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Data Article

Preferential binding of 4-hydroxynonenal to lysine residues in specific parasite proteins in plakortin-treated *Plasmodium falciparum*-parasitized red blood cells

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ABSTRACT

The data show the frequencies by which the amino acid residues lysine, histidine and cysteine of six proteins of the malaria parasite *Plasmodium falciparum* are post-translationally modified by the lipoperoxydation endproduct 4-hydroxynonenal after challenging the parasitized red blood cell with plakortin. Plakortin is an antimalarial endoperoxide whose molecular anti-parasitic effect is described in Skorokhod et al. (2015) [1]. Plakortin did not elicit hemoglobin leakage from host red blood cells and did not oxidize reduced glutathione.

Specifications Table

Subject area	Biology
More specific subject area	Molecular pharmacology
Type of data	Tables, graphs

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How data was acquired	MALDI-TOF spectrometer (MALDI micro MX, Waters, Milford, MA, USA); Luminometer Sirius (Berthold, Pforzheim, Germany)
Data format	Processed data
Experimental factors	Parasitized and non-parasitized red blood cells were kept in in vitro cell culture up to 24 h. Chemical extraction for parameter assessment.
Experimental features	Heme-dependent luminol-enhanced luminescence assay for hemoglobin. Thiol-reaction with DTNB and OD measurement at 412 nm. Mass spectrometric analysis of peptides after trypsin-digestion of proteins.
Data source location	University of Torino, Torino, Italy
Data accessibility	Data are provided with this article

Value of the data

- The data show how endoperoxide-elicited oxidative stress might specifically target parasite proteins of functional importance by binding the lipoperoxidation endproduct 4-HNE.
- Data on endoperoxide-elicited modifications of proteins of a unicellular parasite as molecular cause for cell death may be of interest for similar studies with other microorganisms.
- Data for lethal modifications of specific microbial proteins with lipoperoxidation endproducts such as 4-HNE may suggest a novel strategy for high throughput drug research, e.g. antimalarials.
- The data on host red blood cells (RBC) intactness may be useful to monitor host cell damage by redox active substances.

1. Data

1.1. Frequencies of lysine residues modified by 4-HNE

In Table 1 we list the number of lysine, histidine and cysteine residues (K, H and C, respectively) that were found conjugated with 4-HNE and compare them with the total number of each of these amino acids in the respective protein. The complete list of 4-HNE modified *Plasmodium falciparum* proteins detected after plakortin treatment and specific sites of modification are reported in [1].

There is no particular imbalance between the portion of 4-HNE-conjugated amino acids in the proteins extracted from plakortin-treated parasites. We note that *Plasmodium* proteins contain elevated % of asparagine and lysine residues [2], even more then in vertebrates, where K frequency is 2–3 times higher than that of H and C [3].

Evidently, availability of the amino acid residue for 4-HNE is a crucial factor for binding. C residues engaged in disulfide bridges are no ligands for 4-HNE, while the basic amino acid K might be more frequently exposed to the protein–solvent interface compared to H, and hence be more accessible to 4-HNE binding [4]. The data about unbalanced ratios were published for other proteins such as liver fatty acid-binding protein [5], cytochrome c [6] and alpha-synuclein [7], where 4-HNE binding site distribution between K, H, C was 4:1:1 [5]; 10:1:0 [6] and 2:1:0 [7] respectively.

1.2. Data for hemoglobin release from RBC under plakortin treatment

Culture of RBC infected (parasitized) with trophozoite-stage *P. falciparum* were treated with 0–10 μM of plakortin and hemoglobin release was measured in culture supernatant (Fig. 1). The concentration is indicated in nmol/l. The concentration in the whole RBC suspension was 1 mM and assessed after complete lysis of RBC in NaOH/Triton: this is the highest maximal achievable concentration of hemoglobin in the supernatant corresponding to 100% RBC lysis.

As shown in Fig. 1 the hemoglobin was not released from RBC even at 10 μM plakortin, the highest applied concentration. Very low lysis was detectable in non-parasitized RBC (npRBC) and a still very modest, although double as high value in trophozoite-parasitized mature forms of *P. falciparum*. This