

KAS METABOLİZMASI

Elif ÖZKÖK

26.1. Önemi

Kas dokusu, vücut ağırlığının yaklaşık %40-50 'sini oluşturan kasılma özelliğine sahip en büyük dokudur. Kas dokusu başlıca vücut şeklinin ve postürün (duruşun) sağlanmasında, hareket, kasılma-gevşeme ve ısı üretiminde görevlidir. Bütün bunlar kas hücrelerinin uyarılabilir olması, yani sinir sisteminin aldıkları uyarıları iletebilmeleriyle gerçekleşmektedir. Bu işlevleri yerine getirebilmesi ise besinlerden sağlanan kimyasal enerjiyi mekanik enerjiye çevirmesiyle gerçekleşmektedir.

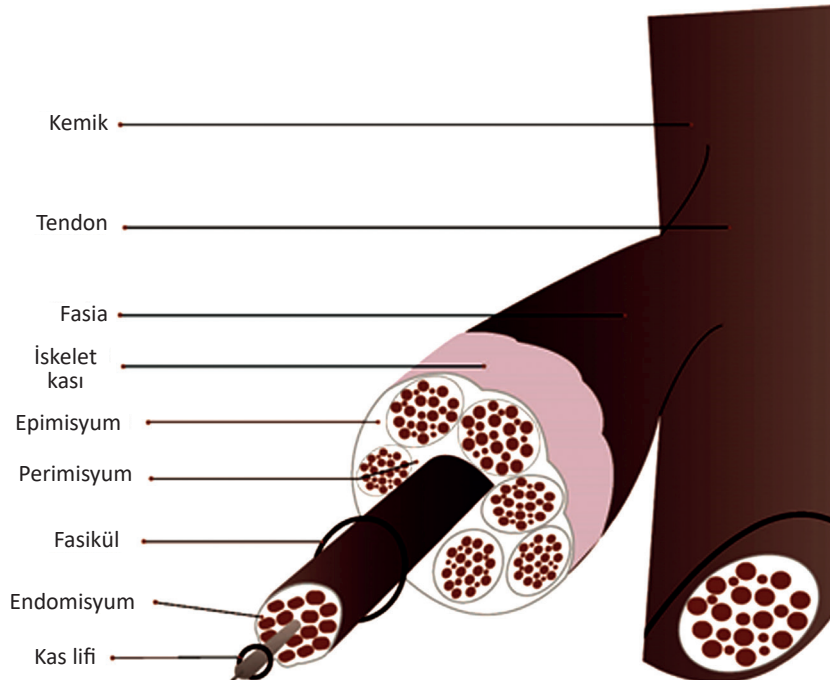
26.2. Kasın Yapısı

Kas dokusu yaklaşık olarak %72-78 su, %19-20 protein, %3 lipit ve %1 glikojenden oluşan; ipliksi şekilde çok sayıda kas hücrelerinin düzenli şekilde

biraraya gelerek kas demetlerini oluşturduğu bir yapıdır.

Vücudumuzdaki her kas, lif veya fibril olarak isimlendirilen silindirik yapıda kas hücrelerinin binlercesinin biraraya gelmesinden oluşmaktadır. Her kas lifinin üzeri "Endomisyum" denen konnektif doku ile sarılmıştır. Yaklaşık 150 lif bir araya gelerek lif demetlerini (fasikül) oluştururlar. Bu demetlerin üzerini saran konnektif doku ise "Perimisyum" adını alır. Fasiküllerin bir araya gelmesiyle iskelet kası oluşur; kasın üzerini de Epimisyum adı verilen konnektif doku sarar (Şekil 26.1).

Bir tek kas lifi hücresi "Sarkolemma" adı verilen hücre zarıyla çevrelenmiş olup, "Sarkoplazma" denen intrasellüler sıvı içinde birbirine paralel olarak yerleşmiş yüzlerce "Miyofibrilden" oluşur.



Şekil 26.1. Kemik kas bağlantısı ve iskelet kasından enine kesitle altbirim ve konnektif doku düzenlenmeleri.

26.7. Kaynaklar

- Bottinelli R, Reggiani C. Human skeletal muscle fibres: molecular and functional diversity. *Prog Biophys Mol Biol* 2000; 73: 195-262.
- Bourgeois JM, Tarnopolsky MA. Pathology of skeletal muscle in mitochondrial disorders. *Mitochondrion* 2004; 4: 441-452.
- Choi B, Hwang JH, Kim J, Cho EM, Cho SY, Hwang SJ, et al. A MELAS syndrome family harboring two mutations in mitochondrial genome. *Exp Mol Med* 2008; 40: 354-360.
- Collins J, Bonnemann CG. Congenital muscular dystrophies: toward molecular therapeutic interventions. *Curr Neurol Neurosci Rep* 2010; 10: 83-91.
- Connolly BS, Feigenbaum AS, Robinson BH, Dipchand AI, Simon DK, Tarnopolsky MA. MELAS syndrome, cardiomyopathy, rhabdomyolysis, and autism associated with the A3260G mitochondrial DNA mutation. *Biochem Biophys Res Commun* 2010; 12: 443-7.
- Finsterer J. Biomarkers of peripheral muscle fatigue during exercise. *BMC Musculoskelet Disord.* 2012; 13: 218.
- Flück M. Functional, structural and molecular plasticity of mammalian skeletal muscle in response to exercise stimuli. *J Exp Biol* 2006; 209: 2239-2248.
- Gordon AM, Homsher E, Regnier M. Regulation of contraction in striated muscle. *Physiol Rev* 2000; 80: 853-924.
- Harris MA, Hammond KM, Fell JM, Morton JP. Regulation of Muscle Glycogen Metabolism during Exercise: Implications for Endurance Performance and Training Adaptations. *Nutrients.* 2018 Mar 2;10(3). pii: E298.
- Huxley H, Niedergerke R. Structural changes in muscle during contraction: interference microscopy of living muscle fibres. *Nature* 1954; 173:971-973.
- Johnson LN. The regulation of protein phosphorylation. *Biochem Soc Trans* 2009; 37: 627-641.
- Karatzafiri C, de Haan A, Ferguson RA, van Mechelen W, Sargeant AJ. Phosphocreatine and ATP content in human single muscle fibres before and after maximum dynamic exercise. *Pflugers Arch* 2001; 442: 467-474.
- Katz A, Andersson DC, Yu J, Norman B, Sandström ME, Wieringa B, Westerblad H. Contraction-mediated glycogenolysis in mouse skeletal muscle lacking creatine kinase: the role of phosphorylase b activation. *J Physiol (Lond.)* 2003; 553: 523-531.
- Lehmann D, Motlagh L, Robaa D, Zierz S. Muscle Carnitine Palmitoyltransferase II Deficiency: A Review of Enzymatic Controversy and Clinical Features. *Int J Mol Sci.* 2017 Jan 3;18(1).
- Longo N, di San Filippo C A, Pasquali M. Disorders of carnitine transport and the carnitine cycle. *Am J Med Genet C Semin Med Genet* 2006; 142: 77-85.
- Manickam AH, Michael MJ, Ramasamy S. Mitochondrial genetics and therapeutic overview of Leber's hereditary optic neuropathy. *Indian J Ophthalmol.* 2017 Nov;65(11):1087-1092.
- McArdle B. Myopathy due to a defect in muscle glycogen breakdown. *Clin Sci* 1951; 10: 13-35.
- Ørngreen MC, Schelhaas HJ, Jeppesen TD, Akman HO, Wevers RA, Andersen ST, et al. Is muscle glycogenolysis impaired in X-linked phosphorylase b kinase deficiency? *Neurology.* 2008; 70:1876-82.
- Melzer K. Carbohydrate and fat utilization during rest and physical activity. *e-SPEN, the European e-Journal of Clinical Nutrition and Metabolism* 6 (2011) e45-e52
- Ørtenblad N, Nielsen J. Muscle glycogen and cell function--Location, location, location. *Scand J Med Sci Sports.* 2015 Dec;25 Suppl 4:34-40.
- Pette D, Staron RS. Myosin isoforms, muscle fiber types, and transitions. *Microsc Res Tech* 2000; 50: 500-509.
- Preisler N, Laforêt P, Madsen KL, Prahm KP, Hedermann G, Vissing CR, et al. Skeletal muscle metabolism is impaired during exercise in glycogen storage disease type III. *Neurology* 2015; 84:1767-71.
- Rivera-Brown AM, Frontera WR. Principles of exercise physiology: responses to acute exercise and long-term adaptations to training. *Physical Medicine and Rehabilitation* 2012 Nov;4(11):797-804.
- Roach PJ. Glycogen and its metabolism. *Curr Mol Med* 2002; 2: 101-120.
- Schiaffino S, Sandri M, Murgia M. Activity-dependent signaling pathways controlling muscle diversity and plasticity. *Physiology (Bethesda)* 2007; 22: 269-278.
- Spangenburg EE, Booth FW. Molecular regulation of individual skeletal muscle fibre types. *Acta Physiol Scand* 2003; 178: 413-424.
- Spriet LL, Watt MJ. Regulatory mechanisms in the interaction between carbohydrate and lipid oxidation during exercise. *Acta Physiol Scand* 2003; 178: 443-452.
- Vissing J, Quistorff B, Haller RG. Effect of fuels on exercise capacity in muscle phosphoglycerate mutase deficiency. *Arch Neurol* 2005; 62:1440-1443.