Background

In Obesity is usually characterized by high prevalence of 25OH vitamin D (25OHD) deficiency. This might be due to either volumetric dilution of vitamin D in the large fat mass or its increased uptake by adipose tissue. Insulin resistance has been described to negatively affect vitamin D levels in obese subjects. To our knowledge, a systematic study on 25OHD levels in Prader-Willi syndrome (PWS), a genetic disorder associated with severe obesity, is not yet available.

Objective and hypothesis

To analyze the 25OHD levels in a pediatric PWS population in comparison with a group of matched controls (CNT), evaluating the possible correlation with insulin resistance, bone mineral density, body composition and GH therapy (GHT).

Methods

52 PWS (25 males; 73% overweight or obese; aged 8-18 years) and 110 CNT (57 males; 95% overweight or obese; aged 9-18 years) gender-, age- and BMI-SD matched were included. None of them was on calcium or vitamin D. 20 PWS were undergoing GHT and 32 was previously treated (stopped at least 12 months before starting the study). Plasma calcium, phosphorus, 25OH vitamin D, PTH, ALP, glycemia and insulin were collected, and HOMA-IR was calculated. All patients were studied using DXA scan (Hologic) in order to obtain bone parameters (lumbar and whole body areal BMD =LabMD and wb BMD; volumetric Lumbar BMD =vBMDAD; and normalized for height wb BMD =n-wbBMD; lumbar Z score =LZscore), and body composition parameters (fat mass and fat free mass expressed as percentage of total body weight =FM% and FFM%); FFM/FM and trunk fat/FM ratio). Statistical analysis was performed using SPSS package 21.0 for Windows; p value <0.05 was considered significant. Local Ethical Committee approved the protocol and informed consent was obtained by parents.

Results

The more relevant findings are reported in the table. In particular 16 PWS (30.7%) and 27 CNT (24.3%) had low 25OHD levels (p=NS). As expected, mean pubertal stage was statistically different, due to delayed puberty in PWS subjects. PWS showed lower HOMA-IR, LZscore, LabMD, wb BMD and n-wb BMD. FFM/FM ratio was not statistically different, while trunk/FM ratio was lower in PWS. Finally, PWS undergoing GHT showed mean 25OHD 28.9±12.5 ng/ml vs previous treated GHT 26.9±12.6 ng/ml (p=NS).

Univariate analysis (Pearson’s) showed in both groups a negative correlation between 25OHD and fat mass% (FM%) (PWS r=-0.3038; p=0.031; CNT r=-0.200; p=0.04), while HOMA-IR, bone density parameters, pubertal stage did not showed correlation with 25 OHD levels in both groups. In PWS GHT did not correlate with 25OHD levels. Pubertal stage showed positive correlation with bone parameters in PWS as such as in CNT group. In PWS, GHT was positively correlated with LabMD, wbBMD and LZscore.

When inserted in Multivariate regression in PWS only FM% remain as negative predictor of vitamin D levels (B =-0.420; p=0.010).

Conclusions.

Our data showed that PWS had similar values of 25OHD compared to age-, gender- and BMI-SD matched CNT. Insulin resistance does not seem to influence 25OHD levels. Bone parameters seems to be influenced by pubertal development and GHT, but not by vitamin D levels. As already described, FM seems to be the only parameter influencing 25OHD levels in PWS population. Finally, GHT does not seem to influence 25OHD levels in PWS.