SUN-638

Numerous epidemiological studies have identified a positive association of exposure to the endocrine-disrupting pesticide dichlorodiphenyltrichloroethane (DDT) and its primary metabolite dichlorodiphenyldichloroethylene (DDE) with the risk of obesity. Of particular concern is the persistent metabolic dysfunction resulting from early life DDT and DDE exposures, evidenced as obesity, glucose intolerance, dyslipidemia, and impaired thermogenesis in adult rodents. However, little is known about the developmental timing and etiopathogenesis of this long-term DDT-related metabolic phenotype. This study aimed to address these knowledge gaps by evaluating endocrine phenotypes and thermogenic parameters at two postnatal (P) time points, P0 and P12, in C57BL/6J mice. Dams were orally gavaged with p,p'-DDT (1.7 mg/kg body weight [BW]), p,p'-DDE (1.31 mg/kg BW), or organic olive oil (vehicle) daily from gestational day (GD) 11.5-P0 or P5. Infrared analysis was then performed during a cold challenge. We further attempted to rescue the cold-challenged P12 mice by administering the β3-adrenergic receptor (AR)-agonist CL316,243 (CL), a direct stimulator of brown adipose (BAT) thermogenesis. At P0, area under the curve analysis revealed higher body temperatures in both males and females exposed to DDE compared to controls during the 9 min cold challenge. In addition, DDE-exposed females lost body heat at a significantly faster rate than sex-matched controls over the 9 min cold challenge. This occurred without any treatment-related differences in resting glucose, suprascapular BAT weight, or body weight for either sex at P0. To assess BAT-autonomous response to β3-AR stimulation during a cold challenge, P12 mice were exposed to the pharmacological β3-AR agonist, or PBS control in a 2x2 exposure design and suprascapular BAT temperature was evaluated via infrared analysis of the suprascapular surface at 10 min intervals. Blood glucose, BAT weight, and BW remained equivocal across all P12 treatment groups. However, BAT temperature was significantly decreased 10 min after cold- challenge in female P12 mice with perinatal DDE-exposure when compared to sex- and perinatal treatment-matched controls. This DDE effect among P12 female mice was rescued by CL. These data suggest that thermogenic dysfunction consequent to perinatal DDE exposure is detectable during postnatal development, well before the emergence of endocrine phenotypes. The CL rescue of the BAT thermogenic impairments observed after perinatal DDE exposure are consistent with DDE toxicities upstream in the β3-AR nerve circuitry. Additionally, the data highlight the emergence of an early postnatal sex divergence in DDE-related thermogenic dysfunction. We speculate that the effects of DDE on suprascapular BAT heat production result from prenatal alterations in sympathetic circuitry.

Thyroid

HPT-AXIS AND THYROID HORMONE ACTION

Long-Term Analysis of Weight Change Following Thyroidectomy

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Introduction: Weight gain is a common source of apprehension for patients undergoing thyroidectomy. However, contradictory reports exist regarding the presence and degree of weight gain following thyroid surgery and all known studies have short term follow-up This study evaluated weight changes following total thyroidectomy (TT) and lobectomy (L) over an extended time period. **Methods:** Retrospective analysis was performed of weight changes following surgery for patients who underwent TT or L (n=387) as compared with those undergoing parathyroidectomy for primary hyperparathyroidism (n=201) in a tertiary referral hospital between 2007-2012. Clinical, demographic and pre- and post-operative weight data was collected with a median follow-up of 55.6 months. **Results:** Postoperative weight change was observed at 1, 6, 12, and 36-months in patients who underwent TT (μ=+0.21kg, μ=+1.33kg, μ=+0.59kg, μ=+0.60kg; $p<0.05$) and at 6-months for patients who underwent L (μ=+0.93kg, $p<0.05$) compared with those who underwent parathyroidectomy. Patients having TT and L showed a general trend of weight gain compared to the control group up to 108-months post-operation; however, this weight gain was non-significant ($p<0.05$). Significant postoperative weight gain was observed in patients who had TT (1-month μ=+0.40kg, 6-months μ=+2.14kg, and 12-months μ=+1.40kg) and L (6-months μ=+1.04kg) for benign conditions compared with the parathyroidectomy group. Patients who had TT gained 0.40kg more than L patients at 12-months post-op ($p<0.05$), but no significant difference existed at other time points up to 108-months. Tukey HSD post-hoc analysis showed weight gain in benign, thyroiditis, and thyroid cancer patient groups was not significantly different from 6-months to 108-months post-operation. Furthermore, neither race nor sex was correlated with weight gain. Relative risks with 95% CI for weight gain following TT and L compared to control are: 1-month TT=1.74, 0.96-3.14, L=1.59, 0.58-2.58; 6-month TT=1.27, 0.85-1.89, L=1.42, 0.85-2.11; 12-month TT=1.44, 0.92-2.28, L=1.34, 0.86-2.36; 24-month TT=1.17, 0.82-1.67, L=1.22, 0.69-1.60. In the group of patients who gained greater than 2kg, those who underwent TT had significant weight increase compared to the parathyroidectomy group at 6-months postoperatively (Mann-Whitney U, $p=0.011$). In the subgroup of patients with weight gain greater than 2kg, those who had L did not have significant weight increase at any time point. **Conclusion:** Weight change following TT when compared with parathyroidectomy is significant shortly after surgery. However, these changes are not significant at long-term follow-up.

Pediatric Endocrinology

PEDIATRIC ENDOCRINE CASE REPORTS I

Advantages of Next Generation Sequencing (NGS) in Hypophosphatemic Disorders Diagnosis. First Case of SLC9A3R1 Gene Pathogenic Variant Detected in a Pediatric Patient.

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