

Inhibition of p38MAPK signal pathway alleviates radiation-induced testicular damage

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Abstract

Background and Purpose: How to prevent the damage of ionizing radiation to testis has become an urgent problem to be solved. The present aim is to investigate whether inhibition of p38MAPK signaling can alleviate radiation-induced testicular damage. **Experimental Approaches:** HE staining was used to measure the morphological changes of epididymis and testis. Immunohistochemistry staining was used to assess the expression of PLZF, SOX9, p-p38MAPK. RNA-Seq was used to profile gene expression. The expression of Mapk14, Atf2, Ddit3 and Ap1m1 genes was detected by qPCR. Western blotting was used to detect the protein expression of p38MAPK and p-p38MAPK. **Key Results:** There was a dose-response relationship between testicular injury and ionizing radiation. The sperm quality was significantly decreased at 6 and 8 weeks after 6Gy of radiation. Radiation led to the decrease of PLZF+ cells and increase of SOX9+ cells in testis. RNA-Seq data showed radiation induced 969 genes changed in the testis. The expression of genes related to p38MAPK signal pathway enriched by GO was significantly increased by qPCR. The expression of p-p38MAPK in testis was significantly increased at 4 weeks after irradiation. SB203580 treatment increased numbers of spermatozoa, the area and diameter of seminiferous tubules and numbers of SOX9+ cells in irradiated mice, which were consistent with the increased sperm movement rate and density under radiation with SB203580 administration. **Conclusions and Implications:** Ionizing radiation significantly changes testicular gene expression, in which p38MAPK signal pathway is activated. p38MAPK inhibitor SB203580 partially alleviates the testicular damage caused by radiation and accelerate the recovery of sperm quality.

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