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Redox Status of Cardiac Cells. Ferritin, Reactive Oxygen and Nitrogen Species.

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It has been demonstrated that ferritin stimulates the free-radical oxidation of rat heart mito-chondria induced by tert-butyl hydroperoxide, at the same time, the dinitrosyl-iron complexes (DNIC) and combination of S-nitrosoglutathione with reduced glutathione inhibit effectively lipid peroxidation cascades in mitochondrial membranes. The antioxidant effect of DNIC was confirmed by their interaction with tert-butyl alkoxyl and alkylperoxyl radicals, superoxide radi-cals and peroxynitrite. In condition of tert-butyl free radical generation, DNIC were rapi-dly destructed, the ferritin inhibiting this process. The destruction of DNIC could be also observed, when superoxide radicals were generated by mitochondrial respiratory chain or xanthine-xanthine oxidase system. DNIC effectively inhibited the destruction of β-carotene induced by peroxynitrite. The nature of destruction of DNIC under the action of peroxynitrite indicates on the possibility of formation of intermediates. Evidently, the mitochondrial enzy-metic systems are able to contribute to the regeneration of these intermediates into initial nitrosyl form. Their formation in the reaction system containing mitochondria, ferritin, S-nitrosoglutathione with reduced glutathione points to the interconnection of the metabolism of iron, superoxide and DNIC. Apparently, the balance between antioxidant and prooxidant reactions in cardiac cells depends on the equilibrium between DNIC, ferritin and “free” Fe ions.