CHRONIC SPINAL CORD INJURY CAUSES UP-REGULATION OF SEROTONIN (5-HT) 2A AND 2C RECEPTORS IN LUMBOSACRAL CORD MOTONEURONS

Hypothesis / aims of study
Spinal cord injury can cause voiding dysfunction and intravenous administration of the 5-HT2A/2C receptor agonist, DOI has been demonstrated to improve urinary function in spinal cord injury rats, while this mechanism has not been well studied. So our objective was to discuss whether the main mechanism ascribes to serotonin 2A and 2C receptors up-regulation in lumbosacral cord motoneurons after chronic spinal cord injury.

Study design, materials and methods
Female Sprague-Dawley rats were used, which were divided into two groups (Spinal cord injury group VS Normal control group). Under urethane anesthesia, Cystometrogram was used to examine the variation of urodynamic parameters before and after successively intrathecal administration of various doses DOI in lumbosacral cord. Additionally, the changes of serotonin 2A and 2C receptors in lumbosacral cord were investigated by immunohistochemical staining section and western blot.

Results
Compared to controls, spinal cord injured rats had higher bladder capacity and post-void residual urine volume, and lower voiding efficiency. DOI could improve voiding efficiency via affecting external urethral sphincter activity after spinal cord injury. Furthermore, Immunohistochemical staining section and western blot showed that serotonin 2A and 2C receptors were up-regulation in lumbosacral cord motoneurons after chronic spinal cord injury.

Interpretation of results
DOI can improve voiding efficiency, and this may be due to serotonin 2A and 2C receptors up-regulation in lumbosacral cord motoneurons in controlling external urethral sphincter activity after chronic spinal cord injury.

Concluding message
Chronic spinal cord injury causes up-regulation of serotonin (5-HT) 2A and 2C receptors in lumbosacral cord motoneurons

References

Disclosures
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