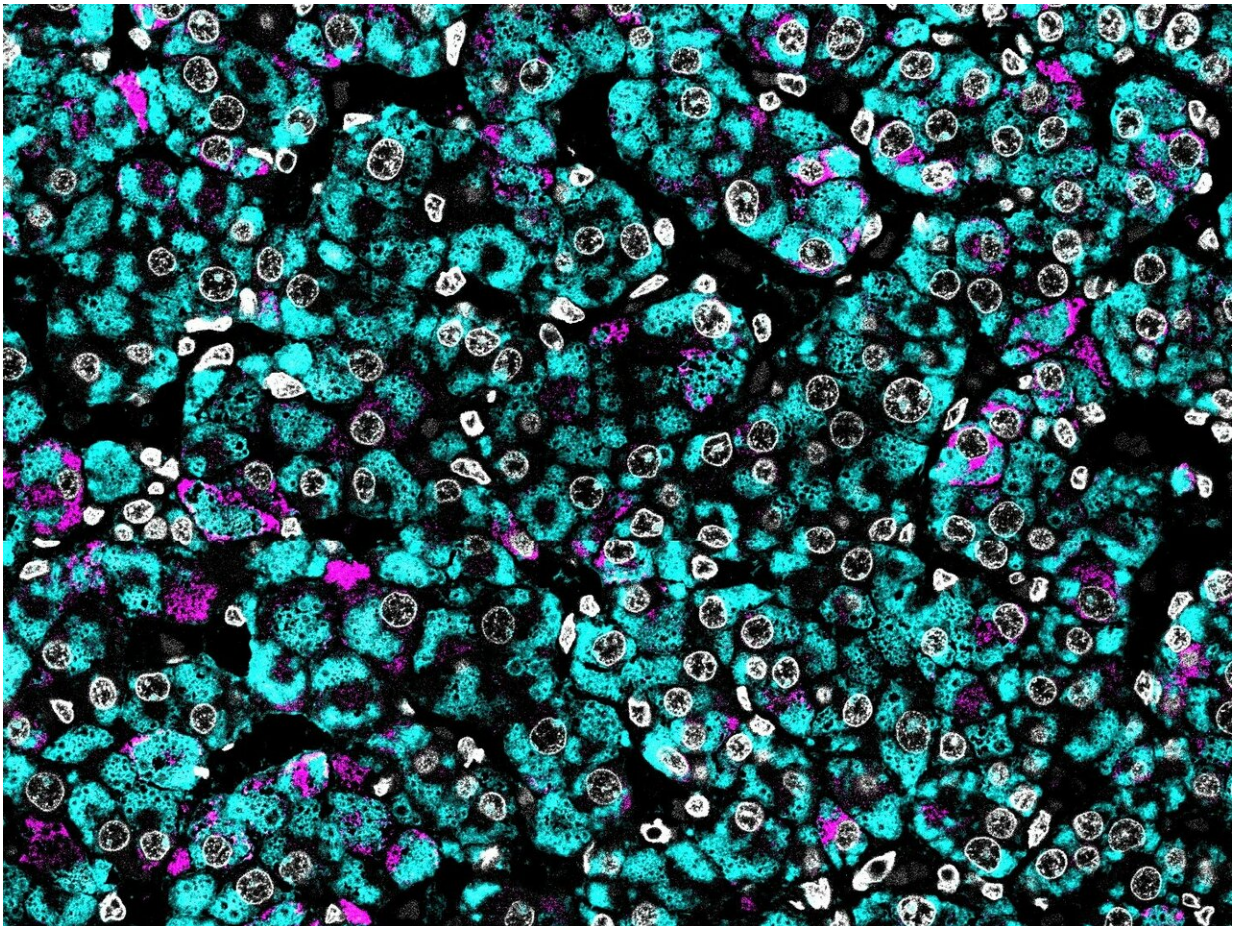


# Penis development needs more than just testes and testosterone

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An immunofluorescence image of a human fetal adrenal gland showing the steroidogenic cells. Credit: Zoe Johnston

Proper development of the fetal penis requires not just testosterone from the testes, but a second hormone produced by other tissues, including the placenta, according to a new study publishing February 14 in the open-access journal *PLOS Biology* from Paul Fowler of the University of Aberdeen, Michelle Bellingham of the University of Glasgow, and colleagues in the UK, France and Sweden. The results reveal a previously unknown pathway of masculinization of the external genitals, and may explain why placental dysfunction is associated with disorders of male genital development.

During development of the male fetus, the testes release [testosterone](#), a [steroid hormone](#) which is converted to 5 $\alpha$ -dihydrotestosterone (DHT) by the genital tubercle, helping to ensure that this primordial structure develops into a penis, rather than into the female clitoris. Recently, penis development was shown to also depend on a second process, called the alternative or "backdoor" [pathway](#), which also ends in the production of DHT but doesn't depend on the production of testosterone by the testes. However, the details of this backdoor pathway, including the source of the DHT precursor, have been unclear.

To learn more about this pathway, the authors used mass-spectrometry to measure levels of different steroids in fetal plasma and [tissue](#) during the [second trimester](#), when the most critical steps in penis development occur. They also analyzed [gene expression levels](#) in various tissues of key enzymes known to be involved in hormone synthesis.

They found that androsterone, a steroid from the backdoor pathway, which can be converted to DHT, was the principal androgen in the male fetal circulation, and that levels of both androsterone and testosterone were lower in the female fetal circulation. They also found that enzymes needed for the backdoor pathway were present primarily in non-gonadal tissue, including the liver and the placenta.

Since androsterone can be made from progesterone, the authors suggest that placental progesterone or related compounds are the likely source of androsterone in the backdoor pathway. While it remains unclear why a sex difference in fetal androsterone levels exists, the authors found high expression in the male genital tubercle of enzymes required to convert androsterone to DHT. The male genital tubercle appears, therefore to be able to convert both testosterone and androsterone into DHT.

"Our results demonstrate that masculinization of the male fetus depends not only on the testes, but also on other tissues, especially the placenta" Fowler and Bellingham said. "They also suggest an explanation for why disorders of placental insufficiency can lead to hypospadias and other abnormalities of growth of the male external genitalia."

**More information:** O'Shaughnessy PJ, Antignac JP, Le Bizec B, Morvan M-L, Svechnikov K, Söder O, et al. (2019) Alternative (backdoor) androgen production and masculinization in the human fetus. *PLoS Biol* 17(2): e3000002. [doi.org/10.1371/journal.pbio.3000002](https://doi.org/10.1371/journal.pbio.3000002)

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