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Opinion Article

The Effect of Estrogen in the Development of Penis

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ABOUT THE STUDY

Hypospadias, a developmental abnormality of the penis, is one of the most frequent congenital abnormalities in humans. Its prevalence has dramatically grown in recent decades, which has been partly linked to our increased exposure to endocrine-disrupting substances. Penis growth is predominantly an androgen-driven process; however oestrogen and xenoestrogens have been shown to influence penis development in both humans and mice. Androgens, which promote male-specific extension and masculinization of the sexually indifferent Genital Tubercle (GT), are principally responsible for penis development in both mice and humans.

In men, when the GT grows, a urethral groove emerges on the ventral surface. The urethra is internalised by the strictly androgen-regulated process of urethral closure, which results in the urethra being central to the penis and glans and ending at its distal tip. Any interruption in androgen signalling during development might inhibit penis outgrowth and urethral closure, resulting in hypospadias, or urethral opening placement that is aberrant. Penis development defects are becoming more widespread, with hypospadias currently affecting up to 1 in every 125 live male newborns. In most developed nations, the prevalence of hypospadias has more than doubled in recent decades.

This is not caused due to improved reporting and is too quick to be explained by genetic alterations, suggesting that this illness has an environmental basis. It is generally known that medicines that inhibit androgens can result in hypospadias. Interestingly, oestrogen and estrogen-mimicking compounds affect penis development and urethral closure in both mice and humans, producing hypospadias. In the daily lives, people are exposed to a variety of substances that have an effect on our endocrine-signaling pathways. Bisphenol A, phthalates, and genistein are some of the most common Endocrine-Disrupting Chemicals (EDCs) that influence oestrogen signalling (xenoestrogens). EDCs are potential contributors to increased hypospadias levels, and new research has found a direct relationship between estrogenic EDC levels in foetal blood and the occurrence of hypospadias in people.

It's yet unclear how EDCs induce hypospadias. According to research, the effects of exogenous oestrogen are transmitted through the testis, where they can directly decrease androgen production from the Leydig cells. This lowers testosterone levels, resulting in hypospadias. However, both oestrogen receptors (ERs), ER (encoded by the ESR1 gene) and ER (encoded by the ESR2 gene), are widely expressed in the growing penis of rats, mice, and humans. Furthermore, aromatase, the enzyme that irreversibly converts testosterone to oestrogen, is found in the growing rat (30) and human penis, indicating the possibility of local oestrogen production and a function for oestrogen signalling in proper penile development.

CONCLUSION

Given the presence of ERs in the penis, a recent study hypothesised that the endogenous oestrogen could play an important role in normal penis development and that exogenous oestrogen or xenoestrogens cause hypospadias by directly affecting developmental processes in the penis rather than through suppressed testicular androgen output. It is crucial to understand the involvement of endogenous and exogenous oestrogen signalling in penis development. A previously unknown hypospadias phenotype in the ER Knock Out (ERKO) mouse is reported, suggesting that endogenous oestrogen signalling is required for patterning and closure of the distal urethra.

Understanding hypospadias requires defining how hormones influence penis patterning and development. The higher rates of delamination in the estrogen-deficient penis generated hypospadias. This not only provides crucial insights into the formation of hypospadias, but also a complete description of the delamination events that occur throughout mouse penis development.

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