density (BMD) and pelvic fractures. We have conducted a pilot cross-sectional study to establish a method of measuring pelvic BMD and to correlate BMD of the pelvis with BMD at other skeletal sites. Postmenopausal women without a history of pelvis and hip fragility fractures were enrolled. Hip, spine, and pelvis DXA scans were obtained using a Hologic DXA machine. Pelvic BMD was calculated using Hologic Research Software from 3 areas of the pelvis (R1: public symphysis, R2: inferior pubic ramus, and R3: superior pubic ramus), corresponding to common fracture locations. Pelvis BMD was the average of the 3 pelvis sites. Pelvic BMD measurement precision error was calculated using the root mean square method (Recommended by International Society of Clinical Densitometry (ISCD)). Statistical analysis was used to compare BMD at different sites. Alpha error was set at 0.05. Of 73 postmenopausal women who were enrolled in the study (average age 64 years, average 15 years postmenopausal), 3% had chronic kidney disease, 7% had type 2 DM, 3% were on corticosteroids and none were smokers. BMD of femoral neck assessed on pelvic DXA was not significantly different from femoral neck BMD measured on standard DXA (P=0.09). To assess pelvis BMD measurement precision, 15 patients underwent 3 separate pelvic DXA images after repositioning. BMD precision error was 0.011g/cm^2 which is slightly lower than the precision total hip BMD at our center (0.007 g/cm^2). BMD of R1, R2, and R3 pelvic areas were measured as 0.44±0.15, 0.41± 0.15, and 0.62 ±0.19 g/cm^2, respectively. Notably, BMD of R3 was significantly higher than the other 2 areas (P<0.001, ANOVA). Average BMD (0.49±0.14 g/cm^2) of pelvis was significantly lower than BMD of femoral neck (0.72± 0.16 g/cm^2), total hip (0.86±17 g/cm^2) and spine (0.97± 19 g/cm^2)(P <0.001). Average BMD of pelvis was significantly lower in participants with osteopenia and osteoporosis of the hip and femoral neck compared to participants with normal BMD in those locations. In summary, we report a precise method of measuring BMD of commonly fractured areas of the pelvis. Pelvis BMD is lower than hip, femoral neck, and spine. Bone density of the pelvis correlates with hip and femoral neck bone density. The results of this pilot study can be used for future studies looking at pelvic low bone density in patients with pelvic fragility fractures which could help identify patients at risk for pelvic fragility fractures and change how osteoporosis is defined based on DXA images.

Diabetes Mellitus and Glucose Metabolism

CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

FDXR Regulates Iron Metabolism and Glucose Metabolism in Liver.

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Iron is an essential cofactor for many proteins that function in electron transport or oxygen transport as heme or iron-sulfur cluster. On the contrary, iron also has the potential to cause oxidative damage if not carefully regulated and when in labial iron excess. Clinical studies show that elevated serum ferritin levels are observed in most patients with type 2 diabetes and non-alcoholic fatty liver disease (NAFLD). In this context, p53 is shown to induces some mitochondrial iron regulatory genes. The role of crosstalk between p53 and iron metabolism has not been sufficiently examined in the pathogenesis of diabetes and NAFLD.

Here, we examined the role of ferredoxin reductase (FDXR), a key mitochondrial regulator for iron metabolism, as p53-inducible gene with focusing on the hepatocyte and liver. We confirmed that p53 induced FDXR expression in HepG2 cells and SKEHP1 cells. Biochemical analysis demonstrated that FDXR regulated ROS levels via iron metabolism. *In vivo* analysis, high-fat diet activated the p53-FDXR pathway in mice liver. We generated transgene expression in mice liver using adenovirus infection carrying shRNA or CRISPR Cas9 system. Treatment with the FDXR knockdown increased hepatic iron content and aggravated glucose intolerance. Besides, forkhead box protein O1 (FOXO1), a key transcriptional factor that induces phosphoenolpyruvate carboxylase and glucose-6-phosphatase increased ratio of nuclear localization, indicating hepatic gluconeogenesis activation. Consistently, biochemical analysis in HepG2 cells demonstrated that FDXR regulated insulin-dependent FOXO1 nuclear exclusion through oxidative stress.

In conclusion, p53-inducible FDXR regulates iron metabolism and oxidative stress. FDXR inhibits iron accumulation and oxidative stress in liver and links to suppression of hepatic gluconeogenesis via insulin-dependent FOXO1 nuclear exclusion. The results

of this study provide important new insights into relationship between iron metabolism and glucose metabolism as well as potentially identify novel therapeutic targets for the treatment of diabetes and NAFLD.

Pediatric Endocrinology

PEDIATRIC PUBERTY, TRANSGENDER HEALTH, AND GENERAL ENDOCRINE

Usefulness of a LHRH Test with Low Dose of Triptorelin Pamoate in the Diagnosis of Precocious Puberty in Girls

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Objective: to determine diagnosis value of a new LHRH test for diagnosis or precocious puberty (PP) correlated with clinical and paraclinical pubertal changes. ***Methods***:79 girls under age 10 years old were referred to our laboratory with diagnosis of precocious puberty went thought a physical exam and bone age /pelvic US review to classify them clinically in probably PP or unlikely PP. A LHRH test was performed with measurement of at least 3 times including baseline measurement of gonadotrophins (LH / FSH) and