# Oligozoospermia with normal fertility in male mice lacking the androgen receptor in testis peritubular myoid cells

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摘要

**Abstract**

Androgens and the androgen receptor (AR) play important roles in the testes. Previously we have shown that male total AR knockout (T-AR(-/y)) mice revealed incomplete germ cell development and lowered serum testosterone levels, which resulted in azoospermia and infertility. However, the consequences of AR loss in particular types of testicular cells remain unclear. Using a Cre-loxP conditional knockout strategy, we generated a tissue-selective knockout mouse with the AR gene deleted in testis peritubular myoid cells (PM-AR(-/y)). Phenotype analyses showed that PM-AR(-/y) mice were indistinguishable from WT AR (AR(+/y)) mice with the exception of smaller testes size. PM-AR(-/y) mice have serum testosterone concentrations comparable with AR(+/y) mice. PM-AR(-/y) mice have oligozoospermia in the epididymis; however, fertility was normal. Although normal germ cell distribution ratio was found, total germ cell number decreased in PM-AR(-/y) mice. Further mechanistic studies demonstrated that PM-AR(-/y) mice have defects in the expression of Sertoli cells' functional marker genes such as tran-ferrin, epidermal fatty acid-binding protein, androgen-binding protein, and other junction genes including occludin, testin, nectin, zyxin, vinculin, laminin gamma 3, gelsolin, connection43, and N-cadherin. Furthermore, there were defects in peritubular myoid cell contractility-related genes such as endothelin-1, endothelin receptor A and B, adrenomedullin, adrenomedullin receptor, and vasopressin receptor 1 a. Together, our PM-AR(-/y) mice provide in vivo evidence for the requirement of functional AR in peritubular myoid cells

to maintain normal Sertoli cells function and peritubular myoid cell contractility, thus ensuring normal spermatogenesis and sperm output.

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