Expression of the androgen receptor in the testis of mice with a Sertoli cell specific knock-out of the connexin 43 gene (SCCx43KO−/−)

Katarzyna Chojnacka, Ralph Brehm, Karola Weider, Anna Hejmej, Marta Lydka, Ilona Kopena-Sobota, Barbara Bilinska

Abstract

Gap junctions are intercellular channels that connect the cytoplasm of adjacent cells, allowing the passage of small molecules (<1 kDa) and thereby the regulation of many different processes. In the male gonad, the most abundant protein that builds gap junctions is connexin 43 (Cx43, GJA1). Specific knock-out of Sertoli cells (SCCx43KO−/−) results in an impaired spermatogenesis up to the Sertoli cell only syndrome. The aim of this study was to compare the testicular expression pattern of the androgen receptor (AR) in wild type (WT) and SCCx43KO−/− mice. In both WT and SCCx43KO−/− testes, the AR staining was restricted to the nuclei of Sertoli, Leydig, and peritubular cells. However, the staining intensity varied between control and mutant mice. In the latter, the AR expression depended on the level of the seminiferous tubule impairment. In tubules with qualitatively normal spermatogenesis, the AR protein expression was similar to that observed in the testes of WT mice. Conversely, seminiferous tubules with an arrest of spermatogenesis at the level of spermatogonial or spermatocyte phase expressed the AR at a lower intensity. In Sertoli cell only tubules (no germ cells in the tubules), the AR immunoreaction was mainly weak or undetectable. Moreover, AR staining was lower in Sertoli and Leydig cells (p < 0.001 and p < 0.05, respectively) of SCCx43KO−/− mice compared to WT mice, as revealed by a semiquantitative analysis. In conclusion, the deletion of Cx43 leads to a partial disruption of the AR signaling pathway, indicating a possible reason for the observed impaired spermatogenesis.

Keywords

Androgen receptor; Testis; Connexin 43 gene; Sertoli cells; Knockout mice; SCCx43KO−/−