# Predicting the effects of endocrine disrupting chemicals on healthy and disease impacted populations of perch (perca fluviatilis) 

A.R. Brown ${ }^{\text {a,* }}$, A.M. Riddle ${ }^{\text {a }}$, I.J. Winfield ${ }^{\text {b,1 }}$, J.M. Fletcher ${ }^{\text {b,1 }}$, J.B. James ${ }^{\text {b,1 }}$<br>${ }^{\text {a }}$ AstraZeneca UK Limited, Brixham Environmental Laboratory, Freshwater Quarry, Brixham, Devon TQ5 8BA, UK<br>${ }^{\mathrm{b}}$ Centre for Ecology and Hydrology, Lancaster Environment Centre, Library Avenue, Bailrigg, Lancaster, Lancashire LA1 4AP, UK

Received 21 June 2004; received in revised form 14 February 2005; accepted 29 March 2005


#### Abstract

Long-term data from the perch population of the north basin of Windermere, UK, were combined with effects data from laboratory toxicity studies of survival, growth and reproduction in other species to assess the likely impact on fish populations of different levels of exposure to nonylphenol (NP) and ethinylestradiol (EE2). A multi-stage Delay Differential Equation fish population model was used to simulate the perch population during two periods when it showed contrasting population structures (1968-1973 and 1983-1988) as the result of a disease outbreak in 1976, and to extrapolate the effects of chemical exposure to EE2 and NP observed in the laboratory to the environment. In the absence of chemical exposure, model simulations predicted population numbers (females) in line with those derived from field survey data. Effects predictions were made for long-term exposure ( 20 years) to low and high doses of EE2 ( 1 and $10 \mathrm{ng} 1^{-1}$ ) and NP ( 1 and $30 \mu \mathrm{~g} \mathrm{l}^{-1}$ ). The sustained high-level exposure of EE2 had a high probability of causing the extinction of a confined fish population such as that in Windermere, however, such an exposure scenario is unlikely. Far greater uncertainty surrounds the prediction of effects due to low-level exposure of fish populations and even though effects may appear to be significant in laboratory studies, such as fecundity lowered by $30 \%$, they may not necessarily translate into significant population effects in the field. This is especially true if life-history data show high natural variability in terms of individual vital rates. Our work suggests that for a decline in the perch population numbers to be significant in Windermere, it would have to be substantially more than $50 \%$. Our model predictions indicated that the post-disease population was generally more vulnerable than the pre-disease population and had a significantly greater probability of declining by $50 \%$ following single chemical exposure, but both populations were equally likely to decline following multiple chemical exposure. Rate of population recovery was shown to be a more sensitive measure in terms of differentiating the effects of low and high chemical exposure as well as the vulnerability of populations with contrasting structures, histories or levels of background stress.


© 2005 Elsevier B.V. All rights reserved.
Keywords: Life-history; Perch; Endocrine disrupting chemical; Ethinylestradiol; Nonylphenol; Population modelling; Windermere

[^0]
## 1. Introduction

Several attempts have been made to correlate fish population size and susceptibility with key environmental variables, mainly temperature and food availability (Beverton and Holt, 1957; Radovich, 1962; McFadden et al., 1967; Cushing, 1969; Nelson et al., 1977). Schaaf et al. (1987) derived an "index of reproductive value" based on the age distribution of egg production, which was shown to be proportional to the time taken to recover from an acute perturbation such as a chemical spill. These authors also highlighted that susceptibility may vary from one species to another and from one population to another within the same species depending on the level of background stress they are already under. Whilst offering a useful initial screen, susceptibility measures fail to provide a true reflection of risk.

Fish population dynamics models reflecting the lifehistory of fish in the wild, have been shown to be highly applicable in ecological risk assessments (Barnthouse, 1993; Schaaf et al., 1993; Brown et al., 2003). Many applications of models have been developed since the 1950s for the assessment of fish populations, to assess stocks, potential yield for fisheries, sustainability and predation (Wilson et al., 1991; Barnthouse, 1996; Aubone, 2004). Also effects such as habitat fragmentation, nutrient enrichment and physical hazards such as power plant intakes have been studied using population models (Summers, 1989; Morita and Yokota, 2002). Application of models to assess the potential effects of toxic chemicals on fish populations is more recent (1980s). Barnthouse et al. (1987) used a logistic model to investigate the effects of toxic contaminants to fish populations and Schaaf et al. (1987) and Barnthouse (1993) applied Leslie matrix models to simulate the effects of pollution on fish populations with different life-history strategies. The Delay Differential Equation (DDE) approach has also been used to assess the effects of chemical stressors on fish populations (Wood, 1999; Brown et al., 2003), but has more generally been applied in agriculture and forestry (Papastamati et al., 2002; Louie et al., 2002; Acevedo et al., 1996) as well as conservation of terrestrial fauna (Schley and Bees, 2003). Individual based models are now generally widely used in many branches of species and population modelling and have been applied to assess the effects of poor environmental quality on fish
populations (Rose et al., 1999; Van Winkle et al., 1993; Jaworska et al., 1997).

Not surprisingly, fewer population models have been developed for coarse fish than for commercially important game fish species. However, several models have been developed for the yellow perch (Perca flavescens) including individual based models to estimate the impact of power plants (Jensen, 1982) and the effects of climate change (Fang et al., 2004), while for the Eurasian perch (Perca fluviatilis) a model for ten Swiss lakes was developed by Tyutyunov et al. (2002) for the prediction of optimal harvesting and Ylikarjula et al. (2002) developed a model in order to understand the role of density dependence in dampening fluctuations in abundance and biomass. In this paper, life-history data for perch (Perca fluviatilis) in the north basin of Windermere, England's largest lake, are combined with effects data for nonylphenol (NP) and ethinylestradiol (EE2) taken from laboratory studies involving other species of fish, and used to estimate the possible impacts of these chemicals via population modelling. The DDE model developed by Brown et al. (2003) has been adopted because of the flexibility of the method to account for short term changes in parameters such as fecundity (spawning takes place over a limited time) and exposure (pest control spray activities that can last for only a few days) whilst being quick to run and therefore suitable for incorporation in a Monte Carlo procedure to estimate risk statistics. The model has been extended to simulate the dynamics of the Windermere perch population which, includes individuals living for up to 15 years. Initially "control" models have been built around the life-history data for pre- and post-disease populations (1968-1973 and 1983-1988, respectively) in the north basin of Windermere. These models were then parameterised with the laboratory effects data to simulate the population responses to low (realistic) and high exposures of ethinylestradiol and nonylphenol. Probabilities of population decline have been estimated for the control and exposure scenarios, based on the uncertainties in the fecundity, growth and mortality data from the laboratory and the field.

## 2. Materials and methods

The Eurasian perch (Perca fluviatilis) is a temperate (and sub-Arctic), freshwater fish extending from Great

# https://daneshyari.com/en/article/9443418 

Download Persian Version:

## https://daneshyari.com/article/9443418

## Daneshyari.com


[^0]:    * Corresponding author. Tel.: +44 1803 882882; fax: +44 1803882974.

    E-mail address: belreception@ astrazeneca.com (A.R. Brown).
    ${ }^{1}$ Tel.: +44 1524 595800; fax: +44 152461536.

