G-41 Free Communication/Poster - Nutrition and Metabolic Health

Saturday, June 1, 2019, 7:30 AM - 11:00 AM

Room: CC-Hall WA2

3529 Board #217 June 1 9:30 AM - 11:00 AM

The effect of Metabolic Syndrome on Exercise Performance in American Football Players From a Mexican University

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(No relationships reported)

PURPOSE: To demonstrate the effect that the presence of the metabolic syndrome (MetSyn) has on the physical performance on American Football Players of a college team in México
METHODS: Seventy six players were included in the study, thirteen had MetSyn (21.7±1.5 yrs) and sixty three were not diagnosed with MetSyn (21.8±1.5 yrs). In order to establish a statistical significance between the physical performance and MetSyn, the data was analyzed in two different ways: With MetSyn (WMS) or absence of MetSyn (AMS) and by groups of similarity of Body Mass and type of execution in the field **Group 1** Offensive and Linemen (OL and DL) and Tight ends (TE). **Group 2** Running Backs (RB), Linebackers (LB) and Quarterbacks (QB). **Group 3** Wide Receivers (WR), Kickers (K), Strong Safeties (SS) and Cornerbacks (CB). The physical performance tests that were measured were: Maximum strength, explosive strength, Isometric hand strength, muscular resistance, power of upper and lower body, lumbar flexibility, agility, speed and cardiovascular resistance
RESULTS: The physical performance tests between WMS revealed better performance in maximum upper body strength 293.46(56.54) against the ABS group 246(36.45), explosive strength with Snatch 165(135-205) against 155(105-205) and Jerk 228.08(31.46) against *204.29(34.70)* tests, as well as muscular resistance test 9.5(1-25) against 3(0-26) repetitions. Lower athletic performance was shown in the WMS group in the speed 5.6(5.23-7.12) sec against 5.21(4.75-822) , agility 8.84(7.1-9.79) seconds against 5.21(4.75-822) and cardiovascular resistance 13.32(10-18) against 11.16(9.06-17.11) min.
CONCLUSIONS: The weight and BMI and the body fat percentage were variables that presented significance difference in the WMS group, coinciding with the authors who affirm that the weight and the percentage of fat have an influence on the physical performance. The physical performance tests in the WMS group revealed better performance in maximum chest force, explosive strength with Snatch and Jerk tests, as well as muscular resistance suggesting a possible favorable relationship to presence MetSyn. We observed a lower athletic performance in the tests of speed, agility and cardiovascular resistance in the players with the presence of MetSyn negatively relating the MetSyn with these motor skills.

3530 Board #218 June 1 9:30 AM - 11:00 AM

Inhibition Of miR-16 In Vitro Decreases Glucose Uptake And Insulin Signaling

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(No relationships reported)

Type 2 Diabetes Mellitus (T2DM) is a fast-growing epidemic and skeletal muscle insulin resistance may be the onset point in the development of T2DM. Recent data have suggested that microRNAs (miR) may play an important role in T2DM glucose intolerance. Specifically, reduced miR-16 content in muscle has been noted in human and rodent models of T2DM. However, regulation of miR-16 and its relation to muscle insulin resistance is largely unexplored.
PURPOSE: To investigate how miR-16 content affects insulin resistance and glucose regulation in myotubes during insulin resistant states.
METHODS: This study was performed in three experiments. Experiment (Ex) 1: To test if miR-16 is necessary for muscle insulin sensitivity, C2C12 myoblasts were cultured to become myotubes. Cells were transfected with a plasmid to inhibit function of miR-16. Ex 2: To test if miR-16 is sufficient to improve insulin resistance, myotubes were treated with a 1-oleoyl-2-acetyl-sn-glycerol (OAG), to simulate lipid overload-induced insulin resistance, cells were transfected with plasmid to overexpress functional miR-16. Ex 3: To test if Primary-miR16 (Pri-miR16) is differently expressed in insulin resistance state, Pri-miR16 level was measured by RT-PCR in both in vivo and in vitro models of insulin resistance. In experiment 1 and 2, glucose uptake and insulin signaling were measured by uptake of 2-NBDG (a fluorescent analog of glucose), and immunoblot of phosphorylation of AKT and IRS1. Data were analyzed by ANOVA or t-test as appropriate, significance was denoted at $p<0.05$.
RESULTS: Ex 1: Insulin-stimulated glucose uptake was ~25% lower in myotubes following miR16 inhibition ($p=0.01$). Insulin signaling was lower in myotubes with miR16 inhibition (31%, $p=0.002$). Ex 2: OAG-induced insulin resistant myotubes exhibited lower glucose uptake ($p=0.01$; 12%). However, overexpression of miR16 did not improve OAG-induced insulin resistance ($p>0.05$). Ex 3: Pri-miR16 level was not different between control and OAG.
CONCLUSION: Reduction of miR-16 content seems to be necessary for glucose handling, however, miR-16 overload is not sufficient to rescue glucose regulation and synthesis of pri-miR16 was not a culprit for reduced miR16 during insulin resistance, therefore it may be due to either degradation or export of miR16 during the miRNA process.

3531 Board #219 June 1 9:30 AM - 11:00 AM

Amelioration Of Diabetes-associated Muscle Atrophy By Transcutaneous Carbon Dioxide Exposure

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(No relationships reported)

PURPOSE: Diabetes has been known to result in attenuated growth and atrophy in skeletal muscle. Recently, it has been reported the Carbon dioxide (CO_2) exposure leads to an increase of muscle mass in normal rats. Therefore, the aim of the present study was to investigate the effects of transcutaneous CO_2 exposure with the hydrogel (eCO2GEL) on diabetic-associated muscle atrophy.
METHODS: Male Goto-Kakizaki (GK) rats were divided into control (GK) and CO_2 exposure (CO_2) groups and male Wistar rats used as a non-diabetic control. The hair on the lower limbs was shaved and the hydrogel (eCO2GEL), which can increase the absorption of CO_2 from skin, was applied. The CO_2 adaptor was attached to the limbs and sealed, and CO_2 gas was administered into the adaptor for 30 min. The CO_2 exposure was performed everyday for 8 weeks.
RESULTS: The muscle weights of soleus and tibialis anterior in the GK group decreased compared with those of the control group. CO_2 exposure attenuated decreased muscle weights in diabetes-associated muscles ($P<0.05$). In addition, the blood flow in skeletal muscle was increased by CO_2 exposure compared with non-CO_2 exposure condition ($P<0.05$). Furthermore, the level of fasting blood glucose in the CO_2 exposure group was significantly decreased compared with the GK group ($P<0.05$).
CONCLUSIONS: These results indicate that the transcutaneous CO_2 exposure may have a therapeutic potential for diabetic-associated muscle atrophy. This amelioration may associate with increased blood flow in skeletal muscle.