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Título	Sympathetic hypoactivity leads to hypocontractility of the corpus cavernosum in sickle cell mice: a mechanism contributing to priapism
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Resumo	Priapism, a prevalent complication in sickle cell disease (SCD) patients, manifests as prolonged and painful erections unrelated to sexual arousal. The detailed mechanisms contributing to this condition, especially regarding sympathetic function in the corpus cavernosum that maintains penile flaccidity, remain to be elucidated. In this study, it was hypothesized that the pathways of the sympathetic nervous system would be down-regulated, thereby contributing to the development of ischemic priapism in sickle cell disease. This study aimed to investigate the contractions induced by stimulation of sympathetic terminals and the expression of tyrosine hydroxylase in the corpora cavernosa of Berkeley SCD mice. C57BL/6 mice (wild-type, WT) and Berkeley SCD mice were used. A total of 22 mice were used in this study, with 11 allocated to the WT group and 11 to the SCD group. Mice corpus cavernosum was dissected free and mounted in 7-mL organ baths containing Krebs solution. Noradrenergic contractions were obtained using electrical-field stimulation (4–32 Hz) in corpus cavernosum strips from WT and SCD mice. Measurements of tyrosine hydroxylase phosphorylated at Ser-31 and total tyrosine hydroxylase protein expressions in cavernosal tissues were also measured by western blot. The neurogenic contractions were significantly lower ($P < 0.05$) in the SCD group compared to WT group at all tested frequencies. The protein expression of both total tyrosine hydroxylase and tyrosine hydroxylase phosphorylated at Ser-31 was significantly decreased by approximately 46.28% ($P = 0.01$) and 55.32% ($P = 0.03$) in cavernosal tissues from the SCD group compared to the control group, respectively. In conclusion, sympathetic hypoactivity characterized by the downregulation of tyrosine hydroxylase contributes to the hypocontractility of the corpus cavernosum in Berkeley SCD mice. This suggests an impairment in the mechanism responsible for maintaining penile flaccidity, potentially predisposing to erections without sexual stimulation, similar to those observed in ischemic priapism. Pharmacological treatments aiming to restore sympathetic tone in the penis might hold promise for addressing ischemic priapism in SCD.
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