A Newly Identified Role for Endocrine Disrupting Chemicals in Erectile Dysfunction

Andrew Pask[1]; Deidre Mattiske[1]; Sarah Marshall[2]; Sam Cripps[1]
1. School of BioSciences, University of Melbourne, Australia
2. Department of Obstetrics and Gynaecology, Monash University, Clayton, Australia

Erectile dysfunction is an extremely prevalent condition globally and has been estimated to more than double in the past thirty years. Genetic mutations or increased reporting alone cannot account for this trend, and thus environmental factors must play a major role in its aetiology. Endocrine Disrupting Chemicals (EDCs) are one likely candidate, given that development and function of the erectile tissues are hormonally dependent. Previously we have shown that exposure to estrogenic EDCs during mouse penis development impacted genes that may impact erection physiology during adulthood. Therefore, this study investigated a role for estrogenic EDCs in the aetiology of erectile dysfunction. Functional testing of the primary erectile tissue in mice was performed following chronic and acute estrogenic EDC exposures. Male mice chronically exposed to the potent estrogen diethylstilbestrol (DES) exhibit abnormal contractility of the erectile tissue, indicative of ED. The treatment did not affect systemic testosterone production yet significantly increased estrogen receptor α (*Esr1*) expression in the primary erectile tissues, suggesting EDCs directly impacted penis function. In response, we isolated erectile tissues from mice and briefly incubated them with DES or the phytoestrogen genistein. These acute-direct exposures similarly caused a significant reduction in erectile tissue contractility, again indicative of erectile dysfunction. Overall, these findings demonstrate a direct link between estrogenic EDCs and erectile dysfunction and show that both chronic and acute estrogenic exposures are likely risk factors for this condition.