Background: Chemerin is an adipocytokine which plays a great role in metabolism of carbohydrates. Chemerin concentration correlates positively with body mass (BM). GH deficiency (GHD) is associated with excess of abdominal fat tissue also in patients with normal BMI. **Objective and hypotheses:** To estimate the chemerin concentration and its correlation with BM and carbohydrate metabolism in non-obese, prepubertal children with isolated GHD before (GHD untreated group) and after 6 months of GH therapy (GHD after 6 months group). **Method:** 32 (22 boys, ten girls) children with GHD (mean height 117.9 cm, -2.77 SD, mean BMI -0.75 SD), mean age 8.87 years. Control group (CG): 18 (11 boys, nine girls) age matched healthy children (mean height 125.8 cm, -0.93 SD, mean BMI -0.28 SD). Serum fasting chemerin was measured in all. In GHD untreated and GHD after 6 months the following exams were done: body composition (bioimpedancy), fasting serum glucose and insulin. Fasting glucose/insulin ratio was calculated. **Results:** The mean serum concentrations of chemerin did not differ significantly between CG, GHD untreated and GHD after 6 months. FGIR was significantly higher in GHD after 6 months comparing to FGIR in GHD untreated (0.076 vs 0.090, $P < 0.01$). In GHD untreated chemerin concentration correlates positively with BM (both with lean and fat mass) and FGIR ($r = 0.35$ and $r = 0.40$ respectively). Δ chemerin (chemerin level GHD after 6 months – chemerin level GHD untreated) correlates negatively with FGIR and negatively with chemerin level in GHD untreated ($r = -0.57$ and $r = -0.59$). Δ chemerin correlates positively with Δ SD BM ($r = 0.44$). **Conclusion:** Chemerin concentration correlates positively with BM and FGIR in prepubertal non-obese children with GH deficiency before start of GH therapy whereas Δ chemerin correlates negatively with FGIR and Δ SD BM. It seems that Δ chemerin levels may influence carbohydrate metabolism during GH therapy in GHD children. **Funding:** This work was supported by Department od Health Sciences, University od Jan Kochanowski, Kielce, Poland (internal grant).

P2-410

Response to GH Treatment in the Very Young with GH Deficiency

Semra Cetinkaya[a], Sukran Poyrazoglu[b], Zehra Aycan[a,c], Zeynep Siklar[d], Merih Berberoglu[d], Zeynep Atay[e], Abdullah Bereket[e], Oya Ercan[f], Eda Mengen[g], Fatma Demirel[c,h], Sukran Darcan[i], Feyza Darendeliler[b]

[a]Dr Sami Ulus Obstetrics and Gynecology, Children's Health and Disease Training and Research Hospital, Ankara, Turkey; [b]Istanbul Faculty of Medicine, Pediatric Endocrinology Unit, Institute of Child Health, Istanbul University, Istanbul, Turkey; [c]Faculty of Medicine, Yildirim Beyazit University, Ankara, Turkey; [d]Department of Pediatric Endocrinology, Ankara University School of Medicine, Ankara, Turkey; [e]Department of Pediatric Endocrinology, Marmara University, Istanbul, Turkey; [f]Cerrahpasa Medical Faculty, Pediatrics Department, Pediatric Endocrinology and Adolescent Divisions, Istanbul University, Istanbul, Turkey; [g]Faculty of Medicine, Division of Pediatric Endocrinology, Cukurova University, Adana, Turkey; [h]Department of Endocrinology, Ankara Children's Hematology and Oncology Training Hospital, Ankara, Turkey; [i]Department of Pediatric Endocrinology, Ege University School of Medicine, Izmir, Turkey

Aim: Data on response to GH treatment in the very young children with GH deficiency is scarce. The aim of this study was to evaluate the growth response in such children in a national multicentre study and to analyse the factors affecting the growth response. **Materials and methods:** In this study, we retrospectively evaluated the files of GH deficiency patients who had started GH treatment between 0–3 years of age who were being followed in 14 different centres from different regions of Turkey between 19 February 2014 and 23 October 2014. The study was approved by the Clinical Studies Ethics Committee. All collected data were obtained from patient hospital records. An electronic case recording form (CRF) was created. The CRF covered demographic features, as well as clinical and laboratory findings of the patients. The CRF was uploaded to the website of FAVOR Web Registry System (www.favorsci.org). Data entered in the registry were also checked for consistency by one of the authors (SÇ). The time given for patient enrolment was eight months. By the end of the deadline the collected patient record data were entered to Microsoft Excel database and subsequently transferred to SPSS for Windows statistical software for statistical analysis. The duration of GH treatment was accepted to be at least 12 mo. The patients were further subdivided according to isolated vs multiple pituitary hormone deficiency (MPHD) and age at onset of therapy: 0–12 months vs 12–36 months. Patients with MPHD received appropriate replacement therapy. **Results:** There were 42 patients with GH deficiency (23 males, 19 females) with a peak GH response (after GH stimulation test or at hypoglycaemia) of 0.69 ± 0.14 ng/ml. 30 had MPHD and 12 had isolated GH deficiency. The mean age at onset of GH therapy was 11.2 ± 1.03 mo. Mean GH dose used was 31.7 ± 1.4 µg/kg per day. Results of GH therapy over 1 year are shown on the Table. There was a significant increase in length SDS ($P = 0.000$), weight SDS ($P = 0.000$) and BMI SDS ($P = 0.02$) over 1-year of therapy. Height velocity over one year showed positive correlation with weight increment ($r = 0.38$), but did not show correlation with birth weight, peak GH level, GH dose and BMI. In MPHD Group 1st year response was significantly higher (16.5 ± 4.2 cm) than in the isolated GH deficiency group (12.8 ± 3.3 cm) ($P = 0.014$). In the group started GH between 0–12 months the response (18.0 ± 4.2 cm) was higher than in the ones started between 12–36 months (13.3 ± 3.1 cm) ($P = 0.013$). There was no difference between girls and boys with respect to the growth response. Neither was a difference in growth response between those with minipuberty or not. Multiple regression analysis did not reveal a significant parameter to explain the differences in growth response. **Conclusion:** Among children with GH deficiency, young children with MPHD respond better than isolated GH deficiency and those children aged between 0–12 months at onset of therapy respond better than 12–36 months children. The most significant factor in growth response was weight gain.