study, Pgrmc1 has been identified to associate with fatty acid synthesis (4). Therefore, we assumed that Pgrmc1 will associate with DCM. By feeding high-fat diet for 8 weeks and injecting streptozotocin (30mg/kg), T2D and DCM were induced. The lipid accumulation was exacerbated in T2D-induced *Pgrmc1* KO heart, and FFA level was also high. Levels of lipid metabolic genes showed the tendency for lipid accumulation and lipotoxicity, and glycolysis was induced in T2D-induced *Pgrmc1* KO heart. Though glycolysis presents higher efficiency for energy production in cardiomyopathy (5), it did not compensate the impairment of mitochondrial respiration in *Pgrmc1* KO heart. High-fat diet and streptozotocin could not be the interfering factors, because suppression of fatty acid oxidation, induction of glycolysis, and impairment of mitochondrial respiration were observed similarly in post-prandial mice which were fed with normal chow. Insulin was excluded for interfering factor as cell line with serum starvation showed mitochondrial suppression and glycolytic induction in flux analyzer analysis in *Pgrmc1* knockdown. Conversely, induction of fatty acid oxidation and suppression of glycolysis were observed in 72 h fasting of *Pgrmc1* KO heart, suggesting the nutrition is closely associated with the metabolic modulation of *Pgrmc1* on heart. This metabolic phenotype of Pgrmc1 KO heart consequently exacerbated DCM by showing high levels of fibrosis, inflammation, endoplasmic reticulum stress, and oxidative stress.

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Steroid Hormones and Receptors

STEROID BIOLOGY AND ACTION

Effects of Androgen Receptor Activation on Angiogenesis

Yen-Nien Huo, PhD.
TAIPEI MEDICAL UNIVERSITY, TAIPEI, Taiwan.

SAT-735

Abstract: The effect of androgen on angiogenesis has been documented. However, its molecular mechanisms underlying has not been well illustrated. Here, we conducted both in vitro migration assay and proliferation assay to investigate whether androgen receptor activation have any impacts on the angiogenesis. Treatment with an androgen receptor (AR) agonist, metribolone (R1881) at a range of concentrations (0.05-5 nM) or dihydrotestosterone (DHT) at a range of concentrations (0.5-2 nM) caused concentration-dependent inhibition of proliferation and migration in human umbilical venous endothelial cells (HUVEC). Blockade of the AR activity by pre-treatment with HF (5 nM), an AR antagonist, or knockdown of AR expression using the lenti-virus shRNA technique abolished the R1881-induced proliferation and migration inhibition in HUVEC, suggesting that AR receptor activation can inhibit endothelial cell proliferation and migration. To further delineate the signaling pathway involved in the AR activation-induced proliferation inhibition, our data indicate that R1881 inhibited proliferation in vascular endothelial cells through activating the AR/cSrc/AKT/p38/ERK/NFκB signaling pathway, which in turn up-regulated the expression of p53, p21 and p27 protein, and finally reduced endothelial cell proliferation. To investigate signaling pathway involved in the AR activation-induced migration inhibition, our data showed that R1881 can reduce the membrane translocation of RhoA and Rac-1, suggesting that inhibition of the RhoA and Rac-1 activity might be involved in the R1881-inhibited endothelial cell migration. Over-expression of RhoA prevented the R1881-inhibited endothelial cell migration and this effect was abolished by pre-treatment with Y27623, a ROCK inhibitor, confirming that inhibiting RhoA activity participated in the R1881-inhibited endothelial cell migration. Using the zebrafish and Matrigel angiogenesis models, we also demonstrated that R1881 inhibited angiogenesis through the AR-mediated pathway in vivo.

Bone and Mineral Metabolism

OSTEOPOROSIS: DIAGNOSIS AND CLINICAL ASPECTS

Computed Tomography Derived Skeletal Muscle Radiodensity Is Better Predictor of Muscle Power Than Skeletal Muscle Area in Community-Dwelling Older Adults.

Heewon Choi, Medical school student[1], Namki Hong, MD[2], Narae Park, Nurse[2], Chang Oh Kim, MD[3], Hyeon Chang Kim, MD[4], Jin Young Choi, MD[5], Yoosik Youm, PhD[6], Yumie Rhee, MD,PHD[2].
[1]Yonsei University College of Medicine, Seoul, Korea, Republic of,[2]Department of Internal Medicine, Severance Hospital, Endocrine Research Institute, Yonsei University College of Medicine, Seoul, Korea, Republic of, [3]Division of Geriatrics, Department of Internal Medicine, Severance Hospital, Yonsei University College of Medicine, Seoul, Korea, Republic of, [4]Department of Preventive Medicine, Yonsei University College of Medicine, Seoul, Korea, Republic of, [5]Department of Radiology, Research Institute of Radiological Science, Yonsei University College of Medicine, Seoul, Korea, Republic of, [6]Department of Sociology, Yonsei University College of Social Sciences, Seoul, Korea, Republic of.

SUN-390

Computed tomography (CT) derived skeletal muscle area (SMA) and muscle radiodensity (SMD) reflect distinctive quantitative, qualitative characteristics of skeletal muscle. Peak jump power reflects the ability to exert force within a limited time, which has greater relationship with mobility and risk of falls. CT-based SMA and SMD may have potential as useful surrogates for muscle function. However, the association of CT-based muscle parameters, especially SMD, with peak jump power has not been investigated yet. Community-dwelling older adults enrolled in the Korean Urban Rural Elderly study from 2016 to 2018 underwent

abdominal CT scans and countermovement two-legged jumping test on ground reaction force platform. SMA and SMD were measured at CT images at L3 vertebral level. Mean age of 1523 patients was 74.7 years and 65.1% was female. For peak jump force, L3SMA was stronger contributing factor than SMD (standardized beta of SMA vs. SMD = 0.16 vs. 0.08 for men; 0.12 vs. 0.05 for women; $p < 0.05$ for all). However, SMD was a better indicator of peak jump power compared to SMA in both sexes (standardized beta of SMD vs. SMA = 0.21 vs. 0.17 for men; 0.15 vs. 0.13 for women; $p < 0.05$ for all). These associations remained robust even after adjustment for age, height, weight, triglyceride, HDL cholesterol, high sensitivity C-reactive protein, and insulin resistance. One standard deviation decrease of SMD was associated with 8% elevated odds of low jump power relative to weight after adjustment for potential confounders (adjusted OR = 1.08, $p < 0.001$), whereas the association between SMA and low jump power was attenuated. SMD improved discrimination for individuals with low jump power when added to SMA and conventional risk factors (Area under the receiver-operating characteristics curve 0.732 to 0.750, p=0.006). SMD was an independent predictor of jump power with additive discriminatory value to SMA and conventional risk factors. Our findings suggest the potential complimentary role of SMD as muscle quality indicator beyond muscle mass as a surrogate for muscle function.

Adrenal

ADRENAL - TUMORS

Pattern and Spectrum of Adrenal Disorders Seen Among Adults in Southern Iraq. A Tertiary Center Experiences

Abbas Ali Mansour, MD,FRCP,FACE, Ali Hussain Ali Alhamza, MD, Ammar Mohammed Saeed Abdullah Almomin, MD, Ibrahim Abbood Zaboon, MD, Nassar Taha Yaseen Alibrahim, MD, Rudha Naser Hussein, MD, Muayad Baheer Kadhim, MD, Adel Gassab Mohammed, MD, Dheyaa Kadhim Jabbar Al-Waeli, MD, Hussein Ali Nwayyir, MD, Haider Ayad Yassin Alidrisi, MD, Ibrahim Hani Hussein, MD, Samih Abed Odhaib, MD, Mahmood Thamer Jallod Altemimi, MD, Husam Jihad Imran, MD.

Faiha Diabetes Endocrine and Metabolism Center, Basrah, Iraq.

SAT-164

Background: Adrenal disorders is rare life-threatening conditions needed high awareness for earlier diagnosis. The aim of this study is to see the pattern and spectrum of adrenal disorders in Southern Iraq.

Methods: Retrospective electronic database analysis of Faiha Specialized Diabetes, Endocrine and Metabolism Center (FDEMC) in Basrah, the largest tertiary referring Center in the Southern Iraq. Only adults 18 years and above analysed.

Results: The total referred patients for presumed adrenal disorders were 5064(6%) of 83473 new patients seen over 11 years for the period of August 2008 to August 2019.The commonest adrenal disease were due to glucocorticoids misuse in 2407/5064 (47.5%),followed by adrenal endocrine hypertension in 883/5064 (17.4%),than adrenal insufficiency in 340/5064 (6.7%), hirsutism in 264/5064 (5.2%), hypopituitarism 85/5064 (1.6%) and congenital adrenal hyperplasia in 78/5064 (1.5%).Rare causes of adrenal disorder were primary aldosteronism in 30/5064 (0.5%), Addison disease in 26/5064 (0.5%), pheochromocytoma in 19/5064 (0.4%), autoimmune polyendocrine syndromes in 19/5064 (0.4%), ACTH independent Cushing syndrome in 17(0.3%), ACTH dependent Cushing syndrome in 4(0.07%), subclinical Cushing syndrome in 4(0.07%), ectopic ACTH syndrome in 1(0.01%), adrenal cyst in 9(0.1%), adrenal myelolipoma in 5(0.09%), adrenocortical carcinoma in 3(0.05%),and paraganglioma in 2(0.04%).One of the paraganglioma were secretory. Patients characteristics for those with glucocorticoids misuse showed that female forming the bulk of cases in 1708/2407 (70.9%), and mean age of 39.5±12.3 years. Urban constitutes 1306/2407 (54.3%),and 629/2407 (26.1%) were illiterates. There were 706/2407 (29.3%) with established type 2 diabetes mellitus(with all the risks of loss of glycemic control) and glucocorticoids misuse causes 105/2407 (4.3%) incident diabetes.

Conclusion: Glucocorticoids misuse constituted the bulk of referral for adrenal disorders in Basrah. A lot of work needed to reduce the prevalence of this new high-risk iatrogenic disease.

Diabetes Mellitus and Glucose Metabolism

GESTATIONAL DIABETES, DIABETES IN PREGNANCY, AND IN UTERO EXPOSURES

Effects of Steroid Hormones on Lipogenesis and Insulin Sensitivity - an Insight into the Involvement of the Wnt Signaling Pathway

Neethu Sara Alex, B.E., Habibur Rahaman Khan, M.Sc., Rudraiah Medhamurthy, PhD.

Indian Institute of Science, Bangalore, India.

SUN-644

Gestational diabetes mellitus (GDM), a condition in which the state of pregnancy induces the development of diabetes, is characterized by heightened maternal insulin resistance. The levels of sex steroid hormones generally increase during pregnancy. It is thought that imbalance in the levels of steroids like estradiol (E_2) and progesterone (P_4) with respect to each other, may increase susceptibility towards GDM. To understand the metabolic effects of these steroids, ovariectomized (OVX) rats were treated with E_2 or P_4 at dosages mimicking the true hormonal status as in pregnancy. E_2 significantly reduced the body weight gain (145.4±1.4% to 108.3±0.8%, $p<0.001$, $n\geq12$) as well as the cumulative food intake (391.3±14.6 g to 312.5±9.0 g, $p<0.001$) over the course of the 23 day-treatment period. It also decreased the quantity of accumulated gonadal white adipose tissue (GWAT) in the body (3.3±0.2 g to 1.1±0.1 g, $p<0.001$) and repressed expression of *lpl* (1.3±0.2 fold, $p<0.05$) and other lipogenesis markers. P_4, on the other hand, enhanced *lpl* expression (3.7±0.2 fold, $p<0.001$), but did not affect the total quantity of GWAT. Further, E_2 treatment brought about an increase in the expression of insulin sensitivity markers like *insr* in the GWAT (4.5±0.6 fold, $p<0.001$) and soleus skeletal muscle (6.2±0.3 fold, $p<0.001$), as well as an increase in the protein levels of GLUT4.