Abstract

Foetal exposure to phthalates is known to adversely impact male reproductive development and function. Developmental anomalies of reproductive tract have been attributed to impaired testosterone synthesis. However, species differences in the ability to produce testosterone have been noted; e.g., following foetal exposure, abnormal clustering of Leydig cells or decreased production of testosterone that is manifested in rats does not occur in mice or humans. Nonetheless, other facets of testicular dysgenesis occur in both rats and mice as well as in some other species tested. We recently published a comprehensive evaluation of the foetal rat testis proteome, following in utero exposure to diethylhexyl phthalate (DEHP), which revealed changes in individual proteins that are known to be factors in cellular differentiation and migration or related to the capacity of the foetal Leydig cell to produce testosterone and fit a pathway network in which each is regulated directly or indirectly by oestradiol. Plasma oestradiol indeed was found to be elevated approximately twofold in 19-day-old DEHP-exposed foetal male rats. In this brief review, we discuss our new findings vis-à-vis 'oestrogen hypothesis' as a cause for testicular dysgenesis syndrome.