Genetic Steroid Disorders: Chapter 4B. Marsupial Pathway in Humans

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Following development of the fetal bipotential gonad into a testis, male genital differentiation requires testicular androgens. Fetal Leydig cells produce testosterone that is converted to dihydrotestosterone in genital skin, resulting in labioscrotal fusion. An alternative “backdoor” pathway of dihydrotestosterone synthesis that bypasses testosterone has been described in marsupials, but its relevance to human biology has been uncertain. The classic and backdoor pathways share many enzymes, but a 3α-reductase, AKR1C2, is unique to the backdoor pathway. Human AKR1C2 mutations cause disordered sexual differentiation, establishing that both pathways are required for normal human male genital development. These observations show that fetal dihydrotestosterone acts both hormonally and as a paracrine factor, substantially revising the classic paradigm for fetal male sexual development.

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