Lactoferrin (Lf) is an 80-kDa iron-binding glykoprotein. Lf is present physiologically in exocrine secretions, eg. tears, saliva, milk, sinovial fluid, seminal fluid and in the secondary granules of neutophils. The precise function of Lf in organism is considered to be very complex and it is still a hot subject of scientific disputation. Lf was documented to act as antimicrobial, antiinflamatory and antitumoral agent. Another of its properties is ability to inhibit hydroxyl radical formation via Fenton reaction. Afinity of Lf to iron is about 260times higher than that of transferrin.

Isoproterenol (ISO) has been used as a model compound to induce infarct-like lesions in the rat and various other animal species. Administration of necrotic dose of ISO caused ischaemia followed by damage of the myocardium. Ischaemia alters iron homeostasis and redox-active free iron, which catalyses ROS-generation. The only possibility for myocardial tissue recovery in acute moycardial infarction represents the reperfusion of ischemic myocardium. But the whole process is associated with damage of myocardium due to burst of hydroxyl radical catalyzed by free iron (Fenton reaction).

In our experiment we studied the effects of Lf in a catecholamine model of myocardial injury. We had four experimental groups divided according to the received medication: control (saline), isoprenaline (ISO, 100 mg/kg s.c.), bovine lactoferrin (La, 50 mg/kg i.v.) and the combination of La+ISO in the above-mentioned doses (La was administered 5 min before ISO). After 24 h a sample of blood was withdrawn and the heart was removed for analysis of various parameters.

Control and lactoferrin treated animals had negligible levels of serum cTnT and there was no statistical difference between myocardial calcium levels in these groups. Isoprenaline brought about a marked cTnT release and myocardial calcium overload. Lactoferrin premedication did not affect the release of cTnT, however, it decreased calcium overload caused by ISO. Lactoferrin significantly elevated levels of total glutathione while significantly decreasing glutathion peroxidase in erythrocytes.

Our experiment did not show the prophylactic administration of bovine laktoferin on myocardial injury caused by s.c. administration of necrogenic dose of ISO. The partial protective effect of lactoferrin is based on inhibition of ROS formation due to chelation of free iron. Our experiment was under the grant GA UK 94/2006/C/Faf.