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CHARACTERISTICS OF MIR-29 BINDING SITES IN mRNAs OF HUMAN MUSCLE GROWTH REGULATING GENES

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It is known today that 39 genes (ACVR1B, ACVR2B, AKT1S1, ATG13, BECN1, BNIP3, CHUK, DDIT4, DEPTOR, EIF4EBP1, FBXO32, FOXO1, FOXO3, GSK3B, HDAC4, IKBKB, IRF1, KLF15, LATS1, LATS2, MAP1LC3A, MAPK8, MSTN, NFKB1, PDCD4, PPARGC1A, PRKAA1, PRKAA2, PTEN, SMAD2, SMAD3, SMAD4, STK3, TGFBR1, TRIM54, TRIM55, TRIM63, TSC1, TSC2) are strongly connected with skeletal muscle atrophy and 27 genes (AKT3, CTNNB1, EIF2B2, FST, IGF1, IGF1R, IRS1, MAPK1, MTOR, MYOD1, MYOG, NFATC1, NFATC2, NFATC3, NFATC4, NUA2, PDK1, PIK3C3, PIK3R1, PLD1, PPP3CA, RHEB, RPS6KB1, RPTOR, SGK1, SMAD7, SRF) are connected with skeletal muscle hypertrophy. Experimental studies showed that some of these genes are regulated by miR-29 family. So it was important to study in silico exact characteristics of binding sites for this miRNA family in mRNAs of skeletal muscle mass regulating genes.

Materials and methods. The nucleotide sequences of mRNAs of 66 human protein-coding genes, regulating the growth of muscle mass, were downloaded from NCBI GenBank (<http://www.ncbi.nlm.nih.gov/genbank/>) in FASTA format. Nucleotide sequences of miR-29a-5p, miR-29a-3p, miR-29b-1-5p, miR-29b-2-5p, miR-29b-3p, miR-29c-5p and miR-29c-3p were downloaded from the miRBase database (<http://mirbase.org>). miRNA binding sites in the 5'-untranslated regions (5'UTRs), the coding domain sequences (CDSs) and the 3'-untranslated regions (3'UTRs) of mRNAs of genes were predicted by using TargetScan program (www.targetscan.org).

Results. As a result of this study it was found that 66 human genes, regulating the growth of skeletal muscles, have 194 binding sites for miR-29 with the level of complementarity that is equal or more than 75%. 14 genes (AKT1S1, CTNNB1, FBXO32, FOXO3, GSK3B, HDAC4, MSTN, NFATC1, NFATC2, PDCD4, PIK3C3, RPTOR, TRIM54 and TRIM63) had 18 binding sites for miR-29(a, b, c) with the level of complementarity ranging from 80% to 85%. MiR-29a had 72 binding sites with the level of complementarity varying from 75% to 81%. MiR-29b also had 72 binding sites with the level of complementarity ranging from 76% to 85%. MiR-29c had 50 binding sites with the level of complementarity varying from 75% to 80%. So we can make a suggestion that the expression of these genes is stronger regulated by miR-29a and miR-29b, causing the atrophy, than by miR-29c, causing the hypertrophy. This hypothesis can be checked in future experiments by measuring the concentrations of these miRNAs and mRNAs in skeletal muscle tissue.