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Data Article

Data demonstrating the anti-oxidant role of hemopexin in the heart

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ABSTRACT

The data presented in this article are related to the research article entitled **Hemopexin counteracts systolic dysfunction induced by heme-driven oxidative stress** (G. Ingoglia, C. M. Sag, N. Rex, L. De Franceschi, F. Vinchi, J. Cimino, S. Petrillo, S. Wagner, K. Kreitmeier, L. Silengo, F. Altruda, L. S. Maier, E. Hirsch, A. Ghigo and E. Tolosano, 2017) [1]. Data show that heme induces reactive oxygen species (ROS) production in primary cardiomyocytes. H9c2 myoblastic cells treated with heme bound to human Hemopexin (Hx) are protected from heme accumulation and oxidative stress. Similarly, the heme-driven oxidative response is reduced in primary cardiomyocytes treated with Hx-heme compared to heme alone. Our *in vivo* data show that mouse models of hemolytic disorders, β-thalassemic mice and phenylhydrazine-treated mice, have low serum Hx associated to enhanced expression of heme- and oxidative stress responsive genes in the heart. $Hx^{-/-}$ mice do not show signs of heart fibrosis or overt

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inflammation. For interpretation and discussion of these data, refer to the research article referenced above.

Specifications Table

Subject area	Health sciences
More specific subject area	Heme/iron biology
Type of data	Text file, Figures
How data was acquired	Olympus BH-2 microscope (Olympus Italia, Milan, Italy), 7300 Real Time PCR System (Applied Biosystems, Life Technologies Italia), spectrofluorimeter (Glomax, Promega Italia)
Data format	Raw, analyzed
Experimental factors	H9c2 (ATCC CRL-1446™) myoblast cell line; mouse neonatal primary cardiomyocytes; $Hx^{-/-}$ mice; β-thalassemia mice; C57BL/6 wild-type mice
Experimental features	Gene expression was analyzed by qRT-PCR and Western blotting. Tissue inflammation was analyzed by histology and immunohistochemistry. Heme content and ROS accumulation were quantified by fluorometric methods.
Data source location	Dept. Molecular Biotechnology and Health Sciences, Torino, Italy
Data accessibility	The data are available with this article.

Value of the data

- These data show that the plasma protein hemopexin (Hx) limits heme accumulation within cardiac cells both *in vitro* and *in vivo*
- In mice, heme-driven oxidative stress associated to Hx exhaustion can be recovered by the administration of the anti-oxidant α-tocopherol
- These finding might be exploited in the future for the development of Hx-based drugs able to prevent cardiac heme accumulation and oxidative stress in hemolytic disorders and/or in pathologic conditions associated with heme overload

1. Data

Data show that heme induced ROS production in primary cardiomyocytes (Fig. 1). Hx limited heme accumulation within H9c2 cell (myoblast cell line) and prevented ROS production. H9c2 cells were treated with heme alone or heme bound to Hx,and heme content, ROS production, the expression of heme- and oxidative stress responsive genes and markers of oxidative stress were evaluated (Fig. 2). These data were confirmed in primary cardiomyocytes isolated from neonatal mice and treated with either heme alone or heme-Hx (Fig. 3) and, indirectly in the heart of $Hx^{-/-}$ mice (Fig. 4). Data in Fig. 5 show that the heart of $Hx^{-/-}$ mice, despite of heme accumulation and elevated ROS [1], did not show sign of fibrosis and inflammation apart a slight increase in the level of Tumor Necrosis Factor (TNF)α and Interleukin (IL)-6 mRNAs.

In vivo, Hx depletion in mouse models of hemolytic disorders, β-thalassemic mice and phenylhydrazine (PHZ)-treated mice, was associated with heme accumulation and oxidative stress in the