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STRUCTURAL CHANGES IN STRIATED MUSCLES OF DYSTROPHIC HAMSTER

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Since the first introduction of a strain of myopathic hamster by Homburger's group[1] in 1962, attention has been given to the myopathy of this animal, especially its cardiomyopathy. This strain has an autosomal recessively inherited polymyopathy, but has no other defective organs or tissues except for these striated muscles.

The present work aimed at checking the degree of damage in various skeletal muscles at different ages. BIO 15.6 male hamsters were used for the studies. After being sacrificed by a blow on the head and bleeding, the animal was fixed on a board and the various muscles were carefully removed including both terminal tendons. The muscles were fixed *in situ* length on dental wax and 4% glutaraldehyde buffered with cacodylate was poured on the materials. They were then teased into small bundles of fibers by forceps 30 min later and transferred to a small bottle containing a glutaraldehyde fixative. The usual methods were then followed of a second fixation of osmium tetroxide, staining *en bloc*, dehydration with alcohol and acetone, and embedding in epoxy resin. A relatively thick section with toluidine blue staining was prepared for light microscopy and a thin section with lead staining for electron microscopy. The cross section view for light microscopy was mainly used to determine the grade of muscle damage and this was done by the criteria according to Bajusz *et al.*[2], slightly modified as follows: ±, less than 4 cells with central nuclei (CN) among 100 cross sectioned cells; +, less than 8 (CN) cells in 100; ++, more than 8 (CN) cells in 100; +++, abundance of CN cells and other abnormalities, ++++, severe abnormalities with necrotic damage.

As shown in Table 1, 12 striated muscles of different ages were tested with fixed materials. All had some degree of abnormalities, particularly severe ones in the shoulder muscle. When the central nuclei were seen in a longitudinal section, they were arranged in a long row. In this same section an abnormal arrangement of nuclei was recognized in a long row although they were not localized at the center but at the periphery (Fig. 1). Other nucleic abnormalities such as perinuclear cavities or pores were sometimes present. Myofibrils usually looked generally normal even in a grade +++ muscle, except for the nuclear anomaly. When the muscle was observed in detail under high magnification, both free polysomes and membrane-bound ribosomes were distributed around the nucleus and submembraneous region, which could not be seen in a normal adult muscle. In a grade ++++ muscle some vacuoles and disarrangement of myofibrils appeared here and there together with abundant CN.

TABLE 1. The Degree of Muscle Damage

		77 days	97 days	110 days	130 days
Arm muscles	M. extensor digitrum communis			±	
	M. extensor carpi radialis brevis				++
Shoulder muscles	M. omotransversarius		+++*	++++*	
	M. omobrachialis				+++
Back muscle	M. lumbodorsal aponeurosis				+++*
Abdominal muscle	M. external adbominal oblique			+	
Esophagus	M. esophagus		+*	+	
Diaphragma	M. diaphragma		+++	++*	
Leg muscles	M. samitendinosus		++	+	
	M. soleus	++			
	M. extensor digitrum longus	+++			
	M. sartorium	++		++++*	

* Appearance of necrotic cell.