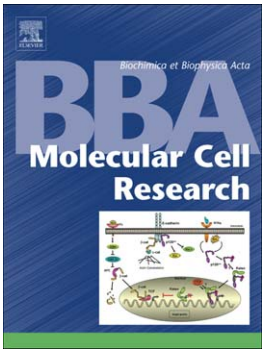

Actin related protein complex subunit 1b controls sperm release, barrier integrity and cell division during adult rat spermatogenesis

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Title: Actin related protein complex subunit 1b controls sperm release, barrier integrity and cell division during adult rat spermatogenesis

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Abstract

Actin remodeling is a vital process for signaling, movement and survival in all cells. In the testes, extensive actin reorganization occurs at spermatid-Sertoli cell junctions during sperm release (spermiation) and at inter Sertoli cell junctions during restructuring of the blood testis barrier (BTB). During spermiation, tubulobulbar complexes (TBCs), rich in branched actin networks, ensure recycling of spermatid-Sertoli cell junctional molecules. Similar recycling occurs during BTB restructuring around the same time as spermiation occurs. Actin related protein 2/3 complex is an essential actin nucleation and branching protein. One of its subunits, Arpc1b, was earlier found to be down-regulated in an estrogen-induced rat model of spermiation failure. Also, Arpc1b was found to be estrogen responsive through estrogen receptor beta in seminiferous tubule culture. Here, knockdown of Arpc1b by siRNA in adult rat testis led to defects in spermiation caused by failure in TBC formation. Knockdown also compromised BTB integrity and caused polarity defects of mature spermatids. Apart from these effects pertaining to Sertoli cells, Arpc1b reduction perturbed ability of germ cells to enter G2/M phase thus hindering cell division. In summary, Arpc1b, an estrogen responsive gene, is a regulator of spermiation, mature spermatid polarity, BTB integrity and cell division during adult spermatogenesis.

Highlights

- Arpc1b of the Arp2/3 complex is an estrogen-regulated gene in the testis
- Intratesticular Arpc1b knockdown led to spermiation and blood testis barrier defects
- These defects are due to reduced recruitment of functional Arp2/3 complex to actin
- Arpc1b knockdown also caused reduction in spermatocyte and round spermatid numbers
- Germ cell loss was caused by apoptosis and block in cell division progression

Keywords: Actin, Arpc1b, spermiation, blood testis barrier, cell division, estrogen