Data in Brief 7 (2016) 730–734

Contents lists available at ScienceDirect

Data in Brief

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

Data article

Data on necrotic and apoptotic cell death in acute myocardial ischemia/reperfusion injury: the effects of CaMKII and angiotensin AT_1 receptor inhibition

Tomas Rajtik [a], Slavka Carnicka [b], Adrian Szobi [a], Zoltan Giricz [c], Jin O-Uchi [d], Veronika Hassova [a], Pavel Svec [a], Peter Ferdinandy [c,e], Tanya Ravingerova [b], Adriana Adameova [a,*]

[a] Department of Pharmacology and Toxicology, Faculty of Pharmacy, Comenius University in Bratislava, Slovak Republic
[b] Institute for Heart Research, Slovak Academy of Sciences & Centre of Excellence, SAS NOREG, Bratislava, Slovak Republic
[c] Cardiometabolic Research Group, Department of Pharmacology and Pharmacotherapy, Semmelweis University, Budapest, Hungary
[d] Department of Medicine, Jefferson Medical College, Thomas Jefferson University, Philadelphia, USA
[e] PharmaHungary Group, Szeged, Hungary

ARTICLE INFO

Article history:
Received 18 December 2015
Received in revised form
19 February 2016
Accepted 3 March 2016
Available online 10 March 2016

Keywords:
Myocardial ischemia/reperfusion injury
CaMKII inhibition

ABSTRACT

Content of particular proteins indicating cellular injury due to apoptosis and necrosis has been investigated in ischemic/reperfused (IR) hearts and ischemic/reperfused hearts treated with CaMKII inhibitor and/or AT_1 receptor inhibitor. This data article provides information in support of the original research article "Oxidative activation of CaMKIIδ in acute myocardial ischemia/reperfusion injury: a role of angiotensin AT_1 receptor-NOX2 signaling axis" [1].

DOI of original article: http://dx.doi.org/10.1016/j.ejphar.2015.12.024
* Correspondence to: Department of Pharmacology and Toxicology, Faculty of Pharmacy, Comenius University in Bratislava, Odbojarov 10, Bratislava 832 32, Slovak Republic. Tel.: +421 2 50117 366; fax: +421 2 50 117 100.
E-mail address: adameova@fpharm.uniba.sk (A. Adameova).

http://dx.doi.org/10.1016/j.dib.2016.03.017

AT_1 receptor blockade
Apoptosis
Necrosis

Specifications Table

Subject area	*Biology*
More specific subject area	*Myocardial ischemia/reperfusion injury; Cellular injury; Protein kinase*
Type of data	*Figures, Text*
How data was acquired	*Immunoblotting, Chemiluminescence*
Data format	*Raw, Analyzed*
Experimental factors	*SDS-PAGE, Western blotting in samples of left ventricles of the hearts subjected to ischemia/reperfusion injury with and without angiotensin AT_1 receptor inhibitor and CaMKII inhibitor*
Experimental features	*Analysis of certain proteins indicating cellular injury due to apoptosis and necrosis*
Data source location	*Bratislava, Slovak Republic*
Data accessibility	*Data is supplied in this article*

Value of the data

- The data can be useful for other researchers investigating signaling pathways associated with CaMKII and AT_1 receptor activation under setting of ischemia/reperfusion in the heart.
- These data also provide significant contribution in understanding of pharmacological properties of AT_1 receptor inhibitors, drugs used in patients at high cardiovascular risk.

1. Data

In ischemic/reperfused (IR) group treated with CaMKII inhibitor, but not with AT_1 receptor inhibitor, Bcl-2/Bax ratio was significantly increased (Fig. 1) and the expression of caspase-3 and 89 kDa apoptotic fragment of PARP1 was changed by neither CaMKII inhibition nor angiotensin AT_1 receptor inhibition (Fig. 2). ~40 kDa necrotic fragment of PARP1 was upregulated in the IR group and no intervention was able to normalize these levels (Fig. 3). In the double-treated IR hearts, the levels of individual apoptotic and necrotic did not differ from those observed in either of mono-treated IR; however, the $p\sim$40PARP1/total PARP1 ratio was increased when compared to the IR group treated with CaMKII inhibitor alone.

2. Experimental design, materials and methods

Samples from the untreated and losartan and/or KN-93-treated hearts subjected to ischemia/reperfusion injury were used to examine protein expression by immunoblotting.

Proteins were separated using the SDS-PAGE and transferred to PVDF membrane (EMD Millipore, USA). The primary antibodies used were Bcl-2 (Sigma-Aldrich, USA), Bax (Cell Signaling, USA), PARP1 (Cell Signaling, USA), Caspase-3 (csp-3) p17 (Santa Cruz Biotechnology, USA) and β-actin (Sigma-Aldrich, USA). A HRP-conjugated donkey anti-rabbit antibody (GE Healthcare Life Sciences, UK) was

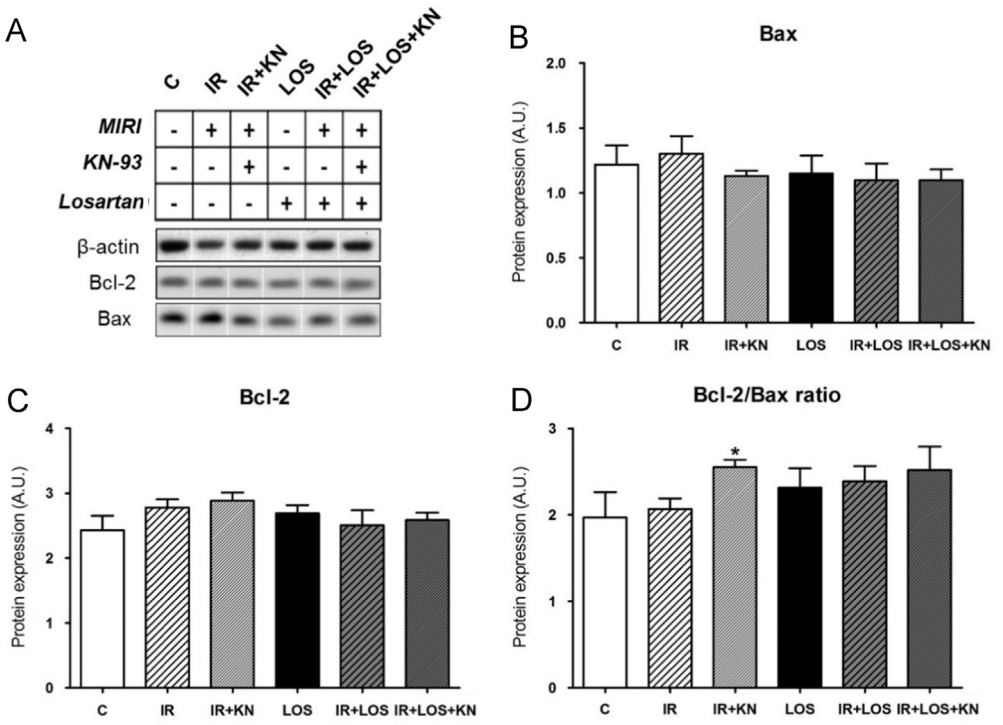

Fig. 1. Expression of Bcl-2 and Bax. (a) Representative western blots (b) Bcl-2 and (c) Bax expression (d) Bcl-2/Bax ratio. The values are expressed as the means ± S.E.M. ($n=5–7$ hearts per group). *$P<0.05$ vs. IR group.

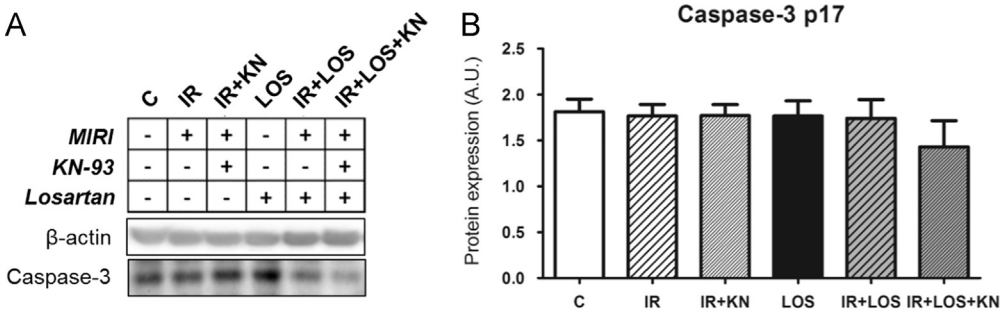

Fig. 2. Expression of cleaved caspase-3. (a) Representative blots (b) content of cleaved caspase-3. The values are expressed as the means ± S.E.M. ($n=5–7$ hearts per group).

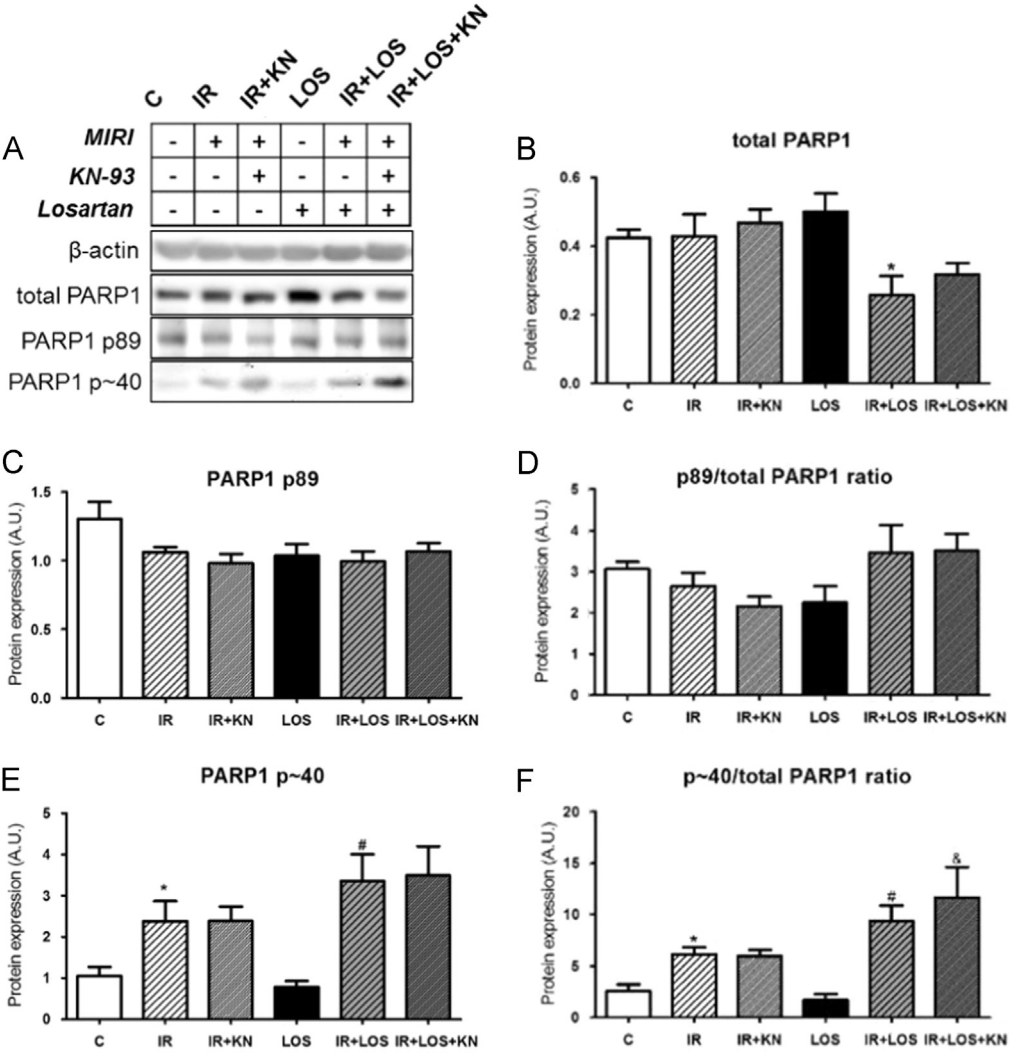

Fig. 3. Expression of total and cleavage fragments of PARP1. (a) Representative blots, (b) expression of total protein, (c) content of apoptotic PARP1 p89 fragment, (d) apoptotic p89 fragment to total PARP1 ratio, (e) necrotic PARP1 $p\sim40$ fragment and (f) ratio of necrotic PARP1 fragment to total PARP1. The values are expressed as the means ± S.E.M. ($n=5$–7 hearts per group). $^{*}P<0.05$ vs. C; $^{\#}P<0.05$ vs. LOS group; $^{\&}P<0.05$ vs. IR+KN.

used as the secondary antibody. The signals of proteins were detected using enhanced chemiluminiscence (Luminata, EMD Millipore, USA) and the blots were acquired using the MyECL Imager (Thermo Scientific, USA).

Data are expressed as the means ± S.E.M. Differences between variables with normal distribution were statistically analyzed by ANOVA with Newman–Keuls post-hoc test. Differences were considered as significant at $P\leq 0.05$.

Acknowledgments

This work was supported by grants VEGA 1/0638/12, APVV 0102-11 and by Hungarian Scientific Research Fund (OTKA K 109737). ZG holds a "János Bolyai Fellowship" from the Hungarian Academy of Sciences. PF is a Szentágothai Fellow of the National Program of Excellence (TAMOP 4.2.4.A/2-11-1-2012-0001).

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2016.03.017.

Reference

[1] T. Rajtik, S. Carnicka, A. Szobi, Z. Giricz, J. O-Uchi, V. Hassova, P. Svec, P. Ferdinandy, T. Ravingerova, A. Adameova, Oxidative activation of CaMKIIδ in acute myocardial ischemia/reperfusion injury: a role of angiotensin AT_1 receptor-NOX2 signaling axis, Eur. J. Pharmacol. 771 (2016) 114–122. http://dx.doi.org/10.1016/j.ejphar.2015.12.024.