Biochemical Pharmacology 82 (2011) 1250–1251

Contents lists available at ScienceDirect

Biochemical Pharmacology

journal homepage: www.elsevier.com/locate/biochempharm

Corrigendum

Corrigendum to "Jingzhaotoxin-II, a novel tarantula toxin preferentially targets rat cardiac sodium channel" [Biochem. Pharmacol. 76 (2008) 1716–1727]

Meichi Wang, Qingping Liu, Haiyong Luo, Jiang Li, Jianzhou Tang, Yucheng Xiao, Songping Liang *

Key Laboratory of Protein Chemistry and Developmental Biology of the Ministry of Education, College of Life Science, Hunan Normal University, Changsha 410081, PR China

The authors regret that legends and citations to Figs. 6–8 were published incorrectly. The third and fifth paragraph from the Discussion section and Figs. 6–8 with legends are reproduced correctly below.

The phylogeny of spider sodium channel toxins based on their amino acid sequences in Fig. 8 indicated that the toxins are divergent in two evolutionary routes, the toxins from the family Theraphosidae and Hexathelidae which has a large body size are clustered in the first group, whereas toxins from the family Agelenopsis which has a small body size are clustered into the second branch, suggesting that they derive from different ancestor. From the phylogeny, we deduce that JZTXs lie between the depressant toxins, HNTXs, HWTXs and the excitatory toxins δ-ACTXs, μ-agatoxins not only in sequence but also in function. HWTXs and HNTXs are depressant spider toxins through the peak current similar to TTX [31]. δ-Atracotoxins induce spontaneous repetitive firing and prolongation of action potentials resulting in neurotransmitter release from somatic and autonomic nerve endings [8]. The μ-agatoxins cause increased spontaneous release of neurotransmitter from pre-synaptic terminals and repetitive

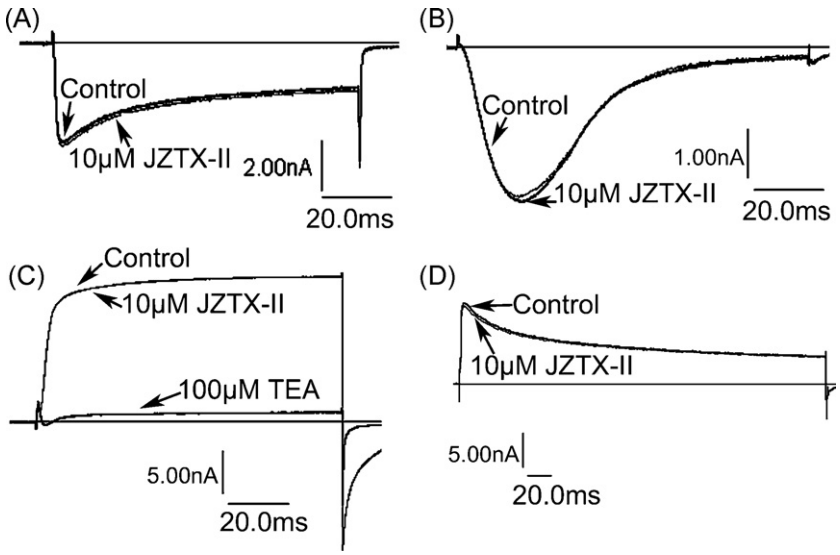

Fig. 6. Effects of JZTX-II to calcium channels and potassium channels. Ca^{2+} has been substituted by Ba^{2+}. (A) High-voltage activated Ca^{2+} currents had been induced by 100 ms depolarization step to 0 mV from a holding potential of −40 mV. 10 μM JZTX-II could not inhibit HVA Ca^{2+} currents (n = 5). (B) Low-voltage activated Ca^{2+} current were elicited by 100 ms depolarization step to −50 mV from a holding potential of −90 mV. 10 μM JZTX-II had no effect to T type calcium currents. (C) Delayed-rectifier potassium currents had been elicited by 100 ms depolarization step to 30 mV from a holding potential of −80 mV. 10 μM JZTX-II has no significant effect on the delayed-rectifier potassium currents (n = 5). (D) Transient outward potassium currents had been induced by 300 ms depolarization step to 30 mV from a holding potential of −80 mV. 10 μM JZTX-II had no effect on the transient outward potassium currents of DRG neurons (n = 5).

DOI of original article: 10.1016/j.bcp.2008.09.008
* Corresponding author. Tel.: +86 731 8872556; fax: +86 731 8861304.
E-mail address: liangsp@hunnu.edu.cn (S. Liang).

0006-2952/$ – see front matter © 2011 Published by Elsevier Inc.
doi:10.1016/j.bcp.2011.06.029

Source	subtype	DIV-S3	Extracellular loop	DIV-S4	
cardiac myocyte	rNav1.5	1604	SIVGTVLSDIIQ--KYFFSPTLFRVIRLAR		1631
DUM(cockroach)	para	1662	SILGLVLSDIIE--KYFVSPTLLRVVRVAK		1689
skeletal muscle	rNav1.4	1420	SIVGLALSDLIQ--KYFVSPTLFRVIRLAR		1447
hippocampal neuron	rNav1.1	1604	SIVGMFLAELIE--KYFVSPTLFRVIRLAR		1631
hippocampal neuron	rNav1.2	1605	SIVGMFLAELIE--KYFVSPTLFRVIRLAR		1632
hippocampal neuron	rNav1.3	1551	SIVGMFLAELIE--KYFVSPTLFRVIRLAR		1578
DRG	rNav1.7	1587	SIVGMFLAEMIE--KYFVSPTLFRVIRLAR		1614
hippocampal neuron	rNav1.6	1594	SIVGMFLADIIE--KYFVSPTLFRVIRLAR		1621
DRG	rNav1.8	1552	SIGSLLFSAIIKSLENYFSPTLFRVIRLAR		1681
DRG	rNav1.9	1420	SIISTLVSRLEDS-DISFPPTLFRVVRLAR		1448

Fig. 7. Analysis of amino acid sequences of receptor site 3 on different rat sodium channel subtypes. The sequences of various sodium channel subtypes are from NCBI databank. The number represents the superimposed position of amino acid of various sodium channels. The putative crucial residues between toxins and sodium channels are shaded in black.

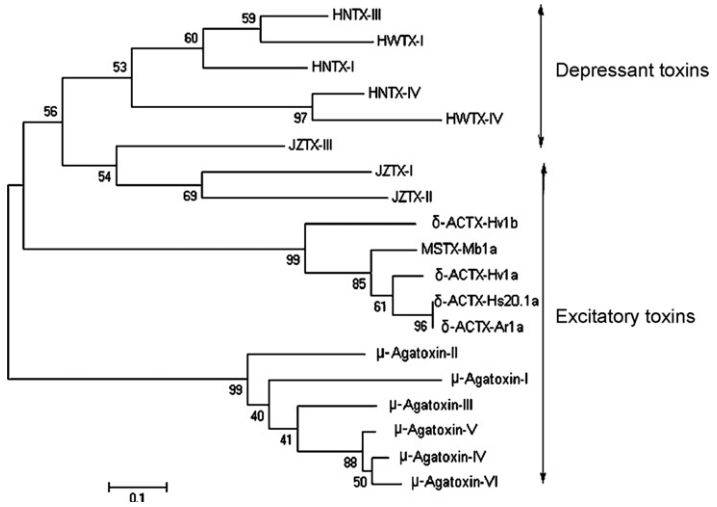

Fig. 8. Unrooted phylogenetic tree of spider sodium channel toxins. The numbers on the branches were the bootstrap percentages supporting a given partition. The main characteristic functions of these spider toxins are shown on the right.

action potentials in motor neurons [32]. JZTXs might give some important insight into toxin evolution. Intriguingly, JZTXs have much more diversity of function than the other three kinds of spider toxins in functions.

Rogers et al. found that Glu^{1613}, Glu^{1616}, Lys^{1617} and six uncharged residues of the IVS3–S4 loop in type rIIa sodium channel that significantly affected LqTx and/or ATX II affinity and constituted an important component of neurotoxin receptor site 3 [35]. Moreover, chimeric Na^+ channels in which amino acid residues at the extracellular end of segment IVS3 of the a subunit of cardiac Na^+ channels were substituted into the type IIa channel sequence had reduced affinity for α-scorpion toxin characteristic of cardiac Na^+ channels. Thus, JZTX-II had opposite affinity. Based on the sequences of various sodium channel subtypes in Fig. 7 and the properties of toxins altering channel gating, JZTX-II might structurally interact with receptor site 3 and its two charged residues (Lys^{13} and Glu^{12}, Fig. 2A) might provide key residues for binding the residues Asp^{1612} and Lys^{1616} in rNav1.5 but not Glu^{1613}, Glu^{1616}, Lys^{1617} in rNav1.2 and other subtypes in brain. Since the two residues are highly conserved in skeletal muscle sodium channel (rNav1.4) and rNav1.5 in Fig. 7, Nav1.4 might be also sensitive to JZTX-II. Sodium channel isoforms preferentially express in specific tissues. Felts and co-workers demonstrated that rNav1.1, rNav1.2, rNav1.3 and rNav1.6 are expressed predominantly in rat embryonic hippocampus neurons [24,36]. Intriguingly, our findings indicated that JZTX-II had no effect on sodium channel subtypes from embryonic hippocampus neurons. We concluded JZTX-II had high affinity to sodium channel subtype on cardiac myocytes but lower affinity to subtypes on DRG neurons and almost no affinity to that on brain. The different option between JZTX-II and LqTx might hint the different interactions between the sodium channel subtypes and toxins. JZTX-II represents a new interaction model.