character of the inflammatory process in the present investigation, with transition from the exudative-necrotic stage into the proliferative stage. In the present experiments, it was evidently this functional integrity of the monocytic macrophages which determined the completed character of phagocytosis and the favorable outcome of the inflammatory process.

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LIMITS OF ULTRASTRUCTURAL HOMEOSTASIS IN SKELETAL MUSCLES UNDER NORMAL CONDITIONS AND DURING MUSCULAR ACTIVITY

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Modern views on the structural basis of homeostasis have been developed by Academician of the Academy of Medical Sciences of the USSR, D. S. Sarkisov [4, 5]. The principles of the supply of materials required for function, which he has argued, stipulate precisely that a change in function of any organ within normal limits is determined by variation of the number of its working structural units. A rapid increase in functional load is possible due to an increase in mass of the working organs or systems of organs [12]. This process, the material basis of adaptation, takes place through hyperplasia of its components [7]. The increase in their number, moreover, may take place at both cellular and intracellular levels [6]. Three forms of adaptive increase in the number of structural components of intensively working organs are distinguished: the first is based on new cell formation, the second on a roughly equal increase in numbers both of cells and of intracellular components, and the third on intracellular regeneration alone [5].

Considering the traditional views on hypertrophy of skeletal muscles during intensive physical exertion (PE) the following question naturally arises: on account of an increase in number of what structure does the mass of the working muscle increase? Accordingly, the aim of this investigation was to determine the limits of fluctuations in the number of dominant ultrastructures of skeletal muscles under normal conditions and during intensive PE.

EXPERIMENTAL METHOD

Experiments were carried out on 162 male albino rats aged 1, 3, and 12 months. PE, in the form of running on a treadmill, took place for 20, 40, 60, and 90 days at a speed of 45 m/min. Rats of the same age, kept under animal house conditions, served as the control. After decapitation, pieces of the pectoralis major (PM) and latissimus dorsi (LD) muscles were excised. Material was prefixed in 4% glutaraldehyde solution, postfixed in 1% OsO_4 solution, and embedded in a mixture of Epon and Araldite. The sections were stained with uranyl acetate

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TABLE 1. Experimental Kinetics of Dominant Components of Muscle Fibers

Test object	C	E	C	E	C	E
PM	1+20		1+40		1+60	
mf	78,42	80,23	80,05	83,86	84,76	90,44
mch	5,91	9,92	5,72	6.42	2,50	1,9
Σmf+mch	84,33	90,15	85,97	90,28	87,26	92,34
	1+90		3+20		3+40	
mf	84,16	86,37	82,23	86,80	82,94	84,37
mch	4,43	6,85	2,91	5,33	5,15	7,83
Σmf+mch	88,59	93,22	85,14	92,13	88,09	92,20
	3+60		3+90		12+20	
mf	81,12	86,45	83,92	88,13	81,04	81,00
mch	6,14	5,05	3,54	4,65	4,82	5,81
Σmf+mch	87,26	91,50	87,46	92,78	85,86	86,81
	12+40		12+60		12+90	
mf	82,73	82,93	82,04	82,43	80,65	80,94
mch	6,11	8,41	6,93	9,27	8,13	11,32
Σmf+mch	88,84	91,34	88,97	91,7	88,78	92,26
LD	1+20		1+40		1+60	
mf	77,35	81,07	79,24	82,34	85,27	91,38
mch	6,52	11,23	6,43	8,09	2,83	2,07
Σmf+mch	83,87	92,80	85,67	90,43	88,10	93,45
	1+90		3+20		3+40	
mf	83,84	86,92	83,07	86,15	83,25	84,14
mch	4,23	6,40	3,21	5,94	4,72	8,33
Σmf+mch	88,07	93,32	86,28	92,09	87,97	92,47
	3+60		3+90		12+20	
mf	82,24	88,16	83,12	87,64	82,94	83,52
mch	6,51	4,53	3,73	5,07	6,04	6,96
Σmf+mch	88,75	92,69	86,83	92,71	88,98	90,41
	12+40		12+60		12+90	
mf	83,24	82,65	81,84	82,35	80,06	81,09
mch	6,82	8,83	7,43	11,37	8,37	12,88
Σmf+mch	90,06	91,48	89,27	93,72	88,43	93,97

and lead citrate and examined in the UÉMV-100K and ÉMK-100AK electron microscopes. After standard to positive prints of equal size, the following parameters were determined with the aid of a test system for stereologic analysis [3]: volume fractions (Vv) of myofibrils (mf), mitochondria (mch), the total volume of these organelles (Vv_{mf} + Vv_{mch}), and Vv of the hyaloplasm (hp) and of all structures not counting mf + mch, designated Vv_i.

EXPERIMENTAL RESULTS

Contractile and energy-producing structures are those which dominate quantitatively and determine qualitative changes in the muscle fibers. Morphometric analysis indicates that the limits of fluctuations of Vv_{mf} and Vv_{mch} in rats between the ages of 1 and 15 months (Table 1) are normally clearly defined quantitatively. The range of changes of Vv of the test structures from the lower to the upper limit characterizes the adaptive capacity of changes in the skeletal muscles under normal conditions. For instance, for mf this is 77.35-85.27%, and for mch 2.5-8.37%. The difference between these parameters can be described as the "maximal structural reserve" (MSR), determining the limits of homeostasis of the muscle fibers. It is these organelles which, on the principle of intermittent activity, can be put into action at maximal PE. MSR for mf was 7.92% and for mch 5.87%. Physical exertion widens the limits of adaptation of the skeletal muscles due to an increase in the number of dominant structures. After running Vv_{mf} changed from 80.23 to 91.38%, and Vv_{mch} from 1.90 to 12.88%. MSR for Vv_{mf} was 11.15%, compared with 10.38% for Vv_{mch}. The adaptive capacity of the dominant components of the muscle fibers after exertion rose for Vv_{mf} by 3.23% and for Vv_{mch} by 5.11%.

As emphasized in [1], an important role in the phenomena of physiological regeneration is played by newly formed intracellular organelles (mf, mch, lamellar complex, etc.). To obtain a more complete picture of reorganization of the ultrastructures of the muscle fibers, integral quantitative changes in their dominant components must be used, i.e., Vv_{mf} + Vv_{mch}.

The limits of fluctuations in the total volume fraction Vv_{mf} + Vv_{mch} in intact rats were from 83.87 to 90.06%, i.e., MSR in this case was 6.19%. During PE the limits of variations of these parameters diverged: Vv_{mf} + Vv_{mch} from 86.81 to 93.97%. In this case MSR was 7.16%. In other words, analysis of the total volume fraction demonstrates the rigidity of the limits of structural homeostasis of muscle tissue.

The stereometric data on the kinetics of the volume fractions Vv_{hp} and Vv_i demonstrate the stability of the homeostatic limits of the remaining components of the muscle fibers.

In the control, for instance, Vv_{hp} was 2.5% and Vv_i 7.5%. PE, which activates biosynthesis of the dominant components of the sarcoplasm, led to reduction of these volumes for Vv_{hp} to 1.2% and for Vv_i to 5% respectively, i.e., here also the limits of possible displacement of structural homeostasis are very small.

Consequently, the results are evidence that fluctuation of the limits of homeostasis of mf and mch do not exceed 7%, even during PE. If we accept the views of those workers who assert that the formation of new muscle fibers in differentiated skeletal muscles takes place through the development of myosatellites [8, 10, 11], we shall therefore have to accept also that an increase in the number of working structures in a muscle takes place entirely on account of the intracellular regeneration of mf and mch (with the additional development of myosatellite cells into mature myons). If, however, we adopt the view that mature muscle fibers can divide longitudinally or transversely, it becomes clearer during PE the mass of muscle tissue increases due to both intracellular and cellular regeneration. However, since the question of the sources of formation of new muscle fibers in the differentiated muscle has not been finally settled [1], the two hypotheses remain equally valid. The quantitative data obtained in the present investigation show that adaptation of skeletal muscles to PE takes place on account of intracellular regeneration.

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