

## Methods to detect effects of xenoandrogens and aromatase inhibiting compounds in the riverine environment.

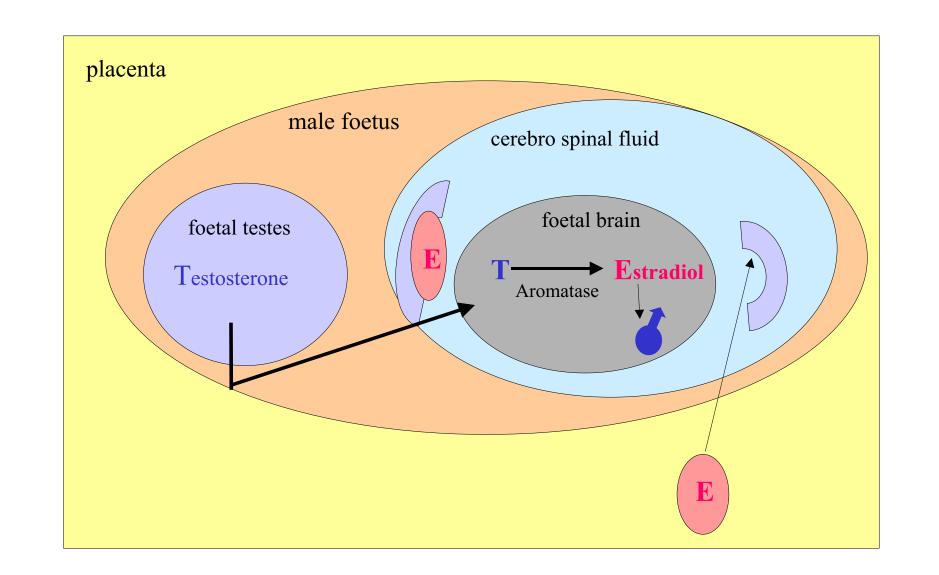


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Investigations of the mechanisms causing the imposex phenomenon in marine snails indicate, that tinorganic compounds impair oestradiol biosyntheses.

Apart from the control of female reproduction in vertebrates oestradiol also acts on male imprinting of the foetal brain (Fig.1a) and testicular function in adult males (Fig 1b).



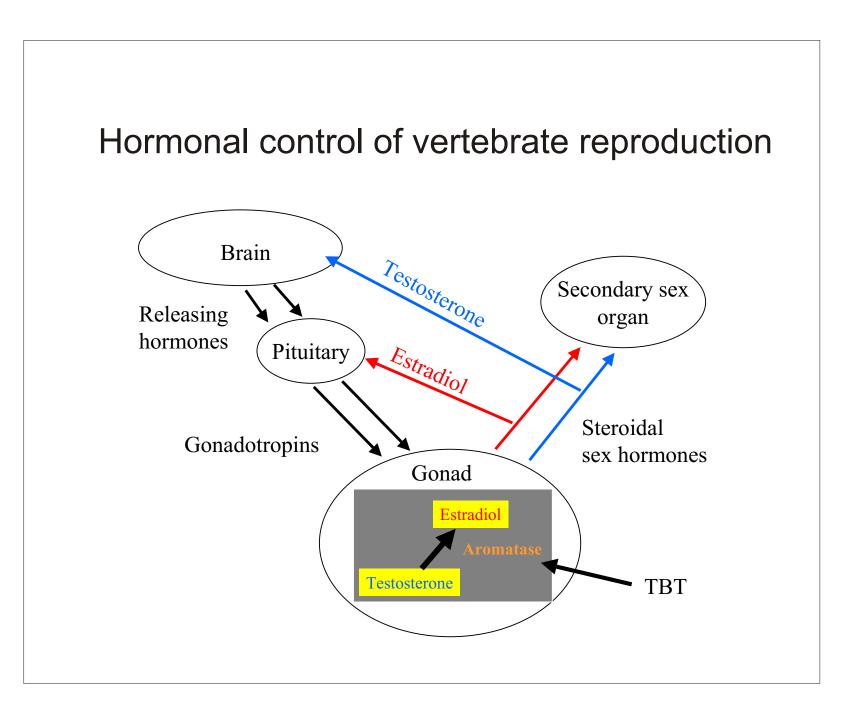
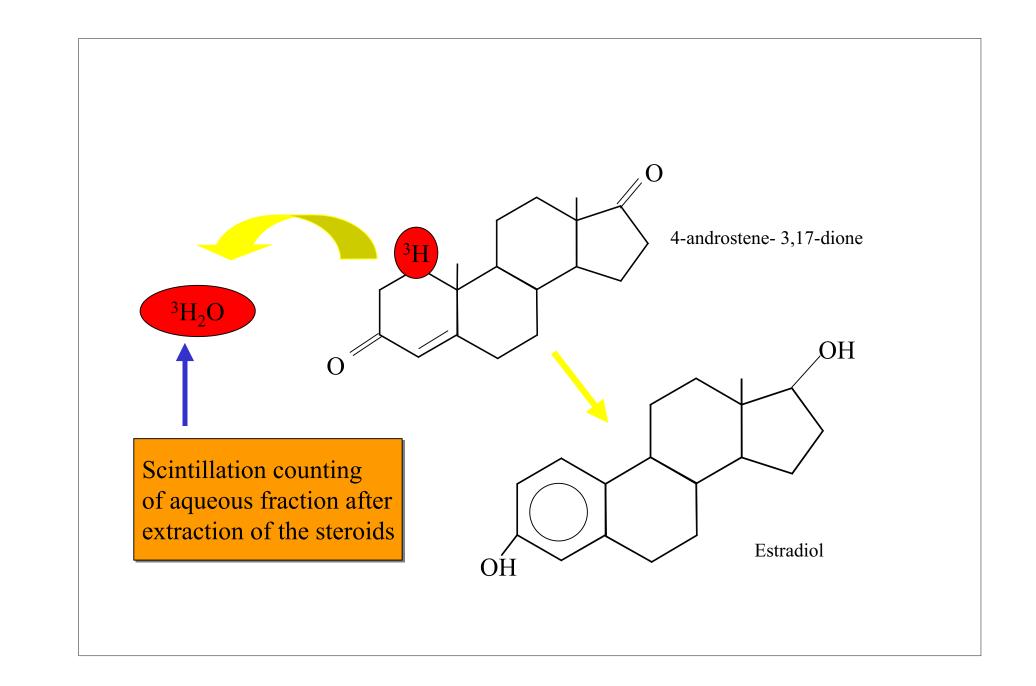


Fig. 1a: Perinatal imprinting of mammalian brain. The foetal testes secrete testosterone (T). Only the in situ conversion of testosterone in the foetal brain causes male imprinting. Estradiol binding proteins EBP which are present in the cerebro spinal fluid prevent the passage of exogenous e.g. maternal estradiol into the brain.

Fig. 1b: In male vertebrates testicular estradiol biosynthesis indicates a testosterone surplus and causes reduced secretion of gonadotropins and thereby reduced testosterone biosynthesis.

Measurements of organic sediment pollution of Hessian surface waters show a high burden of tinorganic compounds especially in harbours. Ecotoxicological risk assessment of tinorganic pollution requires data on effectiveness of the single compounds regarding endocrine disruption in vertebrates as well as in invertebrates. In order to check effects of single compounds on oestradiol biosynthesis, a radiochemical assay (tritiated water technique) has been applied (Fig. 2a). In vitro tests have been carried out with gonadal and neuronal tissue containing aromatase. These tests are useful tools for an initial assessment of body burdens of tinorganic compounds.

1,4µg TBT/L causes 50% reduced oestradiol biosyntheses in in vitro tests with perch ovarian tissue (Fig. 2b).



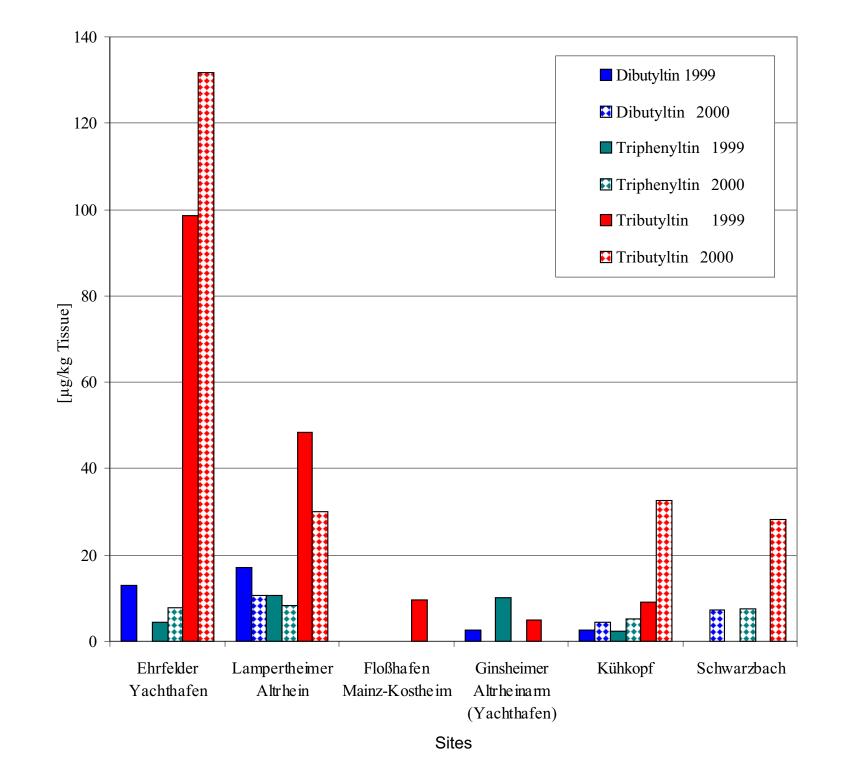
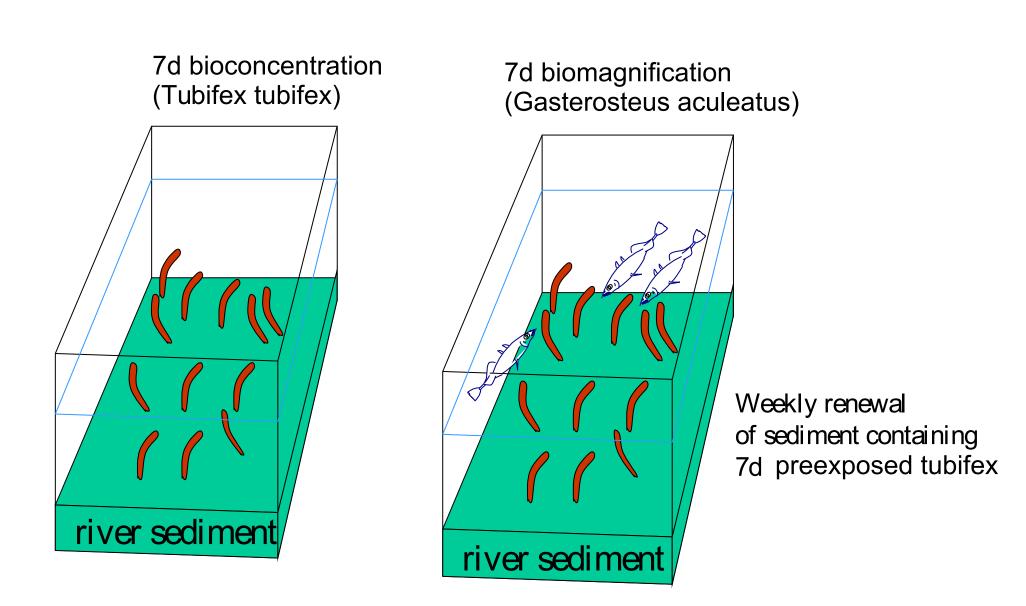


Fig. 2a: Principle of aromatase measurement

Since the bioavailability of sediment bound xenobiotics is not predictable, common schemes of risk assessment do not fit. Data on effective concentrations regarding different limnic organisms in adequate exposure systems are not available. Considering adsorption of the compounds the following experimental systems have been developed. Impacts on androgenic control of reproduction have been demonstrated using testosterone dependent reproductive renal function in *Gasterosteus aculeatus* (Fig. 3b). In this experiments sticklebacks were exposed to tinorganic polluted riverine sediments in laboratory (Fig. 3a). Sticklebacks were fed on *Tubifex tubifex,* which served as a vehicle for the organic contaminants.



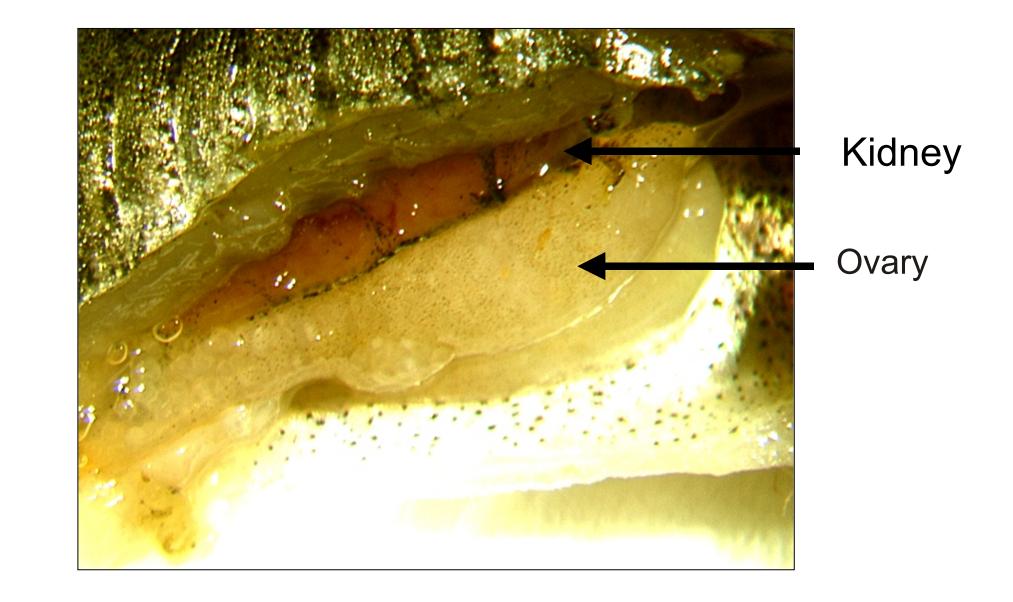
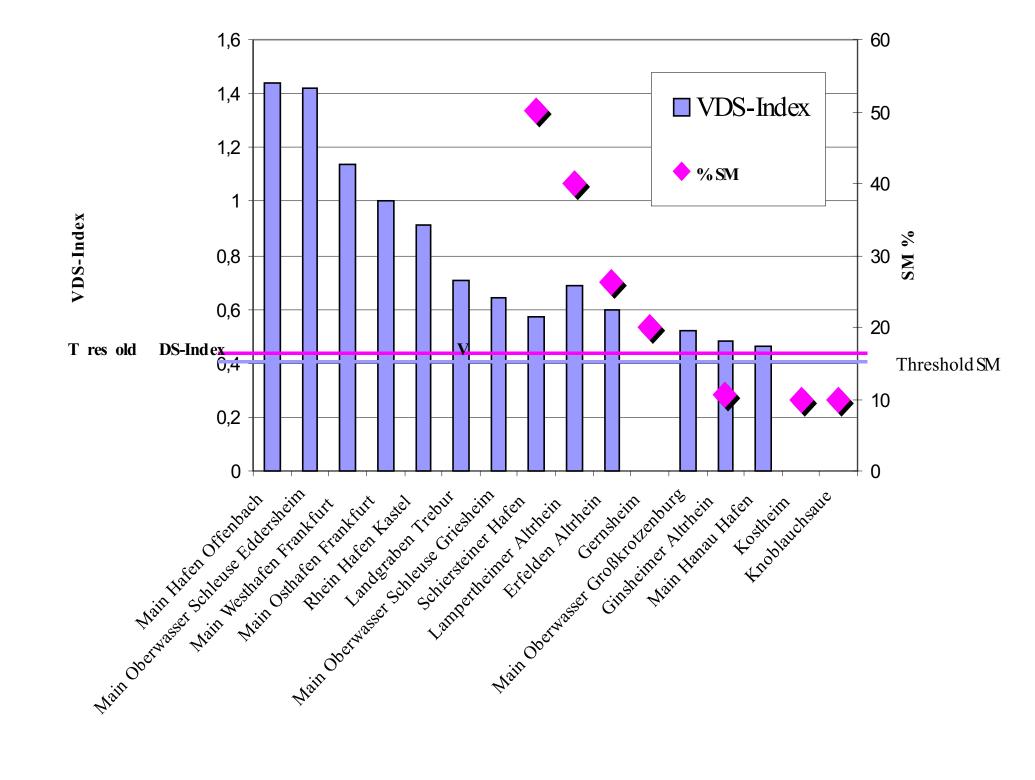


Fig. 3a: Testsystem to monitor endocrine effects of sediment bound compounds in laboratory experiments

Fig. 3b showes the caudal dorsal body cavity of a female stickleback, after 6 weeks exposure to sediments contaminated with tinorganic compounds. The kidney has been transformed to a glue producing gland. In unexposed females renal tissue is not visible, when opening the body cavity in this way.



Invertebrate endocrine toxicity of sediment born tinorganics has been evaluated using the induction of imposex in snails (*Hinia reticulata*) after 4 week exposure to tinorganic contaminated riverine sediments in laboratory experiments. The degree of masculinization of genetic female snail secondary sex organs (induction of penis and vas deferens) is given as VDS index (Fig. 4).

The data on imposex induction are compared with the incidence of pubertas praecox observed in male perch (SM) from the coresponding riverine sites. These data indicate that tinorganic compounds may cause masculinization of vertebrates as well as of invertebrates in the areas under investigation.

Fig. 2b: Body burden of tinorganic compounds in perch caught from several sites.