Abstract

Comparative studies using reptiles as experimental animals in pain research could expand our knowledge on the evolution and adaptation of pain mechanisms. Currently, there are no data reported on the involvement of voltage-gated sodium ion channels on nociception in reptiles. The aim of this study was to investigate the involvement of Nav1.3, Nav1.7, and Nav1.8 ion channels in nociception in Speke's hinge-back tortoise. ICA 121341 (selective blocker for Nav1.1/Nav1.3), NAV 26 (selective blocker for Nav1.7), and A803467 (selective blocker for Nav1.8) were used to investigate the involvement of Nav1.3, Nav1.7, and Nav1.8, respectively. The chemicals were administered intracoelomically thirty minutes before the start of nociceptive tests. ICA 121341 did not cause a significant decrease in the time spent in pain-related behavior in all the nociceptive tests. NAV 26 and A8034667 caused a statistically significant decrease in the mean time spent in pain-related behavior in the formalin and capsaicin tests. Only A803467 caused a statistically significant increase in the mean latency to pain-related behavior in the hot plate test. NAV 26 and A803467 had no observable side effects. In conclusion, Nav1.7 and Nav1.8 are involved in the processing of chemically induced inflammatory pain in Speke's hinge back tortoise. In addition, Nav1.8 are also significantly involved in the development of thermal-induced pain-related behavior in this species of reptile. However, our results do not support the involvement of Nav1.3 on the development of chemical or thermal induced pain-related behavior in the Speke's hinge back tortoise