

ENDOCRINE DISRUPTION IN THE MARINE ENVIRONMENT

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The link between endocrine disruption and marine incidents management is not an obvious one. This paper provides some background information on the problem of endocrine disruption, the current position in marine waters and poses the question of the contribution of shipping to the problem, rather than answering it.

The term endocrine disruption came into common use following the observation that fish showed intersex conditions in UK rivers. Male fish were found to have egg-like structures in their testes and egg yolk protein (vitellogenin) at high levels in their bloodstreams. Work at CEFAS and Brunel University in the UK demonstrated that if fish (trout) were held in the diluted effluent of sewage treatment works they quickly developed high levels of vitellogenin (VTG) in their blood (e.g. Purdom *et al.*, 1994, Routledge *et al.*, 1998). Further from sewage inputs the levels of VTG were lower and it seemed that the effect was caused by chemical(s) in sewage. The chemicals causing the effect were isolated and identified by CEFAS. They were found to be a number of different human derived steroids such as estradiol and the active ingredient of the contraceptive pill; ethinyl estradiol (Desbrow *et al.*, 1998; Routledge *et al.*, 1998).

These compounds were not the only chemicals found to cause fish to change sex and laboratory work on exposures to fish or to cell-lines have produced a long list of industrial and biocidal compounds that are able to mimic natural hormones that have profound effects on reproductive cycles. This list of reproductive endocrine disrupters includes some surfactants such as nonyl-phenols, phthalates used as plasticizers and even some classically persistent and toxic pesticides such as the breakdown product of DDT – ppDDE. (Jobling *et al.*, 1995).

The activity is not always estrogenic in effect (i.e. mimicking female hormones), but can be androgenic (mimicking male hormones) or even more subtle such as anti-androgenic or anti-estrogenic. As more processes and pathways have been examined it has become clear that reproductive effects are not the only targets of hormone mimics and other vital functions such as growth and behaviour can also be affected. Some flame retardant compounds for example can have effects on the thyroid system and impact growth.

The evidence base for widespread effects has been growing rapidly over the last decade. Effects that were previously described as reproductive toxicity have been reconsidered to be a result of endocrine disruption and there are examples of effects from birds (egg shell thinning as a result of DDT exposure) to alligators (penis malformations from pesticide exposure). The reviews by for instance Metzler (2001) and Damstra *et al.* (2002) gather the evidence together. Most of these effects are close to point sources and it seemed likely that in marine systems the dilution, dispersion and breakdown of chemicals (particularly steroids) would reduce concentration below effect levels. There is, however, at least one very good example of a widespread effect of a chemical in coastal waters at very low levels due to bioaccumulation in animals; the case of TBT is well documented.

Tributyltin (TBT) was introduced as a biocide in antifouling paints in the 1960s and was widely used on pleasure craft and ships by the mid 1970s. Research at CEFAS in the 1980s showed that in estuaries with large numbers of pleasure craft Pacific oysters would not grow normally and laboratory experiments showed TBT to be the culprit (Waldock and Thain, 1983). Marine snails proved to be even more sensitive to TBT and at single nanogram per litre (part per trillion) concentrations female whelks (*Nucella lapillus*) grew a penis and died as a result of the vas deferens blocking the oviduct. These effects were described as imposex (male features imposed on females) (Matthiessen and Gibbs, 1998).

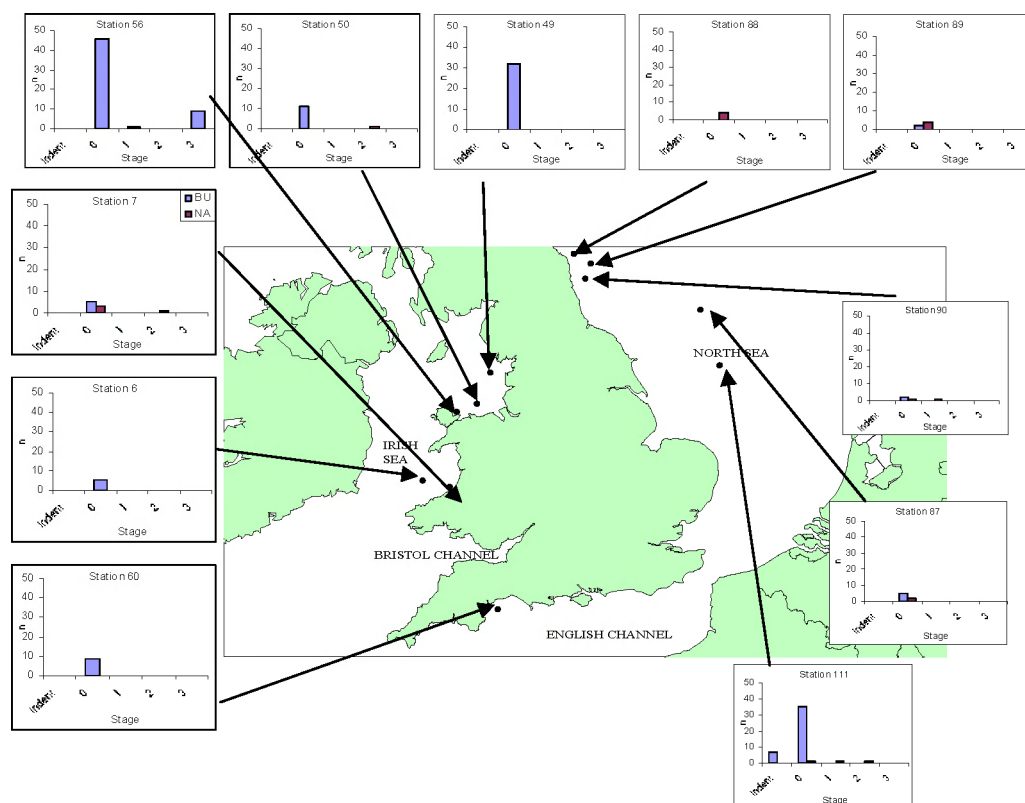


Fig. 1. Imposex in whelks sampled in 1998 (*Buccinum undatum* = blue bars; *Neptunea antiqua* = purple bars. Stages 0 to 3 indicate increasing severity of effect (N= the number of animals tested). (CEFAS unpublished data).

This example is a coastal one since *Nucella* is found on rocky shores. It seemed unlikely at the time that whelks offshore would be affected, but work in the Netherlands (ten Haller-Tjabbes, 1996) and by CEFAS (unpublished data) shown in Fig. 1) showed extensive imposex in larger whelks (*Buccinum* sp. and *Neptunia* sp.). The effects were particularly marked in shipping lanes and close to disposal sites for dredged material.

Is there similar evidence that fish might show endocrine disruption in marine waters? CEFAS used the flounder as a sentinel species to look for effects in estuaries (Allen et al 1999). The results showed that in waters historically impacted by industrial and domestic discharges the level of VTG in male fish was high and there was evidence of intersex in some individuals. During a five-year study (Kirby et al, 2004) the severity of effect was seen to decrease as sewage discharges were replaced and water quality in UK estuaries generally improved. One exception to this trend was the River Tees in the North of England where levels of VTG remained high year

on year. Sewage inputs appeared to be the major driver of effects on fish and if the causative compounds were steroids, they should degrade and not affect animals offshore.

One way to test this assumption was to place fish in cages offshore. This opportunity arose during an international workshop evaluating biological effects techniques (BECPELAG) <http://www.iis.niva.no/PELAGIC/web/index.htm>. In this experiment cod were deployed in cages along putative pollution gradients from land and from offshore oil platforms. The results showed that a very low level of VTG was indeed induced in male cod and those closer to oil platforms had a higher level than those further away.

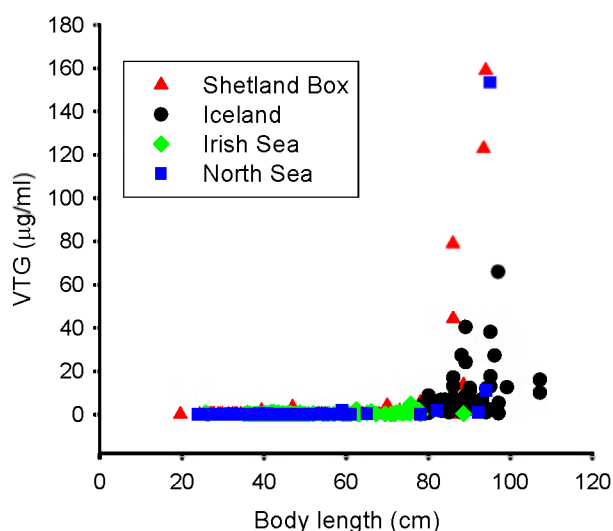


Fig. 2. Concentrations of vitellogenin in the plasma of male cod captured from the NE Atlantic (reproduced with permission from Prof. A. Scott).

This was a surprising finding and led us to look at VTG in wild cod from the North and Irish Seas and from Iceland waters where we assumed any pollution levels would be low. The results showed that small fish had only low concentrations of VTG but larger ones had increased levels. The point at which the production of VTG seemed to increase dramatically (Fig. 2) coincides with a time in the life cycle when the fish are able to take larger prey items such as bottom-dwelling fish and crabs (Scott *et al.*, 2006). From this evidence it seems that the driver for the change in production in VTG may be due to uptake from prey items of more persistent and bioaccumulating compounds that are widespread in the environment at very low levels. Presently we have not yet identified the cause of the problem and the current programme at CEFAS is aimed at quantifying similar effects in prey species such as the dab that live in close association with sediments.

So is there a link of endocrine disruption to shipping such as by accidental spillages of oil and chemicals? For the example of TBT and whelks the link to shipping use of antifouling paints is clear but for endocrine disruption in fish it seems more likely that the cumulative and chronic effects of shipping, land-based and offshore-based industry, or even global distillation of persistent compounds might be to blame. The next challenge is to develop a strategy to identify the causes and trends in the effects. If this is something that is reducing over time we might be looking at a legacy of historical inputs. If the trend shows an increase, the risk assessment is different matter.

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