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Evaluation of the Antimicrobial Triclocarban in a Short-Term Reproduction Assay with the Fathead Minnow (*Pimephales promelas*). Jensen, K.M.*, Cavallin, J.E., Durhan, E.J., Kahl, M.D., Makynen, E.A., Villeneuve, D.L., Wehmas, L., and Ankley, G.T., Mid-Continent Ecology Division, U.S. Environmental Protection Agency, Duluth, MN, USA.

Triclocarban, commercially known as TCC, is a trichlorinated pesticide used extensively as an antimicrobial additive in personal care products. TCC is characterized as a high production volume chemical and recent monitoring programs have shown it is prevalent in aquatic environments. In mammalian systems TCC has little or no androgenic activity alone, but it amplifies androgen receptor-mediated transcriptional activity in vitro and in vivo, suggesting a new mechanism of endocrine action. To evaluate the effects of TCC in fish we used a short-term reproduction assay with the fathead minnow (*Pimephales promelas*) designed to detect chemicals with the potential to disrupt endocrine function controlled by estrogen- and androgen-mediated pathways. In addition to the basic reproduction assay, to specifically assess the ability of TCC to enhance an androgen-mediated response, a subset of fish were cotreated with the synthetic androgen 17β-trenbolone (TB), which masculinizes female fathead minnows, causing the development of cranial nuptial tubercles. Adult male and female fish were exposed for 21 d to nominal concentrations of TCC ranging from 1 to 10 ug/L alone, or in combination with 500 ng/L

TB. Fish exposed to 10 ug/L TCC exhibited signs of toxicity including decreased feeding after 2 weeks, and by the end of the test most were lethargic and several had died. Exposure to TCC reduced fecundity at a concentration of 5ug/L, but not at the low concentration of 1 ug/L. Bioconcentration factors averaged 370 and 2900 at 1 and 5 ug TCC/L, respectively, indicating the potential of the chemical to bioaccumulate. Cotreatment with TCC increased masculinization (i.e., number of tubercles) of the females caused by TB, suggesting that TCC can act as an androgen amplifier in fish as well as mammalian systems. Overall, the results of this study suggest that TCC impairs reproductive success and affects several aspects of endocrine function, and also exerts a synergistic effect on androgen-mediated processes in fish. Given the widespread use of TCC, and its persistence in the environment, further studies are warranted to assess potential ecological risk. *This abstract does not necessarily reflect EPA policy*.