Data in Brief 15 (2017) 234–239

Contents lists available at ScienceDirect

Data in Brief

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

Data Article

Data on the effect of knockout of cytohesin-1 in myelination-related protein kinase signaling

Ruri Tsuneishi [a,1], Naoto Matsumoto [a,1], Misa Itaoka [a], Yuri Urai [a], Minami Kaneko [a], Natsumi Watanabe [a], Shou Takashima [c], Yoichi Seki [a], Takako Morimoto [a], Hiroyuki Sakagami [d], Yuki Miyamoto [a,b], Junji Yamauchi [a,b,2,*]

[a] *Laboratory of Molecular Neuroscience and Neurology, School of Life Sciences, Tokyo University of Pharmacy and Life Sciences, Hachioji, Tokyo 192-0355, Japan*
[b] *Department of Pharmacology, National Research Institute for Child Health and Development, Setagaya, Tokyo 157-8535, Japan*
[c] *Glycobiology Research Unit, The Noguchi Institute, Itabashi, Tokyo 173-0003, Japan*
[d] *Department of Anatomy, Kitasato University School of Medicine, Sagamihara, Kanagawa 252-0374, Japan*

ARTICLE INFO

Article history:
Received 7 July 2017
Accepted 5 September 2017

Keywords:
Cytohesin-1
Myelination
Peripheral nervous system
Protein kinase
Signaling

ABSTRACT

Cytohesin-1 is the guanine-nucleotide exchange factor of Arf6, a small GTPase of Arf family, and participates in cellular morphological changes. Knockout mice of cytohesin-1 exhibit decreased myelination of neuronal axons in the peripheral nervous system (PNS) "Phosphorylation of cytohesin-1 by Fyn is required for initiation of myelination and the extent of myelination during development (Yamauchi et al., 2012) [1]". Herein we provide the data regarding decreased phosphorylation levels of protein kinases involved in two major myelination-related kinase cascades in cytohesin-1 knockout mice.

* Correspondence to: Laboratory of Molecular Neuroscience and Neurology, School of Life Sciences, Tokyo University of Pharmacy and Life Sciences, 1432-1 Horinouchi, Hachioji, Tokyo 192-0392, Japan.
E-mail address: yamauchi@toyaku.ac.jp (J. Yamauchi).
[1] These authors equally contributed to this work.
[2] Contact address: Laboratory of Molecular Neuroscience and Neurology, School of Life Sciences, Tokyo University of Pharmacy and Life Sciences, 1432-1 Horinouchi, Hachioji, Tokyo 192-0392, Japan. Fax: 81 42 676 8841.

http://dx.doi.org/10.1016/j.dib.2017.09.024

Specifications table

Subject area	Biology
More specific subject area	Neurobiology, molecular and cellular neuroscience, developmental biology
Type of data	Figure
How data was acquired	Immunoblotting, polymerase chain reaction
Data format	Raw data, analyzed data
Experimental factors	Protein bands are scanned and densitometrically analyzed.
Experimental features	Immunoblot, agarose gel electrophoresis photograph
Data source location	Tokyo University of Pharmacy and Life Sciences, Tokyo, Japan
Data accessibility	Data is available with this article

Value of the data

- The data set is of value to the scientific community to need the information for signaling molecules controlling myelination.
- The data can provide data for common intracellular signaling cascades involved in myelination.
- The data can promote further research on signaling molecules controlling myelination *in vivo*.

1. Data

The exons 4 to 11 of the *cytohesin-1* gene were replaced with the *neo* gene (Fig. 1A). Deletion of these exons was confirmed by genomic polymerase chain reaction (PCR) and immunoblotting (Fig. 1, B and C). In immunoblotting with an antibody specific for phosphorylated Akt kinase (active Akt), decreased phosphorylation was observed in protein samples from knockout mouse nerves (Fig. 2, A and B). Akt is one of the central kinases controlling myelination [2–5]. Phosphorylation of kinases belonging to the mitogen-activated protein kinase (MAPK)/extracellular signal-regulated kinase (ERK) cascade was also decreased in knockout mouse nerves (Figs. 3–5). MAPK cascade in neuronal and glial cells is composed of ERK1/2, MEK1/2, and B-Raf and is also well known to control myelination [2–5].

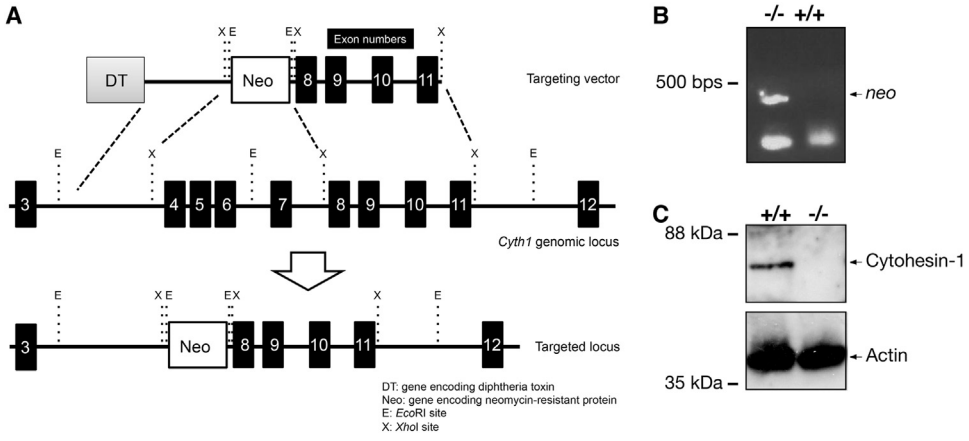

Fig. 1. Cytohesin-1 knockout mouse. (A) Schematic strategy for generating a cytohesin-1 knockout allele. (B) Genomic PCR of cytohesin-1 knockout mouse for the *neo* gene. (C) Immunoblotting of cytohesin-1 knockout mouse sciatic nerve tissue for cytohesin-1.