According to modern concepts, age-related tissue damage and aging are caused by oxidative stress and enhanced reactive oxygen species (ROS) production. Mitochondria are considered to be the main source of ROS; therefore, the functional and structural characteristics of mitochondria during aging are explained within the scope of the Harman’s free radical theory of aging [1].

Variations in mitochondrial ultrastructure have been studied to a lesser extent as compared to functional changes. Age-related alterations in the ultrastructure of mitochondria in various tissues (including myocardium and skeletal muscle) have quite a number of similar indications: mitochondrial swelling, cristae reduction, matrix clarification, and mitochondrial membrane damage [2–7]. Ultrastructural morphometric analysis has shown an age-related decrease in the ratio of the inner mitochondrial membrane surface to mitochondrial volume in hamster myocardium [8], as well as the shortening and elongation of the long and short axes of mitochondria, respectively, in C57BL/6 mice [9]. The number of mitochondria in the subsarcolemma region [10] and interfibrillar mitochondria [11] was lower in the biopsy samples from the skeletal muscle of elderly people (69–70-year-old). At the same time, Tomanek and Karlsson [12] thoroughly analyzed myocardial ultrastructure in young (3- to 6-month-old) and senile (27- to 28-month-old) rats but did not reveal previously described age-related changes in mitochondria such as fragmentation of cristae and reduction of matrix density. Riva et al. [13] who studied the ultrastructure of rat myocardial mitochondria in aging by scanning electron microscopy did not reveal any destructive changes in cristae either. Only two models are known (Caenorhabditis elegans and Drosophila melanogaster), where the correlation between structural parameters and energy regime of mitochondria during aging has been established. These models unambiguously show that oxidative stress plays a key role in age-related alterations of mitochondrial ultrastructure [14–16]. It is known that the brain and muscular tissue suffer from mitochondrial oxidative damage most severely [17]. Hence, it would be extremely relevant to study the mitochondrial ultrastructure of these tissues.

We have performed the comparative analysis of age-related alterations of mitochondrial ultrastructure of the flight muscle, cardiomyocytes, and skeletal muscle. To this end, we studied the morphology of mitochondria of the flight muscle of D. melanogaster (1- and 36-day-old flies; in this line, the lifespan of an individual is 40–50 days); of cardiomyocytes of Wistar and OXYS rats (25-month-old), and of skeletal mus-
Five animals from each age group were used in the experiment for each type of tissue. Flight muscle preparations were obtained as follows: the \textit{D. melanogaster} thoracic segment was placed in chilled fixative for 1 h and then the flight muscle was isolated mechanically using a LEIKA MZ12.5 stereo microscope. The animals were kindly provided by E.G. Pasukova (Institute of Molecular Genetics, Russian Academy of Sciences, Moscow).

The tissue of the left ventricle of heart was used to study the ultrastructure of mitochondria in rat cardiomyocytes. The stuff was taken immediately after decapitation of an animal. Skeletal muscle preparations were obtained from fragments of the gastrocnemius muscle taken from animal’s shank after decapitation. The experimental stuff of the Wistar and OXYS rat lines was kindly provided by N.G. Kolosova (Institute of Cytology and Genetics, Siberian Branch of the Russian Academy of Sciences, Novosibirsk).

Tissue samples for electron microscopy were fixed for 1 h with 4% glutaraldehyde solution in 0.1 M phosphate buffer at pH 7.2–7.4. After the fixation with glutaraldehyde, the tissue was washed for 30 min with 0.1 M phosphate buffer (pH 7.2–7.4) and then fixed with 1% OsO$_4$ in phosphate buffer for 1.5–2 h. After the fixation, the samples were dehydrated in a series of alcohol-containing solutions, where the concentration of alcohol increased (30–40 min each time). The 70-percent alcohol contained a saturated solution of uranyl acetate (1.5%). Samples were left in this solution for 12–20 h at 4–5°C. After dehydration, samples were embedded in Epon-812 and cut using an Ultracut III ultramicrotome (Leica). The preparations were stained with lead citrate according to Reynolds. Mitochondrial ultrastructure was studied using Hitachi HU-11, HU-12, and JEM-1400 electron microscopes.

RESULTS AND DISCUSSION

Ultrastructure of mitochondria from the flight muscle of a 36-day imago of \textit{D. melanogaster} is shown in Fig. 1. Destructive changes can be seen nearly in all mitochondria. Spatial reorganization of the inner membrane is observed in the local regions of mitochondria: the correctly located lamellar cristae disappear and the membranes form a layered structure, where it is impossible to distinguish between the matrix and the intermembrane space. Then the concentric layers of electron-dense membranes are formed in the mitochondria; they gradually increase and eventually lead to complete destruction of the mitochondria [18]. The mitochondrial ultrastructure in the flight muscle of a young (1-day-old) fly \textit{D. mel-