

miRNAs Expression and *in silico* Prediction of Targets Related with Resistant Exercise and Carbohydrate/Protein Supplementation

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MicroRNAs (miRNAs) are small non-coding molecules of RNA that regulates gene expression at the posttranscriptional level. To date, only a handful of studies have investigated changes on the levels of circulating miRNAs (c-miRNAs) in response to exercise. Here, we investigated the response of twelve c-miRNAs to resistance exercise (RE) and carbohydrate or carbohydrate/protein supplementation and evaluated the putative c-miRNA targets by using bioinformatics tool. Samples of blood were collected from 12 recreationally active young males before exercise, 03 and 24 hours afterwards. RT-qPCR was used for quantification of microRNAs and the relative expression data were analyzed using a two-way ANOVA with repeated measures. *In silico* prediction of miRNAs targets was performed using TargetScan version 7.1 (available at: <http://www.targetscan.org/>). As result, we observed four c-miRNAs with significant variation following RE: hsa-miR-133a, hsa-miR-503, hsa-miR-16 and hsa-miR-126. Acute ingestion of dietary protein elicited increases in the expression of hsa-miR-133 and -503 during the first 03 h after RE with decreasing levels over the following 24 h. Protein ingestion also led to decreased levels of hsa-miR-16 24 h after RE. Carbohydrate supplementation triggered increases on the levels of hsa-miR-126 shortly after RE. Among the putative c-miRNA targets, we found genes that have been implicated as regulators of cell proliferation, differentiation, and transformation (e.g. hsa-miR-133a target: FOSL2 gene - FOS-like antigen 2); cell cycle regulators (e.g. hsa-miR-503 targets: CCND2 gene - cyclin D2 and CDCA4 gene- cell division cycle associated 4); and angiogenesis (hsa-miR-16 target: C1QL gene- complement component 1; and hsa-miR-126 target: ADAM9 gene - ADAM metallopeptidase domain 9). Overall, our findings suggest a distinct profile of expression in c-miRNA between dietary carbohydrate and carbohydrate/protein supplementation following RE. As expected, the molecular response in the group that supplemented with protein was more pronounced for c-miRNAs involved in the regulation of myogenesis. Meantime, both treatments revealed a differential expression of c-miRNAs involved in angiogenesis. We conclude that *in silico* analysis can contribute to better understand the molecular mechanisms related to exercise adaptation mediated by miRNAs. Therefore, the differential expression of c-miRNAs and the effects on target genes predicted might be partially responsible for muscle hypertrophy and neovascularization following exercise.

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