Chapter 7
Mitochondria of the Human Transplanted Heart

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Abstract Mitochondrial oxidative phosphorylation function and coenzyme Q_{10} concentration in endomyocardial biopsies (EMB) of human transplanted heart patients (HTx-pts) were documented. Decreasing mitochondrial energy production and diminished CoQ_{10} concentration in EMB of HTx-pts is in relationship with rejection development of the transplanted heart.

Keywords Coenzyme Q_{10}, EMB, HTx-pts, mitochondrial function, rejection

Heart transplantation is an accepted therapy for patients with end-stage heart failure. Years of patient survival after heart transplantation depend on various factors, such as number of rejections, immunosuppression, production of free radicals, function of antioxidant defense system [8], mitochondrial coenzyme Q_{10} content and oxidative phosphorylation function [2, 3, 7].

Cyclosporin A, which even in very low concentrations damages mitochondrial function, is used as an immunosuppressive drug. It inhibits mitochondrial permeability transition pore (MPTP), which is characterized by progressive permeabilization of the inner mitochondrial membrane, permitting passage to protons, ions, and even small proteins, stimulated by osmotic support [6]. Patients with transplanted hearts (HTx-pts) require continual complex medical care and complex therapy for a lifetime. HTx-pts have to be regularly checked for prevention and therapy of infectious diseases [1]. In spite of effective immunosuppression, acute rejection of the transplanted heart is one of the greatest problems in the first year after heart transplantation. Decreased myocardium mitochondrial bioenergetic processes and diminished coenzyme Q_{10} concentration in the transplanted heart are also involved in the pathobiochemical mechanisms of human transplanted heart rejection [3, 4].

CoQ_{10} is a key part of the mitochondrial respiratory chain connected with oxidative phosphorylation. In patients with heart failure, pronounced deficiency of CoQ_{10} was described [2, 5]. Mitochondrial function of the human transplanted heart is shown in Fig. 7.1 [3, 4].

Mean age of patients was 45 years, range 16–63 years. Twenty eight endomyocardial biopsies (EMB) were divided according to the histologically
Fig. 7.1  Relationship between coenzyme Q₁₀ concentration in EMB and degree of rejection of human transplanted heart

Fig. 7.2  Basal mitochondrial respiration in EMB of HTx-pts in relation to degree of rejection (V₁)