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Published in:
2020 SOT Annual Meeting abstract

Publication date:
2020

Document Version
Publisher's PDF, also known as Version of record

[Link back to DTU Orbit](#)

Citation (APA):
Rosenmai, A. K., Ramhøj, L., Johansson, H., & Svingen, T. (2020). Sexually Dimorphic Thyroid Gene Expression Can Lead to Sex-Specific Responses to Thyroid-Disrupting Compounds. In *2020 SOT Annual Meeting abstract* (pp. 212-212). Article 1893 Society of Toxicology.

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Sexually Dimorphic Thyroid Gene Expression Can Lead to Sex-Specific Responses to Thyroid-Disrupting Compounds

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The thyroid hormone (TH) system is central to brain development and is susceptible to perturbation by environmental chemicals. Testing of chemicals for TH disruption is often done in animals, but to allow for the use of alternative test methods, mechanism of effects must be better described. Ex vivo thyroid culture is a tractable model, as it can be used both as an intermediate model system and for characterizing mechanisms-of-action. In this project, we aim to characterize sex differences in thyroid development and developmental windows of TH system vulnerability to chemical exposure to facilitate better design and interpretation of ex vivo thyroid culture. Initial studies using explanted rat fetal thyroids exposed for 72 and 120 h to 100 μ M propylthiouracil (PTU) revealed that i) key genes are dysregulated by PTU exposure and ii) the thyroids have sex-specific transcriptional profiles that give rise to sex-specific responses to chemical insult. Genes, including *Tshr* and *Nkx2-1*, displayed sexually dimorphic expression patterns after 72 h culture, a sex difference that disappeared after 120 h culture. After exposure to PTU, a thyroperoxidase (TPO) inhibitor, we observed compensations in both males and females. In males, these compensatory mechanisms were identified as upregulated *Nis* expression in response to PTU exposure, as well as a trend for upregulated *Tpo* and *Tg* expression with no effects on *Tshr*. In females, there was only a trend towards upregulated *Nis* expression, no effect on *Tpo* and *Tg*, and a downregulation of *Tshr*. These findings suggest sexually dimorphic effects in response to chemical exposure. These sex differences have implications for interpretation of thyroid disruption in animal studies and for further development of test methods for TH disruption. SOT 59th Annual Meeting and ToxExpo 2