indicators of suboptimal adherence. These results can be used to identify children needing additional medical or other support to reach optimal adherence and therefore optimal clinical outcomes.

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Anastrozole Improves Near Adult Height in Boys With Compromised Height Potential, as Monotherapy or in Combination With a GnRH Analogue

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Background: Bone maturation depends mainly on locally produced estrogens by aromatization. Third generation aromatase inhibitors (AIs) are being widely used off-label to improve predicted adult height (PAH) in boys as well as in girls, either as monotherapy or in combination with growth hormone and/or puberty inhibition. They induce reverse binding inhibiting the activity of aromatase (a cytochrome P450 enzyme), which catalyzes the conversion of androstenedione and testosterone to estrone and estradiol, respectively. While numerous studies have shown that AIs delay bone maturation and improve PAH, data on near-adult height (NAH) of children treated with AIs are lacking. **Aims:** To compare results on NAH of boys treated with anastrozole either as monotherapy or in combination with pubertal inhibition (for at least 1yr at onset). **Methods:** 159 boys with advanced bone age (BA) and PAH <170 cm that received anastrozole 1 mg/day p.o. either as monotherapy (n=76, group A) or as co-therapy with a GnRH analogue for at least 1yr and then as monotherapy (n=83, group B) until bone age of 15-16 yrs were included. Data on boys that reached NAH (BA at least 16 yrs with height velocity <2 cm/yr) were analyzed: group A, n=16 with PAH 167.3 and TH 170.9 and group B, n=10 with PAH 165.5 and TH 171.7 cm. Measurements were made on the same height meter by the same examiner. The choice of therapeutic intervention was made randomly. Groups A and B did not differ in terms of age at intervention onset, TH or PAH. During treatment, they underwent a 6-month follow-up that included clinical examination, BA, and laboratory tests at 8:00 hrs (general blood count, lipid chart, LH, FSH, testosterone, estradiol, estrone, and complete calcium metabolism), with lumbar spine DEXA (Dual Energy X-ray Absorptiometry) and X-ray performed annually. **Results:** The duration of anastrozole treatment was 3.9 yrs in group A, and 4.6 yrs in group B (where the GnRHa was administered for at least 1 yr) and the median age at intervention onset was 11.04 and 11.8 yrs, respectively. Both groups had a statistically significant gain in NAH with no difference between them: for group A 3.6 cm (+0.53 SD, p=0.002) and for group B 4.8 cm (+0.71 SD, p=0.0007). Thus, distance from TH was finally 0 cm for group A and -1.5 cm (0.19 SD) for group B. According to the definition of NAH, the adult height of the two groups is expected to be about 2% higher. Follow-up showed no side effects on their biochemical or lipid profile, bone density and vertebral architecture. **Conclusions:** Anastrozole therapy is safe and effective in improving adult height in boys with advanced puberty and poor height prediction, either as monotherapy or in combination with pubertal inhibition.

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Characteristics Associated With Diabetes Device Use Among Youth With Type 1 Diabetes

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Type 1 diabetes (T1D) is a common illness of childhood, requiring lifelong, daily complex management to prevent acute and chronic complications. Studies have shown that use of insulin pumps and continuous glucose monitors (CGM) offers benefit for glycemic control. However, such device use is not universal in adolescents. We aimed to compare baseline socio-demographic and diabetes characteristics associated with diabetes technology (pump and CGM) uptake and continued use in 13-17 year old teens with T1D. Data were derived from a multicenter clinical trial aimed at optimizing self-care and glycemic control in teens with T1D. Socio-demographic and diabetes data were collected quarterly by parent-youth interview and electronic medical record review prospectively over 18 months. Chi-square and t-tests compared characteristics of device and non-device users (pump vs no pump; CGM vs no CGM). The study sample comprised 301 teens (41% male) with mean±SD age 15.0±1.3 years, T1D duration 6.5±3.7 years, and A1c 8.5±1.1%. Most (65%) used a pump at entry or initiated pump therapy during the study; 35% used injection therapy at entry or stopped pump therapy. In contrast, 27% used a CGM at entry or started a CGM during the study, while 73% never used or stopped using CGM. Device users at entry and those who began use had similar characteristics, as did those who never used and those who discontinued device use. Pump users were more likely to use CGM than non-pump users (36% vs 10%, p<.0001). Neither age, sex, nor T1D duration was related to pump or CGM use. Pump users (vs non-pump users) were less likely to have another medical condition (44% vs 59%, p=.01) and more likely to be non-Hispanic white (83% vs 61%, p=.0001); have family annual household income ≥\$150,000 (34% vs 19%, p=.0003), private health insurance (92% vs 74%, p<.0001), a parent with college education or higher (67% vs 46%, p=.0005), and a 2-parent household (88% vs 78%, p=.03). Pump users also had lower z-BMI (0.73±0.80 vs 0.97±0.79, p=.01), performed more frequent daily BG monitoring (4.8±1.8 vs 3.9±2.0, p<.0001), and were less likely to have HbA1c ≥9% at initial and last visits (25% vs 43%, p=.005; 31% vs 49%, p=.01). CGM users (vs non-CGM users) were more likely to be non-Hispanic white (88% vs 70%, p=.009); have family annual household income ≥\$150,000 (44% vs 23%, p=.0001), a parent with college education or higher (78% vs 53%, p=.0004), and private health insurance (95% vs 82%,

p=.005). CGM users also performed more frequent daily BG monitoring (5.2±1.9 vs 4.2±1.9, p=.0002) and were less likely to have HbA1c ≥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.

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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 ≥7 years; QoLISSY-PARENT was completed by the Caregiver for children <7 years, and for some children aged ≥7 years. We report here only the QoLISSY results for children aged ≥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 literature[1], in which the caregivers generally report lower QoL scores on behalf of the child.
[1]Explaining 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

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Diagnosis of Childhood and Adolescent Growth Hormone Deficiency Using Transcriptomic Data

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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 RNA-seq 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