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Data Article

Data on metabolic-dependent antioxidant response in the cardiovascular tissues of living zebrafish under stress conditions

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ABSTRACT

In this article we used transgenic zebrafish lines that express compartment-specific isoforms of the roGFP2-Orp1 and Grx1-roGFP2 biosensors, described in Panieri et al (2017) [1], to test the contribute of the pentose phosphate pathway and of the glutathione biosynthesis in the antioxidant capacity of myocardial and endothelial cells in vivo. The transgenic zebrafish embryos were subdued to metabolic inhibition and subsequently challenged with H2O2 or the redox-cycling agent menadione to respectively mimic acute or chronic oxidative stress. Confocal time-lapse recordings were performed to follow the compartmentalized H_2O_2 and E_{GSH} changes in the cardiovascular tissues of zebrafish embryos at 48 h post fertilization. After sequential excitation at 405 nm and 488 nm the emission was collected between 500–520 nm every 2 min for an overall duration of 60 min. The 405/488 nm ratio was normalized to the initial value obtained before oxidants addition and plotted over time. The analysis and the interpretation of the data can be found in the associated article [1].

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Specifications Table

Subject area	*Biology*
More specific subject area	*Cardiovascular Biology*
Type of data	*Figure, analyzed data (.csv)*
How data was acquired	*Confocal Microscope (*Leica Model SP5 and SP8, Leica Imaging Systems Ltd., Wetzlar, Germany)
Data format	*Graph of time-lapse confocal recordings (60 min)*
Experimental factors	*Oxidation of compartment-specific isoforms of the RoGFP2-Orp1 or Grx1-RoGFP2 sensors in the myocardial (myl7:RoGFP2) or endothelial cells (Kdrl:RoGFP2)*
Experimental features	*Real-time detection of roGFP2 Orp1 and Grx1 roGFP2 probes oxidation in living zebrafish exposed to H2O2 or menadione.*
Data source location	*Molecular Biotechnology Center, University of Turin, Italy*
Data accessibility	*Normalized R/R0 data are presented as mean ± SEM in* Figs. 1–3. *Associated data for each individual fish can be found as.csv files in the* Supplementary section *of the article.*

Value of the data

- These data show real-time changes of the H_2O_2 and E_{GSH} within selected subcellular compartments of the endothelial or myocardial cells of the living zebrafish during oxidative stress.
- Different subcellular compartments are characterized by distinct redox properties and antioxidant capacity.
- The data correlate the effect of metabolic inhibition with the antioxidant capacity of the cardiovascular system in vivo during oxidative stress.
- The data and the transgenic zebrafish lines here presented can stimulate further investigations on the role of oxidative stress in the cardiovascular physiopathology.
- The transgenic zebrafish model might be particularly useful to study tissue-specific and compartment-specific alterations of the redox balance induced by genetic mutations (with a suspected or proved pathogenic role in any human cardiovascular disease) or treatment with different drugs (i.e. cardiotoxicity of some anticancer drugs such as doxorubicin).

1. Data

The data describe the effect of pharmacologic inhibition of the pentose phosphate pathway (6PGD) or the GSH synthesis (GCLC) in the compartmentalized H_2O_2 levels (roGFP2-Orp1 probe) and E_{GSH} (Grx1-roGFP2 probe) of zebrafish cardiovascular tissues during oxidative stress [1]. In the endothelial cells neither the 6PGD nor the GCLC inhibition altered the kinetic and entity of peroxide accumulation in the cytosol, mitochondrial matrix or nuclei in response to exogenous H_2O_2 (Fig. 1A, C, E) while conversely faster and higher degree of peroxide accumulation was observed only in the cytosol in presence of either inhibitors and addition of menadione (Fig. 1B). In the myocardial cells, a trend of higher and faster probe oxidation was observed in the cytosol but not in the mitochondrial matrix when GCLC was inhibited and H_2O_2 administered (Fig. 2A) while both the inhibitors apparently promoted higher peroxide accumulation in the cytosol, mitochondrial matrix and nuclei after menadione treatment (Fig. 2B, D and F). Under the same experimental conditions the blockade of the GSH synthesis was associated with higher oxidation of the cytosolic, mitochondrial and nuclear E_{GSH} in the endothelial cells subdued to H_2O_2 stimulation (Fig. 3A, C, E) while only a trend of increase was observed in the nuclear E_{GSH} after menadione addition (Fig. 3F). Lastly, the inhibition of 6PGD pro-

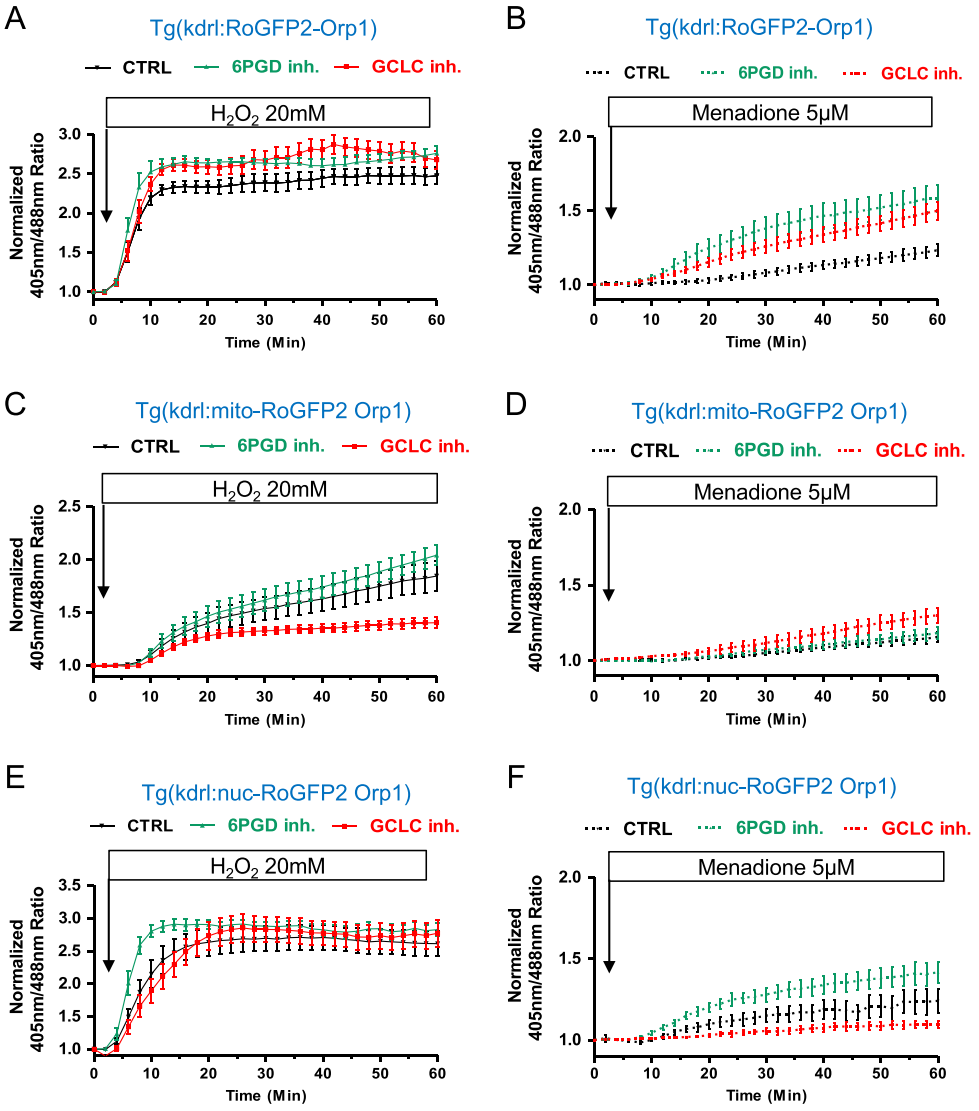

Fig. 1. Effect of PPP and glutathione synthesis blockade on H_2O_2 accumulation in living blood vessels of zebrafish embryos. Time lapse recordings of the compartment-specific Rogfp2-Orp1 probes expressed in the vasculature of transgenic zebrafish embryos at 48hpf. The embryos were pre-incubated with the indicated metabolic inhibitor or DMSO for 24 h before the addition of the oxidizing agents H_2O_2 20 mM (A,C,E) or menadione 5 μM (B,D,F), after which the changes in the 405/488 nm ratio were followed under confocal microscope for 60 min. For each probe, the values were averaged and normalized to the basal 405/488 ratio before treatment addition (set to 1.0) and therefore plotted as mean ± SEM.

moted higher E_{GSH} oxidation in the cytosol and nuclei of the endothelial cells after treatment with H_2O_2 (Fig. 3A and E) or menadione (Fig. 3B and F) but not in the mitochondrial matrix, irrespectively to the oxidant used (Fig. 3C and D).

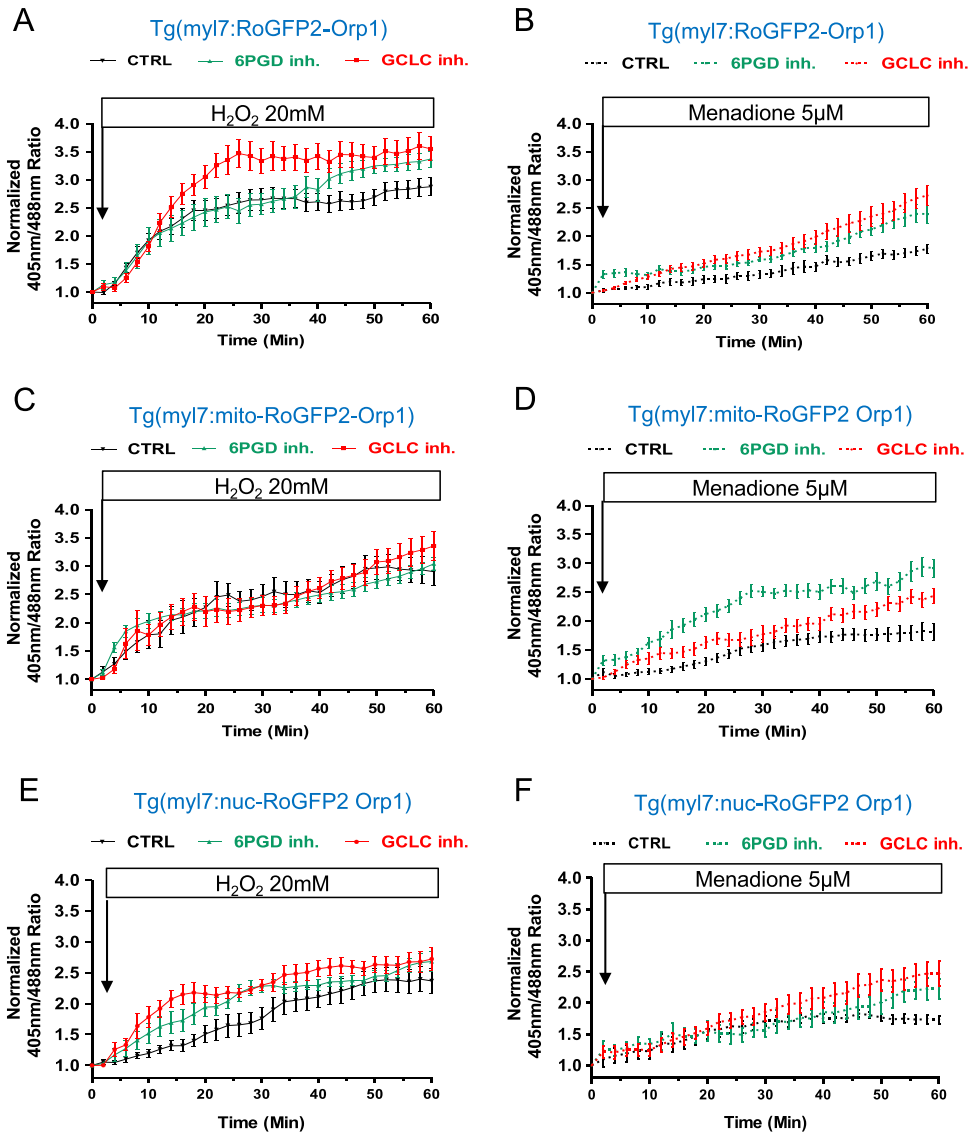

Fig. 2. Effect of PPP and glutathione synthesis blockade on H_2O_2 accumulation in living hearts of zebrafish embryos. Time lapse recordings of the compartment-specific Rogfp2-Orp1 probes expressed in the cardiomyocytes of transgenic zebrafish embryos at 48hpf. The embryos were pre-incubated with the indicated metabolic inhibitor or DMSO for 24 h before the addition of the oxidizing agents H_2O_2 20 mM (A, C, E) or menadione 5 μM (B, D, F), after which the changes in the 405/488 nm ratio were followed under confocal microscope for 60 min. For each probe, the values were averaged and normalized to the basal 405/488 ratio before treatment addition (set to 1.0) and therefore plotted as mean ± SEM.

2. Experimental design, materials and methods

2.1. Experimental design

Specific metabolic pathways can support the redox homeostasis of living heart [2,3] and vessels [4,5] providing antioxidant molecules such as NADPH or glutathione (GSH), to withstand oxidative stress, a condition particularly detrimental for the cardiovascular system [6,7]. Transgenic zebrafish

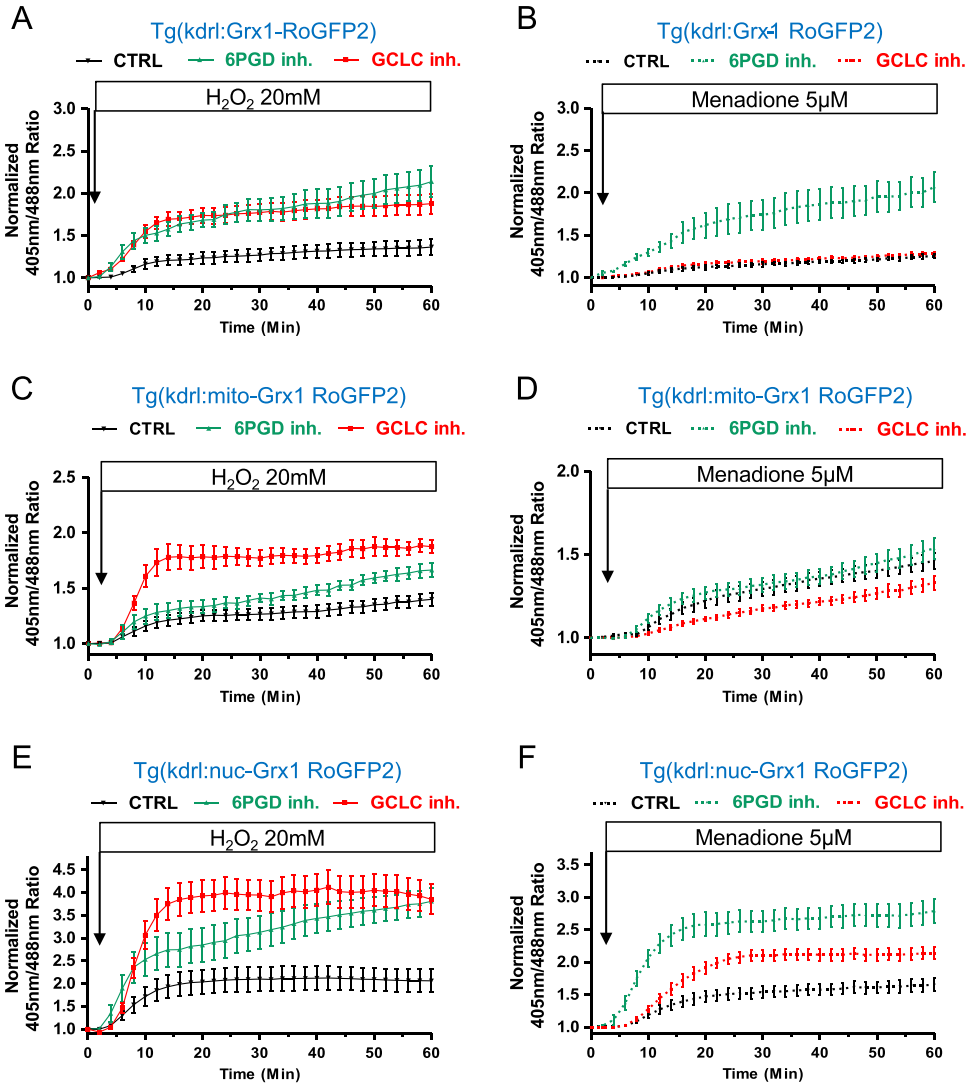

Fig. 3. Effect of PPP and glutathione synthesis blockade on GSH redox homeostasis in living blood vessels of zebrafish embryos. Time lapse recordings of the indicated compartment-specific Grx1-Rogfp2 probes expressed in the vasculature of transgenic zebrafish embryos at 48hpf. The embryos were pre-incubated with the indicated metabolic inhibitor or DMSO for 24 h before the addition of the oxidizing agents H_2O_2 20 mM (A, C, E) or menadione 5 μM (B, D, F), after which the changes in the 405/488 nm ratio were followed under confocal microscope for 60 min. For each probe, the values were averaged and normalized to the basal 405/488 ratio before treatment addition (set to 1.0) and therefore plotted as mean ± SEM.

expressing compartment-specific isoforms of roGFP2-based sensors for H_2O_2 [8] or E_{GSH} measurement [9] in the myocardial or endothelial cells were subdued to metabolic inhibition and thereafter treated with oxidizing agents mimicking acute (H_2O_2) or chronic (menadione) oxidative stress.

2.2. Drug treatments and embryos live imaging

The inhibitors BSO (50 mM) or 6AN (5 mM) were respectively used to block glutamate-cysteine ligase in the glutathione synthesis or the 6-phosphogluconate dehydrogenase in the pentose

phosphate pathway. Embryos at 24hpf were dechorionated and treated with each drug or 2.5% DMSO as vehicle in fish water (5 mM NaCl, 0.17 mM KCl, 0.33 mM CaCl2, and 0.33 mM MgSO4, pH 6.8–6.9) supplemented with 0.003% 1-phenyl 2-thiourea (PTU) for additional 24 h at 28.5 °C. At 48hpf the embryos were anesthetized with 0.05% tricaine, embedded in a 35 mm glass bottom dish (WillCo Wells) using 0.5 mL of 0.5% low melting agarose and covered with 1 ml of fish water supplemented with 0.003% PTU. The dish was therefore mounted on a confocal TCSII SP5X confocal microscope equipped with a motorized stage and a temperature-controlled chamber (set to 28.5 °C). The real-time changes of the roGFP2 probes were recorded for 60 min in a time-lapse experiment after the addition of either H_2O_2 20 mM or menadione 5 μM using an oil-immersion objective HCX PL FLUOTAR 20X (NA 1.0).Time lapse images were acquired for 60 min at 5 different Z-stack positions after sequential excitation at 405 nm and 488 nm every 2 min followed by emission detection at 500–520 nm using a frame resolution of 512 × 512 pixels, a section thickness of 4.0 μm and scanning frequency of 400 Hz. The xy curves represent normalized ratios ± SEM obtained dividing the 405/488 nm ratio at different time points for the ratio before treatment addition (R/R0), set to 1.

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Transparency document. Supplementary material

Transparency data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2017.04.034.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2017.04.034.

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