exposure, olfactory response decreased significantly compared to the control smolts every week. A recently proposed alternative to PBDE’s and HBCDs was the compound tetrabromoethylcyclohexane (TBECH), yet the toxicity and environmental impacts of this compound remain questionable. Work by Park et al. (2011) showed disruption of the thyroid axis in juvenile brown trout after TBECH exposure and highlighted the implications considering the importance of the thyroid axis in mediating the physiological adaptations required prior to migration from freshwater to saltwater.

**V. Metals**

**Copper**

Copper is a widespread source of water pollution in salmon habitat. The highest levels of copper pollution tend to be found in river systems that have been impacted by mining activities. However, copper also enters watercourses through a variety of nonpoint anthropogenic sources. Copper compounds are widely used in bio-fouling treatments on aquatic vessels and fungicides on agricultural crops where leaching can occur (Davis et al. 2001).
Salmon rely on chemical cues to form olfactory memories and return to their natal river system to spawn. Critically, the migratory patterns of adult salmon are disrupted when olfactory function is lost (Baldwin et al. 2003). Copper is broadly toxic to the salmon olfactory nervous system and short-term influxes of copper to surface waters have been shown to interfere with olfactory-mediated migratory behaviours (McIntyre et al. 2008). Baldwin et al. (2003) exposed juvenile coho salmon to 1–20 µg/L of copper and a variety of natural odorants. After only 10 minutes of exposure the neurophysiological response to all odorants was impaired. Dissolved copper not only impairs sensory neurons in the salmonid nose, but also impairs the ability of juveniles to detect and respond to predation cues. A juvenile salmonid with disrupted predator avoidance behaviours stands a much greater risk of being eaten and therefore the likelihood of surviving to reproduce is reduced (Hecht et al. 2007). Sandahl et al. (2007) found that copper exposure up to 20 µg/L for 3 hours significantly impaired the sensory physiology and predator avoidance behaviours of juvenile coho salmon. They stated that as dissolved copper content in surface waters increases, the responsiveness of the peripheral olfactory system to predation cues will diminish until it falls below the threshold required to initiate an appropriate behavioural response. This means a likely outcome is that copper exposed fish will make behavioural decisions that are inappropriately risky for a particular ecological situation. McIntyre et al. (2012) also observed predator avoidance behaviour impairment in juvenile coho salmon exposed to 5–20 µg/L of dissolved copper for 3 hours. Unexposed fish exhibited a sharp reduction in swimming activity in response to two predation cues - conspecific skin extract and the upstream presence of a cutthroat trout predator previously fed juvenile coho – whereas no alarm response occurred in treated fish. The copper treatment also negatively influenced predator evasion once an attack was initiated. Therefore, it became increasingly likely that the juvenile coho would be captured on the first attack after exposure to higher copper exposure concentrations. It was hypothesised that the toxicity of copper to the lateral line mechanosensory system of the juvenile coho salmon may have contributed to the observed reduction in evasion success. Similarly to olfactory receptor neurons, copper is toxic to lateral line neurons that are directly exposed to contaminated waters.

**Aluminium**

Extensive research has been conducted on the effects of aluminium exposure on juvenile salmon life stages, particularly in combination with acidic conditions. Acid rain lowers the pH of river systems, causing aluminium leaching from soils to become more soluble and in turn increasing the presence of inorganic Al, the form most toxic to fish (McCormick et al. 2009). Labile (cationic/inorganic) forms of aluminium can accumulate in fish gills, where high concentrations result in mortality due to respiratory and ionoregulatory dysfunction. Work by Berntssen et al. (1997) demonstrated that Atlantic salmon smolts secrete excessive amounts of mucus in response to low pH/aluminium conditions and use this as a binding defence mechanism to protect from aluminium toxicity.

Population effects from sublethal aluminium/acid exposure can occur through the inhibition of gill Na+/K+ ATPase activity, reducing hyposmoregulatory capacity and in turn decreasing marine survival probability. Following exposure of Atlantic salmon smolts to sublethal acid/aluminium conditions, Kroglund et al. (2007) found gill aluminium concentrations exceeding 20µg/g and depression in gill Na+/K+ ATPase activity. Adult return rates were also impacted with smolt- to-adult survival declining by 20-50% relative to control fish.
These findings were similar to Kroglund & Finstad (2003), where exposure of Atlantic salmon smolts to very low levels of aluminium (6 mg/L) in acidic conditions reduced marine survival by 20–30%. The exposed fish also migrated downstream slightly later than the controls, increasing the probability of smolts missing the optimum migration window. Monette et al. (2010) explored further how acid/aluminium causes hypoosmoregulatory impairment in Atlantic salmon smolts, examining it on a cellular level. After exposure to acid and moderate aluminium concentration (42 µg/L) smolts exhibited a large reduction in the number of total gill mitochondria rich cells, accompanied by an increase in the surface expression of mitochondria rich cells in the filament epithelium. The loss of gill mitochondria rich cells and their individual ion uptake capacity explains impaired ion regulation in freshwater and after seawater exposure. It was hypothesised that the increased surface expression of mitochondria rich cells in the filament epithelium is an attempt to increase the number of active mitochondria rich cells in the gill to compensate for impaired ion regulation. Exposure to acid and low aluminium concentration (11 µg/L), impaired seawater tolerance occurred even though Na+/K+ ATPase activity and mitochondria rich cell numbers were not altered. However, exposure did alter the morphology of the mitochondria rich cells. Cells were smaller within the filament epithelium and less bright, and were more elongate within the lamellar epithelium. Seawater acclimated mitochondria rich cells are typically larger and stain brighter for Na+/K+ ATPase presence. Therefore, the results indicated that low acid/aluminium exposure causes a reversal from seawater to freshwater type mitochondria rich cells, therefore reducing the salt secretory capacity of the gill.

Thorstad et al. (2013) investigated the delayed impact of short-term aluminium exposure on Atlantic salmon smolts in moderately acidified freshwater during the first 37 km of their marine migration. Immediate mortality was 20% higher than the control group during the first 10km of the marine migration, but fish that made it past this point seemed to recover, maintaining a normal migration speed and survival. The authors speculated that the proximate cause for elevated mortality for smolts in the first 10km following exposure was predation by marine fishes, as exposed fish experienced impaired physiological function thus increasing their probability of being eaten. Decreased plasma concentrations of GH were also observed in exposed smolts but no affect on IGF-I was found. Interestingly work by Monette et al. (2008) found the opposite, with a decrease in plasma IGF-I but no affect on GH levels in exposed smolts. GH and IGF-I work both independently and in concert to increase seawater tolerance by stimulating gill Na+/K+ ATPase activity and chloride cell proliferation, so although the results found were different, decreased levels of either hormone indicates impaired hypoosmoregulatory ability.

**VI. Concluding Remarks**

This review demonstrates how xenobiotic compounds in the freshwater environment are a significant driver in the decrease of returning marine salmonids, with lasting impacts on marine survival and population sustainability. Declining marine survival can no longer be attributed solely to changes in oceanic conditions and it is crucial that research into restoring salmon populations no longer considers the freshwater environment in isolation of the marine environment. Extensive evidence verifies impacts of individual contaminants on physiological development, migratory behaviour, early seawater survival, long-term seawater growth and natal stream homing in salmonids. However, cumulative effects of xenobiotic mixtures
and the time period between exposure in freshwater and entry into the sea may also be integral in terms of whether migration and survival in the marine environment is compromised. Therefore, future research efforts should be focussed on addressing these knowledge gaps.

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