

and 2) GH receptor knockout (GHR^{-/-}) mice (GH resistance). Intestines from GH^{-/-} and GHR^{-/-} mice and their respective littermate controls at adult (12-13 mo.) and older adult (19 mo. and 24 mo., respectively) were used for this study. Both length and weight of small and large intestines were measured at the time of dissection. Six sections for the small intestine and four sections for the large intestine were prepared using a swill roll technique. Images were taken at 100x magnification with a Nikon Eclipse E600 microscope, and villus height, crypt depth, and muscle thickness were measured using ImageJ. All measurements were analyzed using Student t-test, and Cohen's d effect size was used to compare the intestinal phenotype between lines. Both GHR^{-/-} and GH^{-/-} displayed altered intestinal gross anatomy and morphology. GHR^{-/-} mice at 13 mo. of age had significantly shorter small and large intestines compared to controls. Morphologically, GHR^{-/-} mice also showed significantly decreased villus height in the duodenum and decreased villus height and crypt depth in the jejunum. Likewise, GHR^{-/-} mice at 24 mo. of age had significantly decreased intestinal length, and circumference in both small and large intestines with significantly decreased crypt depth in the ileum. Similar to GHR^{-/-} mice, GH^{-/-} mice at both 12 mo. and 19 mo. of age exhibited significantly shorter small and large intestines compared to controls with significantly smaller circumferences. Duodenum and jejunum of GH^{-/-} mice also had a significant decrease in villus height in the duodenum and jejunum. In this study, decreased GH action is apparent in significantly affecting intestinal morphology and gross anatomy. GHR^{-/-} and GH^{-/-} mice had similar changes in gross anatomy (i.e. length and circumference) and villus height of the duodenum and jejunum. Similar changes to intestinal gross anatomy and morphology were also seen across the different ages in the mice. These findings suggest that decreased GH action influences the gross anatomy of both small and large intestines, and yet has a distinctive section-dependent impact on intestinal morphology. Research needs to be conducted to understand how these structural changes relate to gut function and assess intestinal phenotype at a younger ages in these mouse lines.

Diabetes Mellitus and Glucose Metabolism

DIABETES DIAGNOSIS, TREATMENT AND COMPLICATIONS

Comparison of Phenotype and Metabolic Abnormalities Among Familial Partial Lipodystrophy Due to LMNA or PPARG Variants.

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Familial partial lipodystrophy (FPLD), a rare autosomal dominant disorder, is characterized by marked loss of subcutaneous (sc) fat from the extremities, and predisposition

to insulin resistance, diabetes mellitus, dyslipidemia and hepatic steatosis. FPLD2 and FPLD3 due to causal variants in *LMNA* and *PPARG*, respectively, are the two most common subtypes. Due to extremely rare prevalence of FPLD3 and limited reports in the literature, whether there are phenotypic differences between the two subtypes remain unclear. Therefore, we compared the anthropometric measurements and prevalence of metabolic abnormalities among 32 FPLD3 subjects (4 M, 28 F; mean \pm SD age, 41 \pm 17.2 y; body mass index (BMI), 26 \pm 4.0 kg/m²) with 271 FPLD2 subjects (66 M, 205 F; age, 37.4 \pm 17.0 y; BMI, 26 \pm 5.0 kg/m²) from two referral centers in the United States. As compared to those with FPLD2, FPLD3 subjects had borderline higher prevalence of hypertriglyceridemia (66% vs 84%; $P = 0.063$), but significantly higher prevalence of diabetes (44% vs 72%; $P = 0.004$), past history of acute pancreatitis (13% vs 52%; $P < 0.001$), and polycystic ovarian syndrome (26% vs 52%; $P = 0.011$). As compared to FPLD2, FPLD3 subjects had similar fasting triglyceride levels (median 208 vs 255 mg/dL; $P = 0.15$), but lower high-density lipoprotein cholesterol levels (median 37.5 vs 30 mg/dL; $P = 0.001$), higher fasting glucose (median 95 vs 115 mg/dL; $P = 0.05$) and HbA1c (median 5.7 vs 7.0 %; $P = 0.005$) levels. Regional body fat was measured by dual energy X-ray absorptiometry in 19 FPLD3 and 105 FPLD2 subjects. In comparison to FPLD2, FPLD3 subjects had higher total fat (median 21.6% vs 26.1 %; $P = 0.018$); upper limb fat (median 20.3% vs 27.3%; $P = 0.003$) and lower limb fat (median 16.0% vs 20.8%; $P = 0.007$). Skinfold thickness measurements by calipers also revealed less severe fat loss from both the upper and lower extremities in FPLD3 subjects compared to FPLD2 subjects. As compared to FPLD2, FPLD3 subjects had significantly higher triceps skinfold thickness (median 5.5 mm vs 7.5 mm; $P = 0.015$); and thigh skinfold thickness (median 5.8 mm vs 11.3 mm; $P = 0.001$). There were no significant differences in the prevalence of fatty liver, plasma alanine aminotransferase and aspartate aminotransferase levels in the two subtypes. We conclude that compared to FPLD2 subjects, those with FPLD3 have milder lipodystrophy phenotype but paradoxically present with more severe metabolic complications, especially diabetes, dyslipidemia and polycystic ovarian syndrome. It is likely that this discrepancy could be due to early recognition of FPLD2 because of severe fat loss versus initial diagnosis of FPLD3 subjects due to severe metabolic complications leading to discovery of milder fat loss.

Thyroid

THYROID DISORDERS CASE REPORTS II

Use of Weekly Levothyroxine Regimen for Rapid Normalization of Thyroid Hormone Levels:

A Case Report

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