

# Briefing Paper



## The Effect of Endocrine Disruptors on Fish

- Research shows Endocrine Disrupting Chemicals (EDCs) are causing abnormalities in sexual development, thus reducing fecundity in many plants and animals, including fish species.
- Current management strategies are not enforcing the precautionary principle.
- Urgent action is required to reduce EDCs reaching and bioaccumulating in aquatic ecosystems.

Endocrine Disrupting Chemicals (EDCs) are substances which interfere with endocrine systems and hormonal activities. EDC are threatening biodiversity on a large scale, with currently over 200 plant and animal species known or suspected to be affected by EDCs (Miyamoto and Burger, 2003). Aquatic organisms are at enormous risk from EDCs, which enter watercourses through both diffuse and point source pollution. There is a wide range of known EDCs, these include natural (e.g. oestrogen) and synthetic hormones (such as ethynylestradiol; found in contraceptive pills), industrial chemicals (such as alkylphenols, bisphenol A and ethoxylates), pesticides (tributyl tin. TBT), fungicides and herbicides (including atrazine, diazinon and permethrin) and some chemicals in domestic products (including some surfactants).

The Salmon & Trout Association (S&TA) believes that the precautionary principle should be implemented now to reduce the quantity of these substances reaching our watercourses, before potentially serious and irreversible damage becomes evident.

### Impacts

EDCs have been shown to cause most damage to fish at larval or development stages, by causing abnormalities in sexual development, behaviour and fertility (Gross-Sorokin *et al.*, 2006). Early life stages of other organisms are also generally more vulnerable than adults. The adverse alterations reported in the sexual development of exposed fish include:

- Gonadal intersex (gonads contain eggs and sperm developing simultaneously);
- Feminisation of reproductive ducts;
- Reduced gonad (testis and ovary) growth rate and size;
- Reduced gamete quality; and
- Increased levels of vitellogenin (egg-yolk protein), indicative of exposure to feminizing chemicals.

The molecular pathways causing these effects are poorly understood, in all probability diverse, and the wide range of effects caused by EDC exposure makes them difficult to assess and monitor. As well as causing intersex and feminisation, EDCs have been shown to affect courtship and territorial behaviour in three-spined stickleback (*Gasterosteus aculeatus*: Bell, 2001). Research on Atlantic salmon (*Salmo salar*) has also shown oestrogenic compounds can significantly inhibit the development of smolt physiology (Madsen *et al.*, 1997). EDC can also differ in the species they affect; for example, steroidal oestrogens can have a potent effect on fish but little endocrine effect on invertebrates such as copepods (Sumpter and Johnson, 2005). Inter-species susceptibilities are

poorly understood, with potential for significant vulnerability in species important for ecosystem stability and functioning.

Research from the UK clearly shows an association between the incidence of fish feminisation and their exposure to effluent discharges (Centre for Ecology and Hydrology (CEH), 2002; Jobling *et al.*, 1998). Data from fish populations also correlates the severity of intersex with the proportion of oestrogenic effluent (Jobling *et al.*, 2006). However, diffuse urban and rural pollution is also known to contribute to endocrine-disrupting effects.

Research on wild roach (*Rutilus rutilus*) in the UK indicates that more severely feminized fish are found in the older year classes, suggesting the condition is progressive (Environment Agency, 2004). These delayed effects mean exposure during early life stages could result in irreversible changes which remain undetected until the offspring reaches maturity (Lyons, 2006; Jobling *et al.*, 2006). Other studies indicate that some EDCs have the ability to bioaccumulate and can be passed on to offspring, resulting in transgenerational effects. For example, laboratory experiments on the Pacific oyster (*Crassostrea gigas*) have shown that exposure to the industrial chemical nonylphenol results in poor survival rates in subsequent generations (Nice *et al.*, 2003).

The occurrence of intersex individuals in fish populations is affecting their reproductive success and possibly their population stability. Studies assessing gamete quality of wild intersex roach have found moderately to severely feminised male fish have reduced sperm quality and quantity (on average 50% less), and were less able to release their milt compared with 'normal' males (Jobling *et al.*, 2002). Elevated levels of vitellogenin in male fish have also been shown to disrupt kidney function (Herman and Kincaid, 1988), and result in calcium loss and lipid diversion from the scales, which can make the fish more susceptible to disease (Carragher and Sumpter, 1991).

EDCs are a widespread problem, now known to affect freshwater, estuarine and even marine fish. Research from the Tyne, Mersey and Solway estuaries have shown signs of feminisation in male flounder (*Platichthys flesus*) (Allen *et al.*, 1999; Kleinkauf *et al.*, 2004). A study by CEFAS (2006) found cod (*Gadus morhua*) sampled from both the North and Irish Sea had elevated concentrations of vitellogenin, which correlated to the size their diet changed from mainly nektonic (free-swimming) to benthic organisms. This worryingly suggests cod are accumulating EDCs through their food-chain as the benthic organisms live in sediments which typically contain far higher concentrations of oestrogenic compounds than seawater (Scott *et al.*, 2006).

A synthetic oestrogen, ethinylloestradiol (EE2), commonly used in the contraceptive pill, is causing particular concern due to its widespread occurrence, high potency and persistent nature (Santos *et al.*, 2007). Concentrations in UK freshwater systems have been shown to reach 3.4 ng l<sup>-1</sup> (Williams *et al.*, 2003). This is very worrying as laboratory experiments have shown EE2 exposure in zebrafish (*Danio rerio*) of 1.67ng l<sup>-1</sup> increases vitellogenin synthesis (Fenske *et al.*, 2001), and 3 ng l<sup>-1</sup> exposure reduces reproductive development in males (Fenske *et al.*, 2005). A recent study by Santos *et al.*, (2007) demonstrated that current environmental concentrations of EE2 decreased the quality and quantity of gametes produced by both male and female zebrafish.

Oestrogen contamination of drinking water has also been attributed to reproductive disorders and reduced sperm counts in humans (Sharpe and Skakkebaek, 1993).

### **Actions being taken**

In 2004, the Environment Agency concluded that the effects of endocrine disruption on fish were sufficient to develop a risk management strategy for biologically active effluents that discharge into the aquatic environment. The Endocrine Disruption Demonstration Programme was established to assess the effectiveness and costs associated with removing steroid hormones at sewage treatment works (STW). This programme will run from 2005-2010 (Environment Agency, 2004).

The well-documented endocrine disruptor TBT, which leaches into aquatic systems from boat anti-fouling paint, was shown to cause intersex in dogwhelks, oysters and mussels. It now faces a European ban for most applications, coming into force on the 1<sup>st</sup> January 2008.

The S&TA helped lobby for ban of cypermethrin, a pyrethroid insecticide used in sheep dipping, which was shown to cause endocrine disruption in fish, including salmon (*Salmo salar*) and brown trout (*Salmo trutta*) (Jaensson *et al.*, 2007), and fatal impacts on aquatic invertebrates. The lobbying resulted in a temporary suspension on the sale of cypermethrin in February 2006. Further action is still awaited from the Government as to whether the ban will become permanent or if the destructive chemical will be reintroduced. In our view, evidence of both toxic effects and risks from endocrine disruption would make any reintroduction unwise and indefensible, and certainly in direct contravention of the Precautionary Principle.

The Environment Agency has funded the development of a model which predicts the concentrations of three steroid oestrogens, oestradiol (E2), oestrone (E1) and EE2, and their associated risk of causing endocrine disruption in fish in 10,313 river reaches in England and Wales (William *et al.*, 2008). The model calculated that 39% of river reaches were 'at risk' from causing endocrine disruption effects in fish. The regions at greatest risk were Thames, Midlands and Anglian regions, with 67%, 55% and 50% respectively of river reaches in the area predicted to be 'at risk' (William *et al.*, 2008).

### **Call for Further Action**

The S&TA feel the severity of the problem of EDCs requires urgent attention. We feel precautionary action should be implemented now to reduce the exposure to EDCs in the aquatic environment before it is too late. We feel sufficient evidence exists to demand:

- Universal threshold levels for total endocrine loads and specific oestrogen threshold standards in STW discharges by 2012.
- Immediate action to reduce natural and synthetic steroid oestrogens in priority STW discharges in 'at risk' areas.
- Reduction in the use of other manufactured chemicals with endocrine activity and replacement with safer alternatives. Priority must be given to the prevention of the release, rather than end-of-pipe solutions.
- The development of new assays and screening methods for the identification of endocrine disruptors relevant to humans and wildlife.
- Research to be adequately funded, prioritised and co-ordinated. All species in decline to be investigated for potential effects of endocrine disruptors, and all relevant data to be made publicly available wherever possible.
- Reassessment of current toxicity tests and chemical standards, as current assessments are dominated by single-chemical exposure studies. The environmental reality is that these chemicals occur mixed with other in effluents and in rivers, and are therefore likely to instigate additive effects. Precautionary limits/standards should therefore be set to take this into account.
- Phasing out of persistent chemicals known to accumulate in the environment.
- Investigations into population level impacts caused by EDC exposure, including population declines and loss of genetic diversity.
- Further research into the other effects of EDC exposure as, beside reproductive disruption, these chemicals have the potential to affect other biological functions also under hormonal control such as growth, development, metabolism, immune response and metamorphosis (in amphibian species).

### **Conclusion**

The full extent of endocrine disruption and its effects on the environment are not fully understood. However, there is a consensus amongst experts that they are far more widespread than currently confirmed by experimentation.

The future of commercial and recreational, marine and freshwater fish populations could be threatened by the effects of EDC exposure. There is also significant potential to perturb whole ecosystems, from which many societal benefits arise. The S&TA strongly believes that this issue needs addressing now to help safeguard our native fish populations, and to enable us to be confident of attaining the Water Framework Directive (WFD) requirement of 'good ecological status' in all of our waterbodies by 2015.

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