Data in Brief 15 (2017) 376–381

Contents lists available at ScienceDirect

Data in Brief

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

Data Article

Data demonstrating the role of peroxiredoxin 2 as important anti-oxidant system in lung homeostasis

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ARTICLE INFO

Article history:
Received 22 August 2017
Received in revised form
19 September 2017
Accepted 26 September 2017
Available online 30 September 2017

ABSTRACT

The data presented in this article are related to the research paper entitled "peroxiredoxin-2 plays a pivotal role as multimodal cytoprotector in the early phase of pulmonary hypertension" (Federti et al., 2017) [1]. Data show that the absence of peroxiredoxin-2 (Prx2) is associated with increased lung oxidation and pulmonary vascular endothelial dysfunction. Prx2$^{-/-}$ mice displayed activation of the redox-sensitive transcriptional factors, NF-kB and Nrf2, and increased expression of cytoprotective system such as heme-oxygenase-1 (HO-1). We also noted increased expression of both markers of vascular activation and extracellular matrix remodeling. The administration of the recombinant fusion

DOI of original article: http://dx.doi.org/10.1016/j.freeradbiomed.2017.08.004
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http://dx.doi.org/10.1016/j.dib.2017.09.062

protein PEP Prx2 reduced the activation of NF-kB and Nrf2 and was paralleled by a decrease in HO-1 and in vascular endothelial abnormal activation. Prolonged hypoxia was used to trigger pulmonary artery hypertension (PAH). Prx2$^{-/-}$ precociously developed PAH compared to wildtype animals.

Specifications Table

Subject area	*Health Sciences*
More specific subject area	*Oxidation, peroxiredoxin-2 and pulmonary artery hypertension*
Type of data	*Text file, Figures*
How data was acquired	Image Quant Las Mini 4000 Digital Imaging System (GE Healthcare Life Sciences). Densitometric analyses were performed using the ImageQuant TL software (GE Healthcare Life Sciences).
Data format	*Raw analyzed*
Experimental factors	*C57B6/2J as wildtype mice and Prx2$^{-/-}$ mice*
Experimental features	*Protein expression was analyzed by Western-blotting.* Oxidized proteins were revealed by the Oxyblot Protein Oxidation Detection Kit (EMD Millipore); MDA pulmonary levels were evaluated by Oxiselect MDA Immunoblot kit (GE Healthcare).
Data source location	*Dept. of Medicine, LURM, Policlinico GB Rossi, University of Verona and AOUI Verona; Verona; Italy*
Data accessibility	*Data are available with this article*

Value of the data

- Our data show that the absence of Prx2 is associated with increased lung oxidation and abnormal pulmonary vascular leakage.
- Treatment with fusion protein PEP Prx2 prevents the activation of redox related transcriptional factors and modulates anti-oxidant systems in both wildtype and Prx2$^{-/-}$ mice.
- PEP Prx2 significantly reduces protein oxidation in lung from exposed to prolonged hypoxia used to trigger pulmonary artery hypertension.

1. Data

Data show increased lung oxidation (Fig. 1A) and abnormal pulmonary vascular leakage in the absence of Prx2 (Fig. 1B). This was paralleled by the activation of redox-sensitive transcriptional factors NF-kB and Nrf2 in lung from Prx2$^{-/-}$ compared to wildtype animals (Fig. 2A). Indeed, in Prx2$^{-/-}$ we observed (i) increased expression of heme-oxygenase 1 (HO-1), a Nrf2 related cytoprotective system; (ii) markers of vascular endothelial activation such as endothelin-1 (ET-1) and vascular cell adhesion molecule -1 (VCAM-1) and (iii) marker of extracellular matrix remodeling as the platelet growth factor- B (PDGF-B) that has been recently function linked to the development of pulmonary artery hypertension (Fig. 2B). To verify the role of Prx2 as important anti-oxidant system in pulmonary homeostasis, we administrated the recombinant fusion protein PEP Prx2 at the dosage of 3 mg/Kg/d ip or vehicle for 4 weeks [1–3]. As shown in Fig. 2, PEP Prx2 significantly reduced both