p=.005). CGM users also performed more frequent daily BG monitoring (5.2 \pm 1.9 vs 4.2 \pm 1.9, p=.0002) and were less likely to have HbA1c \geq 9% after 18 months (27% vs 42%, p=.03). In summary, we found distinct socio-demographic and diabetes-specific factors associated with device use in adolescents with T1D. These findings provide an opportunity to address barriers associated with device non-use in order to expand device implementation, especially in underserved adolescents with T1D.

Pediatric Endocrinology GROWTH AND GROWTH HORMONE

Comparison of Quality of Life Responses From Caregiver and Children Aged ≥7 Years Using the Quality of Life in Short Stature Youth (QoLISSY) Questionnaire, Following 12 Months of Growth Hormone Treatment With Either a Weekly Somatrogon or a Daily Genotropin Injection Schedule

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Objective: Paediatric growth hormone deficiency (pGHD) affects 1/4,000 children. Treatment with daily sub-cutaneous injections of recombinant human growth hormone (r-hGH) increase height velocity and quality of life (QoL). A recent randomised controlled clinical trial (NCT02968004) evaluated the efficacy/safety of weekly Somatrogon (hGH-CTP) and daily Genotropin in pGHD. QoL (an exploratory endpoint) was evaluated using the validated Quality of Life in Short Stature Youth (QoLISSY) questionnaire, which includes three subscales (physical, social, emotional) and total score.

Methods: The QoLISSY core module was administered to patients (aged 3-11 years [girls], 3-12 years [boys]) and parents in US, UK, Australia, New Zealand, Belarus, Russia, Ukraine and Spain, at Baseline (BL) and 12 months after treatment start. The QoLISSY-CHILD was completed by children aged \geq 7 years; QoLISSY-PARENT was completed by the Caregiver for children <7 years, and for some children aged \geq 7 years. We report here only the QoLISSY results for children aged \geq 7 years (reported from either child or parent).

Results: For Total QoLISSY-PARENT, for children aged ≥7 years in the Somatrogon group (N=26), mean scores are 53.65 (BL) and 65.52 (month 12) with mean change of 13.01 (95% Confidence Interval [CI]: 3.99, 22.02). In the Genotropin group (N=28), mean scores are 55.89 (BL) and 63.66 (month 12) with mean change of 6.60 (CI:-0.21, 13.40). For Total QoLISSY-CHILD in the Somatrogon group (N=35), mean scores are 61.48 (BL) and 74.69 (month 12) with mean change of 13.00 (CI: 5.81, 20.19). In the Genotropin group (N=35), these scores are 60.96 (BL) and 69.03 (Month 12) with mean change of 7.84 (CI: 2.71, 12.97). Scores of >70 indicate a good QoL.

Conclusions: QoL in children aged ≥ 7 years improved, following 12 months of either treatment, whether this

was reported by caregiver or child. However, these data show that the baseline scores and 12 month scores from the QoLISSY-PARENT in both treatment groups were numerically lower than those reported by the child. This is consistent with the literature¹, in which the caregivers generally report lower QoL scores on behalf of the child.

¹Explaining parent-child (dis)agreement in generic and short stature-specific health-related quality of life reports: do family and social relationships matter? Quitmann et al *Health and Quality of Life Outcomes* 2016 vol 14, Article 150

Pediatric Endocrinology GROWTH AND GROWTH HORMONE

Diagnosis of Childhood and Adolescent Growth Hormone Deficiency Using Transcriptomic Data Terence Garner, MSc¹, Adam Stevens, PhD¹,

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Background: We have shown that gene expression (GE) data have promise as a novel tool to aid in the diagnosis of childhood growth hormone deficiency (GHD)¹. Our previous study compared GE data in children with GHD to healthy control children of normal stature. The aim of this study was to assess the utility of GE data in the diagnosis of GHD in childhood and adolescence using non-GHD short stature children as a control group.

Methods: GE data were obtained from patients undergoing growth hormone stimulation testing via a sample of blood taken at the start of the test. Arginine and glucagon stimulation tests with a cut-off for peak GH of <7mcg/L (IDS iSYS assay) were used for the diagnosis of GHD. GE was assessed in peripheral blood mononuclear cells via RNAseq using the Illumina HiSeq 4000 platform. Data were taken for the 271 genes whose expression was utilised in our previous study. The synthetic minority oversampling technique was used to balance the dataset and a random forest algorithm applied to predict GHD status. Boruta was used to assess which of the genes were contributing to the predictive capacity.

Results: Twenty-four patients were recruited to the study, with eight subsequently diagnosed with GHD. Of the eight patients diagnosed with GHD, three had two stimulation tests and five had one stimulation test with anterior pituitary hypoplasia (in addition one patient had an arachnoid cyst and another a thin stalk). Median (range) peak GH was 2.5 (0.1 - 5) mcg/L in the GHD group and 11.0 (7.4 - 31) mcg/L in the non-GHD group.

There were no significant differences in gender, age, auxology (height SDS, weight SDS, BMI SDS) or biochemistry (IGF-I or IGFBP-3 SDS) between the GHD and non-GHD subjects. 82 of the 271 genes used in our previous study were above the threshold of detection for RNA-seq in this study. A random forest algorithm using these 82 genes gave an AUC of 0.97 (95% CI 0.93 - 1.0) for the diagnosis of GHD. Boruta was able to identify 65/82 genes with predictive capacity greater than permuted data within the dataset. Using a gene ontology approach the top fifty