Study Links Water Pollution With Declining Male Fertility

New research strengthens the link between water pollution and rising male fertility problems. The study, by Brunel University, the Universities of Exeter and Reading and the Centre for Ecology & Hydrology, shows for the first time how a group of testosterone-blocking chemicals is finding its way into UK rivers, affecting wildlife and potentially humans. The research was supported by the Natural Environment Research Council and is now published in the journal *Environmental Health Perspectives*

The study identified a new group of chemicals that act as 'anti-androgens'. This means that they inhibit the function of the male hormone, testosterone, reducing male fertility. Some of these are contained in medicines, including cancer treatments, pharmaceutical treatments, and pesticides used in agriculture. The research suggests that when they get into the water system, these chemicals may play a pivotal role in causing feminising effects in male fish.

Earlier research by Brunel University and the University of Exeter has shown how female sex hormones (estrogens), and chemicals that mimic estrogens, are leading to 'feminisation' of male fish. Found in some industrial chemicals and the contraceptive pill, they enter rivers via sewage treatment works. This causes reproductive problems by reducing fish breeding capability and in some cases can lead to male fish changing sex.

Other studies have also suggested that there may be a link between this phenomenon and the increase in human male fertility problems caused by testicular dysgenesis syndrome. Until now, this link lacked credence because the list of suspects causing effects in fish was limited to estrogenic chemicals whilst testicular dysgenesis is known to be caused by exposure to a range of anti-androgens.

Lead author on the research paper, Dr Susan Jobling at Brunel University's Institute for the Environment, said: "We have been working intensively in this field for over ten years. The new research findings illustrate the complexities in unravelling chemical causation of adverse health effects in wildlife populations and re-open the possibility of a human - wildlife connection in which effects seen in wild fish and in humans are caused by similar combinations of chemicals. We have identified a new group of chemicals in our study on fish, but do not know where they are

coming from. A principal aim of our work is now to identify the source of these pollutants and work with regulators and relevant industry to test the effects of a mixture of these chemicals and the already known environmental estrogens and help protect environmental health."

Senior author, Professor Charles Tyler of the University of Exeter, said: "Our research shows that a much wider range of chemicals than we previously thought is leading to hormone disruption in fish. This means that the pollutants causing these problems are likely to be coming from a wide variety of sources. Our findings also strengthen the argument for the cocktail of chemicals in our water leading to hormone disruption in fish, and contributing to the rise in male reproductive problems. There are likely to be many reasons behind the rise in male fertility problems in humans, but these findings could reveal one, previously unknown, factor."

Bob Burn, Principal Statistician in the Statistical Services Centre at the University of Reading, said: "State-of-the-art statistical hierarchical modelling has allowed us to explore the complex associations between the exposure and potential effects seen in over 1000 fish sampled from 30 rivers in various parts of England."

The research took more than three years to complete and was conducted by the University of Exeter, Brunel University, University of Reading and the Centre for Ecology & Hydrology. Statistical modelling was supported by Beyond the Basics Ltd.

The research team is now focusing on identifying the source of anti-androgenic chemicals, as well as continuing to study their impact on reproductive health in wildlife and humans.