Data in Brief 15 (2017) 567–572

Contents lists available at ScienceDirect

Data in Brief

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

Data Article

Data on the expression of leptin and leptin receptor in the dorsal root ganglion and spinal cord after preganglionic cervical root avulsion

Kai-Ting Chang [a,b], Yi-Lo Lin [b,c], Chi-Te Lin [b,d,e], May-Jywan Tsai [b], Wen-Cheng Huang [b,f,g,h], Yang-Hsin Shih [f], Yi-Yen Lee [h], Henrich Cheng [a,b,f,g,i], Ming-Chao Huang [b,e,h,j,*]

[a] Institute of Pharmacology, School of Medicine, National Yang-Ming University, Taipei, Taiwan
[b] Neural Regeneration Laboratory, Department of Neurosurgery, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan
[c] Graduate Institute of Veterinary Pathobiology, College of Veterinary Medicine, National Chung Hsing University, Taichung, Taiwan
[d] Department of Nursing, Central Taiwan University of Science and Technology, Taichung, Taiwan
[e] Basic Medical Education Center, Central Taiwan University of Science and Technology, Taichung, Taiwan
[f] Department of Neurosurgery, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan
[g] Department of Medicine, National Yang-Ming University, Taipei, Taiwan
[h] Division of Pediatric Neurosurgery, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan
[i] Center for Neural Regeneration, Neurological Institute, Taipei Veterans General Hospital, Taipei, Taiwan
[j] School of Medicine, Taipei Medical University, Taipei, Taiwan

ARTICLE INFO

Article history:
Received 16 August 2017
Received in revised form
11 September 2017
Accepted 3 October 2017
Available online 7 October 2017

Keyword:
Leptin
Leptin receptor
Microglia
Root avulsion

ABSTRACT

Leptin (Lep) is mainly, although not exclusively, secreted by adipocytes. In addition to regulating lipid metabolism, it is also a proinflammatory factor and involved in the development of neuropathic pain after peripheral nerve injuries (PNI) (Lim et al., 2009; Maeda et al., 2009) [1,2]. Leptin or its messenger ribonucleic acid expression has been found in various brain regions normally and in the dorsal horn after PNI (Lim et al., 2009; Ur et al., 2002; La Cava et al., 2004; White et al., 2004) [1,3–5]. However, the expression pattern of Lep and Leptin receptor (LepR) after preganglionic cervical root avulsion (PCRA) is still unknown. We provide data in this article related to Chang et al. (2017) [6]. Here, our data showed a profound Lep and LepR expression in the neurons of dorsal root

DOI of original article: http://dx.doi.org/10.1016/j.lfs.2017.08.016
* Correspondence to: Division of Pediatric Neurosurgery, Neurological Institute, Taipei Veterans General Hospital, No. 201, Shi-Pai Road, Section 2, Taipei 112, Taiwan.
E-mail address: mchuang@vghtpe.gov.tw (M.-C. Huang).

http://dx.doi.org/10.1016/j.dib.2017.10.005

ganglion (DRG) after PCRA. Moreover, the expression of Lep and LepR were also identified in significant portions of the neurons and microglia located in the dorsal horn. The roles of these increased expressions in the development of neuropathic pain after PCRA deserve further study.

Specification Table

Subject area	Neuroscience
More specific subject area	Leptin (Lep), Leptin receptor (LepR), Neural trauma, spinal cord, dorsal root ganglion (DRG)
Type of data	Image (immunofluorescence)
How data was acquired	Fluorescent microscope (Axioskop 2 Carl Zeiss, LLC, United States)
Data format	Raw
Experimental factors	DRG (Fig. 1) and dorsal horn (DH) of spinal cord (Figs. 2 and 3) was obtained from C57BL/6J mice after preganglionic cervical root avulsion (PCRA)
Experimental features	The DRG and DH were labeled using primary antibodies raised against leptin, its receptor, ionized calcium binding adaptor molecule 1 (Iba1, microglia), neuronal nuclei (NeuN, neuron), and glial fibrillary acidic protein (GFAP, astrocyte).
Data source location	Taipei, Taiwan.
Data accessibility	Data within this article

Value of the Data

- Generalized expression of Lep and LepR was noted 7 days after PCRA in DRG neurons. However, it has been reported that around 95% of DRG neurons die after PCRA [7]. The functions and/or effect of the leptin produced by neurons undergoing apoptosis remain to be clarified.
- Substantial intracellular expression of Lep was noted in the neurons of spinal cord after PCRA. It is known that extracellular leptin acts on its receptor located in the cell membrane of neurons. However, the roles of the intraneuronal Lep in the development of neuropathic pain have not been studied.
- Significant amount of Lep was noted in the dorsal horn of the spinal cord in neurons and microglia, but not in astrocytes. The control mechanism and the significance of this *de novo* production of Lep are of interest for future investigation.
- It is reported that LepR has been found to be increased after PNI in neurons, and to a lesser extent in astrocytes [1]. But after PCRA, we found that significant number of microglia has LepR expression. Does this difference be relevant to the different clinical presentation of the neuropathic pain after PNI and PCRA requires further research.

1. Data

We showed that both Lep and LepR were expressed in the DRG on day 7th after PCRA (Fig. 1). By using double immunolabeling for Lep/LepR and markers of specific cell populations, we found that after PCRA, there are increased expression of leptin in the dorsal horn [6]. This increased leptin