Steroid 5-Reductase Isoenzymes from Men and Women

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Introduction

In mammals, there are two steroid 5-reductase isoenzymes, type 1 and type 2, and new research indicates that each has a distinct physiological role. The theory stated in this article is that the type 1 gene defines a female isoenzyme, whereas the type 2 gene determines a male isoenzyme. A male isoenzyme is specified by the gene. The following observations led to this hypothesis. First, in mice, a mutation in the 5-reductase type 1 gene reduces female fertility. Fecundity and parturition are inhibited, while male reproduction is unaffected. Second, in mice and men, mutations in the 5-reductase type 2 gene impede normal virilization but do not prevent virilization. In females, it does not affect the development of reproductive function. Dihydrotestosterone is a powerful androgen metabolite that is produced from testosterone by the action of 5areductase isoenzymes. Mutations in the type 2 isoenzyme induce a 46, XY sex development condition known as 5a-reductase type 2 deficiency, which was first discovered forty years ago. There have been several reports of mutations in the encoding gene in various ethnic groups. Female external genitalia is prevalent in afflicted 46, XY people, however, Mullerian ducts regress and the internal urogenital tract is male. Most afflicted males are reared as girls, but virilization happens throughout puberty, and male social sex emerges often thereafter. Fertility can be accomplished in some afflicted guys using assisted reproduction procedures, and people with male social sex report a more satisfying sex life and quality of life than affected males [1].

Dihydrotestosterone is a potent androgen metabolite generated from testosterone by the action of 5areductase isoenzymes. Mutations in the type 2 isoenzyme cause a 46, XY sex development disorder known as 5a-reductase type 2 deficiency, which was found forty years ago. There have been multiple reports of mutations in the encoding gene in diverse ethnic groups. Female external genitalia is more common in 46, XY persons, however, Mullerian ducts regress and the internal urogenital tract is male. Most affected boys are raised as females, although virilization occurs throughout adolescence, and male social sex appears often thereafter. Fertility can be achieved in some affected men by assisted reproduction treatments, and men with male social sex have a more happy sex life and a higher quality of life than women.

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The majority of affected boys are raised as females, however, virilization occurs until adolescence and male social sex arises often after that. Fertility can be restored in some affected guys by assisted reproduction treatments, and men with male social sex enjoy a better sexual life and a higher quality of life.

The development of the male phenotype is governed by three hormones. Fetal testes produce two hormones: antimullerian hormone (AMH) and testosterone. AMH is a peptide hormone released by Sertoli cells and aids in the regression of the Mullerian ducts, therefore, blocking the formation of female internal genitalia (uterus) and fallopian tubes) in the male fetus. Testosterone is the primary androgen generated by the testes, and it stimulates male fertility. development via two routes: first, the steroid works directly; second, the steroid acts indirectly; and finally, the steroid acts indirectly to transform the Wolffian ducts into the epididymis, vasa deferentia, Seminal vesicles, and ejaculatory ducts Second, testosterone is a hormone. The third hormone is a prohormone for the synthesis of dihydrotestosterone of male virilization [4].

Three hormones regulate the development of the masculine phenotypic. The fetal testes produce two hormones: antimullerian hormone (AMH) and testosterone. AMH is a peptide hormone released by the pituitary gland. Sertoli cells, as well as the regression of the Mullerian ducts, are provided. As a result, the development of female internal genitalia (uterus) is prevented as well as the fallopian tubes) in the male fetus, Testosterone is the male hormone. It is the primary androgen generated by the testes, and it stimulates male sexual development. Development via two routes: first, the steroid works directly; second, the steroid acts indirectly; and third, the steroid acts indirectly to transform the Wolffian ducts into the epididymis, vasa deferentia, and vasa recta. Seminal vesicles and ejaculatory ducts are two types of seminal vesicles. Second, testosterone is a male hormone prohormone for the production of dihydrotestosterone, the third hormone .virilization of males.

Steroid 5-reductase catalyzes the breakdown of a double bond between carbons 4 and 5 in several steroid substrates. Early research revealed that the enzyme was multipurpose, decreasing male sex hormones like testosterone to make the more powerful androgen, dihydrotestosterone, and female sex hormones like progesterone to produce the inert progestin, 5-pregnan-3,20-dione. The fact that 5-reductase can both activate and deactivate steroids suggests that the enzyme has various endocrine functions. Cloning investigations in numerous mammalian species revealed two distinct but related genes encoding distinct isoenzymes.

Two isoenzymes of the 5a-reductase catalyze the conversion of testosterone to dihydrotestosterone. Within five minutes of radioactive testosterone injection to intact male rats, the primary radioactive steroid recovered in prostate nuclei and the principal radioactive steroid coupled to the androgen receptor is dihydrotestosterone rather than testosterone. Dihydrotestosterone promotes prostate growth and midline fusion, as well as elongation and expansion of the urogenital tubercle and urogenital folds. All activities occur in the formation of the penis and scrotum.

The 5a-reductase isoenzymes

The 5a-reductase isoenzymes are highly selective for steroids with a D4,3-keto structure, such as progesterone, 17ahydroxyprogesterone, and testosterone. Isoenzyme 1 (encoded by the SRD5A1 gene) and isoenzyme 2 (encoded by the SRD5A2 gene) were separate genes that encode two distinct 5a-reductases (encoded by the SRD5A2 gene). Both genes include five exons and four introns and encode proteins with significant amino acid sequence similarity (about 60%), indicating the potential of a common ancestor throughout evolution. The human 5a-reductase-2

gene (SRD5A2) encodes a 254 amino acid protein and is found on the short arm of chromosome 2 at band 23. This protein prefers progesterone over testosterone. This gene is expressed in foetal organs such as the genital tract, the prostate, the epididymis, and the seminal vesicle. The 5a-reductase-1 gene (SRD5A1) is located in band 15 on the short arm of chromosome 5 and encodes a 259 amino acid protein type 1 5a-reductase This gene is expressed in embryonic tissues, although at low levels, and at birth is expressed in nongenital skin and liver SRD5A1 expression is increased in adults seen in the brain, liver, and nongenital skin [8]. The function of 5areductase 1 is unclear. In the prostate (epithelial and epithelial cells), Both 5a-reductase genes are expressed in stroma cells. The 5a-reductase type 2 is prevalent in cells [5].

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