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JOURNAL ARTICLE

Suppression and recovery of spermatogenesis following spinal cord injury in the rat

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Recently, we reported that changes in spermatogenesis in adult rats during acute phase (within 2 weeks) of spinal cord injury (SCI) were associated with a suppression of pituitary-testis hormone axis, and these effects mimic those that occur after hormone deprivation. In this study, we examined the long-term (>4 weeks) effects of SCI on spermatogenesis and its recovery. Results of this study reveal that while serum follicle stimulating hormone, luteinizing hormone, and testosterone levels in SCI rats recovered within 1 month after the injury, their spermatogenesis continued to regress. By 3 months, spermatogenesis in 70% of SCI rats has totally regressed, characterized by the absence of proliferating spermatogonia; these effects could not be prevented by an otherwise effective regimen of testosterone treatment. Sertoli cells in the regressed seminiferous tubules exhibited unusual behavior, characterized by the formation of multiple cell layers and/or aggregates that extended into the tubular lumen. Active spermatogenesis was observed in nine of the 19 SCI rats by 6 months, seven of which had complete spermatogenesis, but with persisting abnormalities. These results demonstrate that SCI results in total, but reversible, regression of spermatogenesis. Failure to prevent such effects by an otherwise effective exogenous testosterone regimen suggests that non-endocrine factors are involved in the SCI effects on spermatogenesis. The unusual Sertoli cell localization in the regressed testes may have been triggered by the loss of proliferating spermatogonia and may be involved in subsequent spermatogenic recovery.

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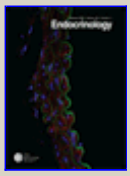

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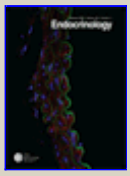

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