



Does exposure to domestic wastewater effluent (including steroid estrogens) harm fish populations in the UK?



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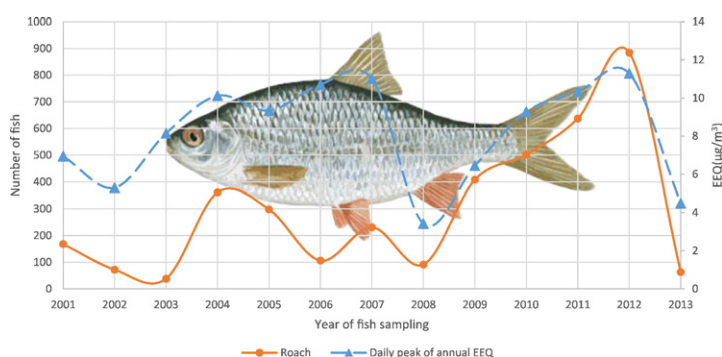
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HIGHLIGHTS

- Fish density data for four species at 38 sites over several years were examined.
- No clear relationship between fish density and wastewater estrogen exposure.
- Temporal variation of *Rutilus rutilus* (roach) over several years was examined in two rivers.
- No clear negative impact of temporal change in wastewater estrogen exposure on roach.
- Wastewater estrogen exposure is not catastrophic for four common cyprinid species.

GRAPHICAL ABSTRACT



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ABSTRACT

Historic fisheries data collected from locations across the UK over several years were compared with predicted estrogen exposure derived from the resident human population. This estrogen exposure could be viewed as a proxy for general sewage (wastewater) exposure. With the assistance of the Environment Agency in the UK, fisheries abundance data for *Rutilus rutilus* (roach), *Alburnus alburnus* (bleak), *Leuciscus leuciscus* (dace) and *Perca fluviatilis* (perch) from 38 separate sites collected over 7 to 17 year periods were retrieved. From these data the average density (fish/m²/year) were compared against average and peak predicted estrogen (wastewater) exposure for these sites. Estrogen concentrations were predicted using the LF2000-WQX model. No correlation between estrogen/wastewater exposure and fish density could be found for any of the species. Year on year temporal changes in roach population abundance at 3 sites on the middle River Thames and 4 sites on the Great Ouse were compared against estrogen exposure over the preceding year. In this case the estrogen prediction was calculated based on the upstream human population providing the estrogen load and the daily flow value allowing concentration to be estimated over time. At none of the sites on these rivers were temporal declines in abundance associated with preceding estrogen (effluent) exposure. The results indicate that, over the past decade, wastewater and estrogen exposure has not led to a catastrophic decline in these four species of cyprinid fish.

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1. Introduction

For thousands of years man's activities have disturbed the river environment. The river can be exploited as a food, drinking water and

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irrigation resource, used as a highway for goods transport, a generator of energy, and a conduit for our waste products. Rivers are also feared as a source of flooding, so they may be excavated to ensure they act as efficient drains. Many of these human activities have had damaging impacts on the river as a habitat for fish. The fish that live in our rivers are at, or near, the top of a complex food web. Unfortunately, the abundance of fish in rivers have not been consistently recorded through history, but it would appear that serious declines in some major rivers in the UK occurred from the 1930s to 1950s. Inadequate treatment of sewage and industrial waste led to the disappearance of fish in the lower reaches of big rivers like the Trent (Mann, 1989), Mersey (Jones, 2006) and Thames rivers (Wheeler, 1979). Fortunately, an increasing appreciation of the amenity value of rivers, legislation, industrial decline, and more investment in water treatment has largely eliminated the problem of gross organic pollution, at least in the UK, with the exception of occasional combined sewer overflows. However, it has been increasingly recognised that as individuals we now consume many more pharmaceuticals and personal care products (PPCPs) than ever before. Sewage treatment plants (STPs) were never designed to remove all of such micropollutants. Could it be that we are now harming our river environment and fish through this insidious 'invisible' pollution (Daughton and Ternes, 1999)?

When we examine the tissue of freshwater wild fish, we can certainly find many hydrophobic pollutants present (Jurgens et al., 2015), but what evidence do we have that chemicals can harm fish individuals and populations? There are, of course, examples of extreme one-off pollution events with industrial, oil and farm waste killing fish (Giger, 2009; Kubach et al., 2011; Kennedy et al., 2012; Eros et al., 2015). But our concern here is with chronic pollution. The strongest evidence seems to be related to metals. Soil acidification thanks to 'acid rain' from coal combustion led to the release of the toxic monomeric forms of Al into upland streams and lakes, leading to fish kills in the 70s and 80s (Henriksen et al., 1984). Freshwaters with high metal concentrations associated with mine waste or heavy industry have also had a recorded impact on fish populations (Filipek et al., 1987).

Thus, there are examples of fish kills due to exposure to acutely toxic chemicals at pollution hot-spots. But what of the chemicals routinely discharged in domestic sewage effluent? The chronic sub-lethal phenomena of endocrine disruption, associated with sewage effluent, has had and continues to have a major influence on our thinking regarding PPCPs. There is overwhelming evidence that a ubiquitous component of sewage effluent has led to endocrine disruption effects in resident wild roach (*Rutilus rutilus*) (Jobling et al., 1998; Jobling et al., 2006). The most likely agents being the natural and synthetic steroid estrogens excreted by humans (Desbrow et al., 1998). Similarly, there is evidence that increasing exposure to wastewater effluent elevates the level of the stress hormone cortisol in fish, at least in stickleback (Pottinger et al., 2016). Recently, a disastrous decline in Asian vultures has been strongly linked to the non-steroidal anti-inflammatory agent diclofenac (Oaks et al., 2004). Given that diclofenac is a common constituent of sewage effluent, this has now risen as a concern for fish in rivers too (Schwaiger et al., 2004; Cuklev et al., 2011). So now both the steroid estrogens and diclofenac have been identified by the European Union as requiring special monitoring, with a view to control at a later stage (COM(2011)876). It is also recognised that freshwater fish will be exposed to a wide range of pharmaceuticals and this chronic exposure is a concern (Fent et al., 2006). Given the fear and uncertainty over this chronic exposure to PPCPs, there are increasing arguments that an end of pipe solution at STPs will be needed to protect aquatic wildlife (Eggen et al., 2014; Oehlmann et al., 2014; Stamm et al., 2015). But is this fear justified? We know that if the synthetic estrogen ethinylestradiol reaches a high enough level some fish populations will collapse (Kidd et al., 2007). It can be presumed that our consumption of PPCPs has been growing steadily since the 1970s (Richardson and Ternes, 2014), so it would seem a reasonable question to ask how fish populations have fared since then? Rather surprisingly, examining responses in the abundance

of wildlife populations to chemical or estrogen exposure has not been a frequently asked question in the aquatic environment (Mills and Chichester, 2005; Johnson and Sumpter, 2016). In contrast, such approaches are seen as central in the terrestrial environment, such as with neonicotinoid pesticides and bees (Woodcock et al., 2016).

Unfortunately, until recently there has been little systematic collection of data on fish populations in rivers. However, some species that were relatively common in many UK lowland rivers have declined or disappeared, was this due to chemicals or estrogens even? These include the migrating salmonids (*Salmo salar* and *Salmo trutta*) and Barbel (*Barbus barbus*) but these declines are most closely linked with habitats becoming unsuitable (Johnson and Sumpter, 2014). We are sadly aware that there has been a decline in eel numbers in many parts of the world. But the evidence suggests that the eel decline, which started in the early 1980s, occurred in a period of reduced chemical challenge (Jurgens et al., 2015). Eel populations appeared to have done better in the much more polluted post-war period. There are, however, quite a lot of encouraging information on cyprinid fish, such as bream (*Abramis brama*), whose average length for 5 year olds increased from 1966 to 1976 in the Dutch Rhine (Slooff and Dezwart, 1983) and whose condition steadily improved in several major German rivers from 1992 to 2014 (Teubner et al., 2015). Data appear to show that UK cyprinid populations have been recovering since reaching a low-point in the 1950–1970s period (Mann, 1989; Robinson et al., 2003). However, although encouraging, the limited information available is too coarse and not sufficiently focused to address whether the chemicals routinely present in domestic sewage effluent are harming wildlife populations.

To begin addressing the question in a more systematic way, we compared routine fish population monitoring data collected in the UK by the Environment Agency of England and Wales with predicted wastewater effluent exposure. This study tested the following hypotheses:

- Any fish population (average density) will be severely harmed by average exposure to domestic wastewater
- Any roach population will be severely harmed by temporal increases in domestic wastewater exposure

It should be pointed out the intention of this study was not to identify the most important environmental factors that stimulate fish population abundance and aid recruitment in UK rivers. The complex interactions of flow, temperature, habitat, disease, and position of the Gulf Stream in the North Atlantic, amongst others, are all likely to be playing a role together. Nor will simple population data, such as we use here, reveal sub-lethal impacts that could hamper fish performance and well-being. The aim was to see whether it was possible to rule out sewage and estrogen exposure as having a consistent and seriously damaging impact on fish populations.

2. Materials and methods

2.1. Fisheries monitoring data

The fisheries data were collected for the National Fisheries Monitoring Programme by the Environment Agency of England and Wales. Only sites where the electro-fishing method was used for counting were examined. The method involves a boom boat applying a 50 Hz pulsed DC current to the water. Downstream runs may be up to 2 km between dividing locks or be of shorter duration, such as around islands or weir pools (Table 1). The sampling runs were mainly carried out in close proximity to the river margins, as the method is somewhat ineffective at depths greater than 1.5 m. The electric current stuns the fish, which on floating to the surface are collected, identified, counted, and their fork length recorded before being returned to the water. For the data examined in this study, fish down to 21 mm in length were recorded. The fish counts were recorded and can be normalised to the survey area. This sampling method is not suitable for counting bream, which are

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