THE EFFECT OF GLUCOCORTICOIDS ON THE HISTOLOGY OF RAT GENITAL ORGANS.

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Abstract: Glucocorticoids (GCs) exert profound effects on the structure and function of rat genital organs due to the broad distribution of glucocorticoid receptors within reproductive tissues. This study summarizes the histological alterations induced by endogenous stress-related or exogenously administered GCs in both male and female rats. In males, GCs cause degeneration of seminiferous tubules, thinning of the germinal epithelium, spermatogenic arrest, and apoptosis of germ and Leydig cells, ultimately impairing steroidogenesis and reducing fertility. In females, GCs promote follicular atresia, apoptosis of granulosa and theca cells, degeneration of oocytes, and thinning of the endometrial lining, leading to disrupted folliculogenesis and impaired reproductive capacity. These morphological changes are mediated through glucocorticoid receptor–dependent genomic pathways, suppression of gonadotropin secretion, oxidative stress, and activation of apoptotic cascades. Overall, glucocorticoids induce marked histopathological alterations in rat reproductive organs, highlighting their significant role in stress-related reproductive dysfunction.

Keywords: Glucocorticoids; Rat genital organs; Histology; Testis; Ovary; Spermatogenesis; Folliculogenesis; Leydig cells; Apoptosis; Stress hormones; Reproductive system; Steroidogenesis; Endometrium; Gonadal structure.

Introduction: Glucocorticoids (GCs) are stress-related steroid hormones that play an important role in maintaining physiological balance, but excessive or prolonged exposure can negatively affect the reproductive system. Because glucocorticoid receptors are widely expressed in rat gonadal tissues, these organs are highly sensitive to GC-induced structural changes. In males, GCs can disrupt spermatogenesis and damage germ and Leydig cells, while in females they may promote follicular atresia and impair oocyte quality. Understanding how glucocorticoids alter the histology of rat genital organs is essential for clarifying the mechanisms of stress-related reproductive dysfunction.

Methods and materials: Twenty-four adult Wistar rats (12 males, 12 females; 180–220 g) were used. They were kept under standard conditions (22 ± 2°C, 12/12 h light–dark cycle) with free access to food and water. All procedures followed institutional ethical guidelines.

Rats were randomly divided into two groups:

1.Control – no treatment.

2.Glucocorticoid-treated – received dexamethasone 1 mg/kg intraperitoneally daily for 14 days.

Materials for the study were taken from the rats and then stained with eosin and hemotaxillin. Afterwards, a histological analysis of the material was carried out under a microscope.

Results:

Male Genital Organs

Testes

Histological examination of the testes in glucocorticoid-treated rats revealed significant alterations compared to controls. The seminiferous tubules showed partial degeneration with disorganization of the germinal epithelium. The epithelium was thinner, and the lumen appeared reduced in size. Spermatogenic arrest was observed, particularly at the spermatocyte and spermatid stages, leading to a decreased number of mature spermatozoa. Increased apoptotic cells were detected within the seminiferous epithelium, confirmed by pyknotic nuclei and cytoplasmic shrinkage. Leydig cells were reduced in number, exhibited cytoplasmic vacuolization, and in some areas showed signs of degeneration. Sertoli cells also displayed vacuolation and disrupted cell junctions, indicating impaired support for germ cell development.

Epididymis

The epididymal tubules of treated rats demonstrated thinning of the pseudostratified columnar epithelium and decreased stereocilia height. The lumen contained fewer spermatozoa, and epithelial cells showed signs of degeneration, including cytoplasmic vacuolation and nuclear pyknosis. No inflammatory infiltration was observed.

Female Genital Organs

Ovaries

Ovarian histology in glucocorticoid-treated rats showed an increased number of atretic follicles compared to controls. The granulosa and theca cells of growing and antral follicles exhibited cytoplasmic shrinkage, nuclear condensation, and detachment from the basal membrane, indicating apoptosis. Oocyte degeneration was evident, with shrinkage, vacuolation, and irregular zona pellucida. The number of corpora lutea was reduced, suggesting impaired ovulation. Stromal tissue appeared less vascularized and partially fibrotic in some regions.

Uterus

The endometrium of glucocorticoid-treated rats was markedly thinner, with reduced stromal cell density. Uterine glands were smaller and fewer in number, and epithelial cells showed decreased mitotic activity. The myometrium did not show significant changes, but the overall uterine architecture appeared less robust compared to controls.

Conclusion

Glucocorticoid exposure induces significant histopathological changes in the genital organs of rats. In males, it leads to degeneration of seminiferous tubules, apoptosis of germ and Leydig cells, and disruption of spermatogenesis. In females, glucocorticoids promote follicular atresia, granulosa and theca cell apoptosis, oocyte degeneration, and thinning of the endometrium. These structural alterations suggest that both stress-related and pharmacological elevations of glucocorticoids can impair reproductive function. The findings highlight the importance of understanding glucocorticoid-mediated effects on reproductive tissues and provide a basis for further studies on the mechanisms underlying stress- and therapy-induced reproductive dysfunction.

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