Research Square

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Height-Adjusted Bone Mineral Density and Growth in Children with Mucopolysaccharidosis: A Single-Center Retrospective Cohort with Preserved Endocrine Axes

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Abstract

Objective.

Skeletal involvement in the mucopolysaccharidoses (MPS) is well characterized, but two questions remain open: how to read bone mineral density (BMD) independently of skeletal dysplasia, and whether the endocrine axes are affected. We described growth, standard and height-adjusted BMD, and endocrine parameters in a pediatric MPS cohort from a region where consanguinity is common.

Methods.

We reviewed the records of 42 patients younger than 18 years followed at a single center between January 2020 and December 2023, reporting according to STROBE. Anthropometry was converted to age- and sex-specific z-scores. Areal BMD from lumbar DEXA was adjusted for height-for-age z-score using the Zemel method. Annual growth velocity was calculated only for patients with a follow-up interval of at least nine months. Puberty was staged clinically by Tanner criteria. Calcium metabolism, 25(OH)D, and the thyroid, adrenal and gonadal axes were assessed. Analyses were exploratory, and no correction for multiple comparisons was applied.

Results.

Girls made up 52.4% of the cohort; median age was 7.3 years (1.7–17.0) and median age at diagnosis 2.5 years, and 88.1% were receiving enzyme replacement therapy (ERT). Mean baseline height SDS was − 2.76 ± 2.54, and 61.9% met the criterion for short stature. Height SDS fell over follow-up (median change − 1.01 SDS; Wilcoxon $p < 0.001$). Among the 34 patients with both measurements, low BMD by standard DEXA dropped from 64.7% to 20.6% after height adjustment. Parathyroid hormone (PTH) tended to be higher in short-statured patients (median 50.0 vs 38.0 pg/mL; $p = 0.054$), whereas vitamin D overlapped between the two groups (17.9 vs 18.2 ng/mL; $p = 0.805$). In patients with adequate follow-up, annual growth velocity correlated strongly with chronological age (rho = − 0.61) and with baseline height SDS (rho = 0.59; both $p < 0.001$). Thyroid, adrenal and gonadal parameters showed no clinically meaningful deviation.

Conclusion.

In subtypes with marked skeletal dysplasia, standard DEXA systematically underestimates BMD; bone assessment in MPS should rest on the height-adjusted score. The tendency for PTH to rise independently of vitamin D in short stature is a hypothesis-level finding that needs confirmation. The broadly preserved endocrine axes point to the skeletal–growth axis as the primary problem in MPS.

Introduction

The mucopolysaccharidoses are rare metabolic disorders caused by an inherited deficiency of lysosomal hydrolases, which leads to the accumulation of glycosaminoglycans (GAGs) in tissues. Depending on the affected enzyme, heparan, dermatan, keratan or chondroitin sulfate builds up in lysosomes, disrupts cell function and drives progressive organ damage [1]. The clinical picture is multisystemic; coarse facies, hepatosplenomegaly, corneal clouding, valvular disease and dysostosis multiplex are prominent in types I, II and VI [1, 2]. In most patients, growth failure is the first diagnostic clue.

Prevalence differs between populations. Meikle and colleagues reported a combined birth prevalence of about 1 in 5,000 for all lysosomal storage disorders [3]. The high rate of consanguineous marriage in Türkiye makes autosomal recessive diseases more common than expected; this rate exceeds the national average in Southeastern Anatolia, and the number and phenotypic range of MPS cases in the region follow directly from that demography [4]. In Turkish multicenter series the consanguinity rate reaches 79%, with a family history in more than half of the cases [5].

Despite the detailed description of skeletal involvement, the interpretation of BMD in MPS remains contested. Standard DEXA reports low BMD in 40–60% of patients [5, 6, 7], yet how much of this reflects genuine mineral loss rather than the effect of short stature on an areal measurement is unclear. Data on the endocrine axes are scarcer still, and most evidence comes from case reports or small subtype-specific series. Regional data that describe growth, bone and endocrine parameters together in the same cohort are almost absent.

This study has three aims. The first is to show, in a regional pediatric cohort marked by frequent consanguinity, wide phenotypic range and near-universal ERT, how far height adjustment shifts the clinical decision threshold, particularly in subtypes such as type IVA where skeletal dysplasia is most severe. The second is to combine bone data with concurrent growth and endocrine measurements, in order to distinguish whether growth failure in MPS reflects an endocrinopathy or direct skeletal involvement. The third is to test whether the link between short stature and PTH is independent of vitamin D status. Subtype-level observations are hypothesis-generating only, given the small number of patients per cell.

Materials and Methods

Study design and population. This was a single-center, retrospective cohort study, reported in line with the STROBE recommendations for observational studies. Records of patients followed with a diagnosis of MPS at the Pediatric Endocrinology and Metabolism outpatient clinic of Gaziantep University Faculty of Medicine between January 2020 and December 2023 were retrieved from the institutional electronic system. The study was approved by the local Clinical Research Ethics Committee (approval no: 169; date: 07.06.2023). The Declaration of Helsinki was followed; individual consent was not required given the retrospective design.

Inclusion criteria were a diagnosis of MPS confirmed biochemically and/or genetically, age below 18 years at the last evaluation, and available laboratory and anthropometric data. Patients aged 18 years or older at first evaluation, and those with missing data severe enough to limit the primary outcomes, were excluded. Forty-two patients met these criteria (patient selection is summarized in Figure S1). All 42 contributed anthropometric data; analyses of annual growth velocity were restricted to the 39 patients with a follow-up interval of at least nine months, and DEXA-based analyses were limited to patients with available scans. Because MPS is rare and the design retrospective, all eligible patients in the period were included and no a priori sample-size calculation was performed.

Data collection and parameters. All data had been recorded during routine follow-up; no additional procedure was performed for research. Chronological age was calculated as the interval between the last clinical evaluation and the date of birth. Height, weight and body mass index (BMI) were converted to age- and sex-specific z-scores using the ÇEDD-Çözüm (ChildMetrics) application, which is based on the 2015 Turkish national reference of Neyzi and colleagues [8]. Pubertal status was determined clinically by Tanner staging; stage 1 was taken as prepubertal and stages 2–5 as pubertal. Bone age was assessed from a hand–wrist radiograph using the Greulich–Pyle atlas.

Annual growth velocity was calculated as the difference in height between two consecutive evaluations divided by the elapsed time. Because the intervals varied widely (0.9–106 months) and very short intervals inflate the annualized rate artificially, growth velocity was defined only for patients with a follow-up interval of at least nine months; shorter intervals were not treated as valid.

Biochemical assessment included total calcium, inorganic phosphorus, ALP, intact PTH, 25(OH)D and fasting glucose. For 25(OH)D, > 20 ng/mL was taken as normal, 12–20 ng/mL as insufficiency and < 12 ng/mL as deficiency [9]. Thyroid function was assessed with free T4 and TSH; IGF-1, ACTH, cortisol, LH, FSH, estradiol (in girls), total testosterone (in boys) and anti-Müllerian hormone (AMH) were also measured.

Bone mineral density. Areal BMD was measured at the lumbar spine (L1–L4) by DEXA and converted to an age- and sex-specific z-score. To remove the misleading effect of MPS-related short stature on the areal measurement, the height-for-age adjustment recommended by the ISCD for short children was applied using the method of Zemel and colleagues, yielding a height-adjusted z-score (aBMD-HAZ) [5, 7, 10]; the same approach has been used in MPS cohorts by Lin and colleagues [6]. All measurements were performed on a single densitometer.

Statistical analysis. Analyses were performed with SPSS 26.0 (IBM Corp., Armonk, NY). Normality was tested with the Shapiro–Wilk test. Because of skewed distributions and extreme z-scores, continuous variables are given as median (min–max), with mean ± SD added for approximately normally distributed variables. Two-group comparisons used the Mann–Whitney U test, categorical variables the chi-square or Fisher exact test, paired height SDS the Wilcoxon signed-rank test, and correlations the Spearman coefficient. Factors associated with height-adjusted BMD were examined by linear regression; because of the small sample, the multivariable model was limited to age, puberty and baseline height SDS, and

subtypes with only 2–3 patients were not entered. The significance threshold was $p < 0.05$. Since the study was exploratory, no formal correction for multiple comparisons was applied, and subgroup and correlation findings should be regarded as hypothesis-generating.

Results

General characteristics; The general features of the cohort are shown in Table 1. Girls made up 52.4% of patients, and median age was 7.3 years (1.7–17.0). Diagnosis had been made at a median age of 2.5 years, with a median follow-up of 37.1 months. Type VI predominated (57.1%), followed by type IVA (23.8%); type I accounted for 9.5% and types II and III for 4.8% each. ERT was used by 88.1% of patients.

Table 1
General characteristics and bone health parameters (n = 42)

Variable	Value
Sex — female, n (%)	22 (52.4)
Age, years — median (min–max)	7.3 (1.7–17.0)
Age at diagnosis, years — median	2.5 (0.7–9.8)
Follow-up, months — median	37.1
MPS type VI, n (%)	24 (57.1)
MPS type IVA, n (%)	10 (23.8)
MPS type I, n (%)	4 (9.5)
MPS type II, n (%)	2 (4.8)
MPS type III, n (%)	2 (4.8)
Pubertal (stage ≥ 2), n (%)	7 (16.7)
Enzyme replacement therapy, n (%)	37 (88.1)
Baseline height SDS — mean ± SD (median)	−2.76 ± 2.54 (− 2.42)
Short stature (height SDS < − 2), n/N (%)	26/42 (61.9)
Annual growth velocity (≥ 9 months), cm/year — median	3.3 (n = 39)
Standard DEXA z-score — mean ± SD (median)	−3.07 ± 2.26 (− 3.3)
Height-adjusted DEXA — mean ± SD (median)	−1.01 ± 1.29 (− 1.1)
Standard DEXA ≤ − 2 (paired), n/N (%)	22/34 (64.7)
Height-adjusted DEXA ≤ − 2 (paired), n/N (%)	7/34 (20.6)
Vitamin D deficiency (< 12 ng/mL), n (%)	11 (26.2)
Vitamin D insufficiency (12–20), n (%)	14 (33.3)
Normal vitamin D (> 20), n (%)	17 (40.5)
Vitamin D, ng/mL — median	17.9 (10.0–44.6)
PTH, pg/mL — median	42.5 (17.0–108.5)
Calcium, mg/dL — median	9.6 (8.8–10.8)
Phosphorus, mg/dL — mean ± SD	4.41 ± 0.36
ALP, U/L — mean ± SD	198 ± 74

Growth; Mean baseline height SDS was − 2.76 ± 2.54, and 61.9% of patients met the short-stature criterion. In the 42 patients with paired baseline and last height SDS, the value fell from − 2.76 ± 2.54 to − 4.11 ± 3.08, with a median change of − 1.01 SDS. Height SDS decreased in 35 patients, increased in 6 and was unchanged in 1 (Wilcoxon $p < 0.001$; Fig. 1). This pattern shows that linear growth failure progressed despite ERT.

Short-stature comparisons are shown in Table 2. Short-statured patients were older (median 8.6 vs 4.0 years; $p = 0.004$) and had a markedly lower growth velocity (2.8 vs 5.4 cm/year; $p = 0.006$). This fits the view that the height deficit accumulates over time in MPS.

Table 2
Comparison by short-stature status.

Variable	**Short (n = 26)**	**Normal (n = 16)**	**p**
Age, years — median	8.6	4.0	0.004
Annual growth velocity, cm/year — median	2.8	5.4	0.006
Standard DEXA z-score — median	−3.90	−1.00	< 0.001
Height-adjusted DEXA — median	−1.19	−0.24	0.026
Vitamin D, ng/mL — median	17.9	18.2	0.805
PTH, pg/mL — median	50.0	38.0	0.054

Bone mineral density. Mean standard DEXA z-score was − 3.07 ± 2.26 and the height-adjusted z-score was − 1.01 ± 1.29. Among the 34 patients with both measurements, standard DEXA ≤ − 2 was present in 64.7% (22/34), falling to 20.6% (7/34) after height adjustment. The shift is shown at the individual level in Fig. 2; the adjusted z-score rose clearly in most patients.

In the short-stature group, standard DEXA was markedly lower (median − 3.90 vs − 1.00; $p < 0.001$), but the difference in height-adjusted DEXA fell to trend level (− 1.19 vs − 0.24; $p = 0.026$). Standard DEXA differed across subtypes ($p = 0.023$); all evaluable type IVA patients had DEXA ≤ − 2, whereas the between-subtype difference disappeared after height adjustment ($p = 0.23$) (Table 3). The subtype data rest on few patients per cell and are given for description only.

Table 3
Bone and metabolic parameters by MPS subtype

Variable	Type I (n = 4)	Type II (n = 2)	Type III (n = 2)	Type IVA (n = 10)	Type VI (n = 24)	p
Baseline height SDS	−1.66	−0.15	1.36	−2.49	−3.44	0.037
Standard DEXA z-score	−1.60	−0.10	3.20	−4.00	−3.35	0.023
Height-adjusted DEXA	−1.00	0.62	0.95	−1.76	−1.07	0.23
Vitamin D, ng/mL	18.2	11.1	10.1	25.2	15.6	0.059
PTH, pg/mL	41.3	35.8	91.8	37.9	45.2	0.166

Vitamin D, PTH and endocrine parameters. Vitamin D deficiency was found in 26.2% and insufficiency in 33.3%; only 40.5% were normal [9]. Vitamin D levels overlapped between the short and normal-stature groups (median 17.9 vs 18.2 ng/mL; p = 0.805). PTH, by contrast, tended to be higher in the short-stature group (50.0 vs 38.0 pg/mL; p = 0.054). With vitamin D equal between the two groups, the tendency of PTH to separate suggests that the difference may relate to MPS-specific skeletal dysplasia and increased bone turnover rather than to vitamin D insufficiency [5]. The subtype data, however, do not fully agree with this reading: the highest PTH was seen in the two type III (Sanfilippo) patients, who were not short-statured but had marked vitamin D deficiency (median PTH 91.8 pg/mL; vitamin D 10.1 ng/mL), and the between-subtype PTH difference was not significant (p = 0.166). This inconsistency keeps the group-level, vitamin-D–independent PTH tendency at the level of a hypothesis. Calcium and phosphorus were within the reference range in all patients.

The endocrine axes are summarized in Table 4. Thyroid function was within euthyroid limits (median TSH 2.17 mIU/L; free T4 0.96 ng/dL). Adrenal axis values were normal. The wide spread of gonadotropins and sex steroids reflects the pubertal heterogeneity of the cohort. IGF-1 showed no systematic decline that would suggest growth hormone deficiency despite the marked short stature. No clinically meaningful endocrinopathy was found in the thyroid, adrenal or gonadal axes.

Table 4
Endocrine axis parameters

Parameter	Median (min–max)	n
IGF-1, ng/mL	57.5 (23.5–185.0)	18
TSH, mIU/L	2.17 (0.70–3.97)	38
Free T4, ng/dL	0.96 (0.67–1.38)	38
ACTH, pg/mL	16.8 (6.9–48.5)	39
Cortisol, µg/dL	7.48 (2.56–19.33)	33
LH, IU/L	0.22 (0.04–3.83)	31
FSH, IU/L	1.88 (0.30–6.64)	29
Estradiol (girls), pg/mL	7.8 (0.0–103.3)	10
Testosterone (boys), ng/dL	0.10 (0.10–310.1)	15
AMH, ng/mL*	24.4 (6.0–24.5)	7

A portion of the AMH values clustered at the upper assay limit and are therefore given for description only.

Factors associated with annual growth velocity. In the 39 patients with an adequate follow-up interval, annual growth velocity correlated strongly and inversely with chronological age (rho = − 0.61; $p < 0.001$) and strongly and positively with baseline height SDS (rho = 0.59; $p < 0.001$). The decline of growth velocity with age reflects the expected developmental pattern; the link with baseline height SDS shows that velocity falls as growth failure deepens. There were positive correlations with standard DEXA (rho = 0.62; $p < 0.001$) and ALP (rho = 0.47; $p = 0.002$), and an inverse correlation with PTH (rho = − 0.32; $p = 0.050$). No relationship with vitamin D was found ($p = 0.54$) (Table 5).

Table 5
Spearman correlations of annual growth velocity (≥ 9-month follow-up, n = 33–39).

Variable	Spearman rho	p
Age	−0.61	< 0.001
Baseline height SDS	0.59	< 0.001
Standard DEXA z-score	0.62	< 0.001
ALP	0.47	0.002
PTH	−0.32	0.050
Vitamin D	0.10	0.54

Factors associated with height-adjusted DEXA. On univariable analysis, baseline height SDS was significantly related to height-adjusted DEXA (B = 0.21; 95% CI 0.05–0.37; p = 0.013; Fig. 3). In the multivariable model adjusted for age and puberty, baseline height SDS remained borderline (B = 0.20; 95% CI − 0.03 to 0.43; p = 0.079); age (p = 0.74) and puberty (p = 0.57) were not significant (Table 6). Model strength was low (R^2 = 0.18; n = 36). The result suggests that height adjustment removes most of the misclassification in standard DEXA, while a small, height-independent mineral deficit cannot be excluded in the patients with the most severe growth failure. Because of the small subtype groups, subtypes were not entered into the model.

Table 6
Linear regression for height-adjusted DEXA z-score (n = 36).

Variable	Univariable B [95% CI]	Multivariable B [95% CI]; p
Baseline height SDS	0.21 [0.05–0.37]; p = 0.013	0.20 [− 0.03 to 0.43]; p = 0.079
Age	—	0.03 [− 0.13 to 0.18]; p = 0.74
Pubertal (stage ≥ 2)	—	−0.41 [− 1.88 to 1.05]; p = 0.57

Discussion

The interpretation of low areal BMD in MPS has long been divided between two views. On one side, data showing that GAG accumulation directly impairs chondrocyte function and matrix mineralization treat low z-scores as a real mineral loss [6, 11]. On the other, Polgreen and colleagues [7] and Kor and colleagues [5] showed that adjustment for height-for-age z-score sharply reduces the frequency of low BMD, and read most of the low standard scores as a measurement artifact driven by short stature. Our data lean toward the second view: about two-thirds of the low-BMD frequency seen on standard DEXA disappears once height adjustment is applied. Even in this cohort, where skeletal dysplasia is prominent, adjustment removed most of the misclassification in the standard measurement: a low-density frequency reaching 64.7% fell to 20.6% after adjustment. The finding confirms the strong effect of short stature on the DEXA measurement in MPS and agrees with multicenter data that place height adjustment as a routine step [5, 6, 7]. At the same time, the borderline effect of baseline height SDS in the multivariable model suggests that a small, height-independent real deficit cannot be excluded in the most severe patients; the two views are therefore not fully exclusive. Beyond confirming this effect, we show how far it shifts the clinical decision threshold: the low BMD seen in every evaluable type IVA patient largely disappeared after adjustment. Uncorrected readings could lead to false-positive diagnosis and unnecessary antiresorptive treatment in these patients.

The multivariable model supports the need for height adjustment, but the result is fragile. Baseline height SDS was significant on univariable analysis and borderline in the multivariable model, with the lower confidence limit close to zero. This suggests that a small, height-independent mineral deficit probably exists in the patients with the most severe growth failure, but could not be confirmed with this sample. The only notable subtype-level observation is the relatively high adjusted DEXA in type III;

because it rests on two patients, it should be treated as a hypothesis for further work rather than a type III–specific bone phenotype [12].

Growth failure dominated the clinical picture of the cohort. Our frequency of short stature (61.9%) is in line with earlier series: Kor and colleagues reported 67.5% [5] and Lin and colleagues 81% [6]. The decline of annual growth velocity with age (rho = − 0.61) reflects the natural course in childhood and runs in the same direction as the observation by Lin and colleagues that bone parameters correlate negatively with age [6]. The definition of growth velocity mattered: strong and consistent relationships emerged only after it was restricted to patients with a follow-up interval of at least nine months. Rates annualized over very short intervals are artificial and probably added noise to earlier analyses. The positive correlation of growth velocity with ALP supports the reading that bone formation activity is higher in fast-growing patients. The availability of MPS subtype-specific growth charts suggests that height may be better interpreted against disease-specific references than against standard population curves [13, 14, 15, 16]. The contribution of ERT to linear growth varies with disease type and age at treatment start [17]; gains in height z-score have been reported in MPS VI patients treated early [18, 19], and MPS I registry data likewise support a positive but incomplete effect of early ERT [20]. In our cohort, the decline of height SDS over follow-up is consistent with the literature showing that ERT does not fully normalize linear growth. Lin and colleagues showed that absolute BMD rose in patients on ERT, but that HAZ-adjusted z-scores did not differ clearly between treated and untreated groups [6]; Fung and colleagues likewise reported a limited effect of ERT on bone mineral content [11].

Vitamin D insufficiency or deficiency was found in 59.5% of patients. This is broadly in line with the 58.8% reported by Kor and colleagues [5] and the 87.5% reported by Fung and colleagues [11], and supports an effect of MPS on vitamin D homeostasis. In Türkiye, the rate in children without chronic disease is around 40% [21] and rises markedly in the presence of chronic disease [9]. The high frequency of insufficiency fits the hypothesis that GAG accumulation may reduce UV absorption in the skin and vitamin D absorption in the gut [5].

The PTH finding calls for caution. At the group level, the higher tendency of PTH in the short-stature group with vitamin D equal between groups ($p = 0.054$) suggests that secondary hyperparathyroidism may relate to MPS-specific increased bone turnover rather than to vitamin D insufficiency [5]. The subtype data, however, show the opposite pattern: the highest PTH was seen in the two Sanfilippo patients, who were not short-statured but had low vitamin D, and the between-subtype PTH difference was not significant. These signals, which do not agree across two levels of the same dataset, show that the relationship between PTH and growth is at the level of a hypothesis rather than evidence; the exploratory design and the absence of correction for multiple comparisons also call for this caution. The finding should be tested in larger series stratified by vitamin D status.

The broadly preserved endocrine axes are a finding of direct clinical value. The absence of clinically meaningful deviation in the thyroid, adrenal and gonadal parameters supports the view that growth failure in MPS relates more to skeletal dysplasia and the direct effect of GAG accumulation than to a

classic hormonal deficiency. Even so, occasional endocrine involvement has been reported in MPS, in particular precocious puberty in Sanfilippo patients [22]; follow-up for pubertal development is therefore reasonable.

Limitations

The main limitation is the retrospective design, which brought missing data for some variables and protocol heterogeneity. Pooling five MPS types with clearly different skeletal phenotypes weakens subtype-level inference; with only two patients each in types II and III, the findings for these groups are descriptive only and can be strengthened only through multicenter collaboration. ERT was heterogeneous in drug and duration and was not adjusted for in the analyses. In some patients, extreme height z-scores beyond the reference range (down to − 13) are extrapolations, so the median was used as the measure of central tendency. Growth velocity was defined only for patients with a follow-up interval of at least nine months, which reduced the short-interval artifact, but the overall spread of follow-up times remained wide. Record inconsistencies, such as a height-adjusted value present without a standard DEXA record in two patients, should be resolved with a standard protocol in future prospective work. Finally, because no correction for multiple comparisons was applied, all subgroup and correlation findings should be regarded as hypothesis-generating.

Conclusion

In children with MPS, growth failure and vitamin D insufficiency are common regardless of ERT use. Standard DEXA systematically underestimates bone density in subtypes with marked skeletal dysplasia; readings given without height adjustment may lead to false-positive diagnosis and unnecessary treatment. Clinical decisions should therefore rest on the height-adjusted DEXA score. The tendency for PTH to rise independently of vitamin D in the short-stature group is a hypothesis-level observation that points to a possible contribution of increased bone turnover in MPS and needs confirmation. The broadly preserved endocrine axes indicate that follow-up should focus first on the skeletal–growth axis. Adding height-adjusted bone density, vitamin D supplementation and periodic endocrine screening to routine assessment would strengthen the management of these patients.

Declarations

Conflict of interest:

The authors declare that they have no conflict of interest.

Ethics approval:

The study was approved by the local Clinical Research Ethics Committee of Gaziantep University (approval no. 169; date 07.06.2023) and conducted in accordance with the Declaration of Helsinki.

Consent for publication

Written informed consent for publication of identifying images and clinical details was obtained from the parents or legal guardians of the participants.

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Author Contribution

S.A. conceptualized and designed the study, collected and processed the data, performed the literature review, and drafted the main manuscript text. M.K.1 (Murat Karaoğlan) performed data analysis and interpretation, and provided resource support. M.K.2 (Mehmet Keskin) provided study supervision, critical revision, and overall leadership. All authors reviewed and approved the final manuscript.

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Not applicable.

Data Availability

he datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Figures

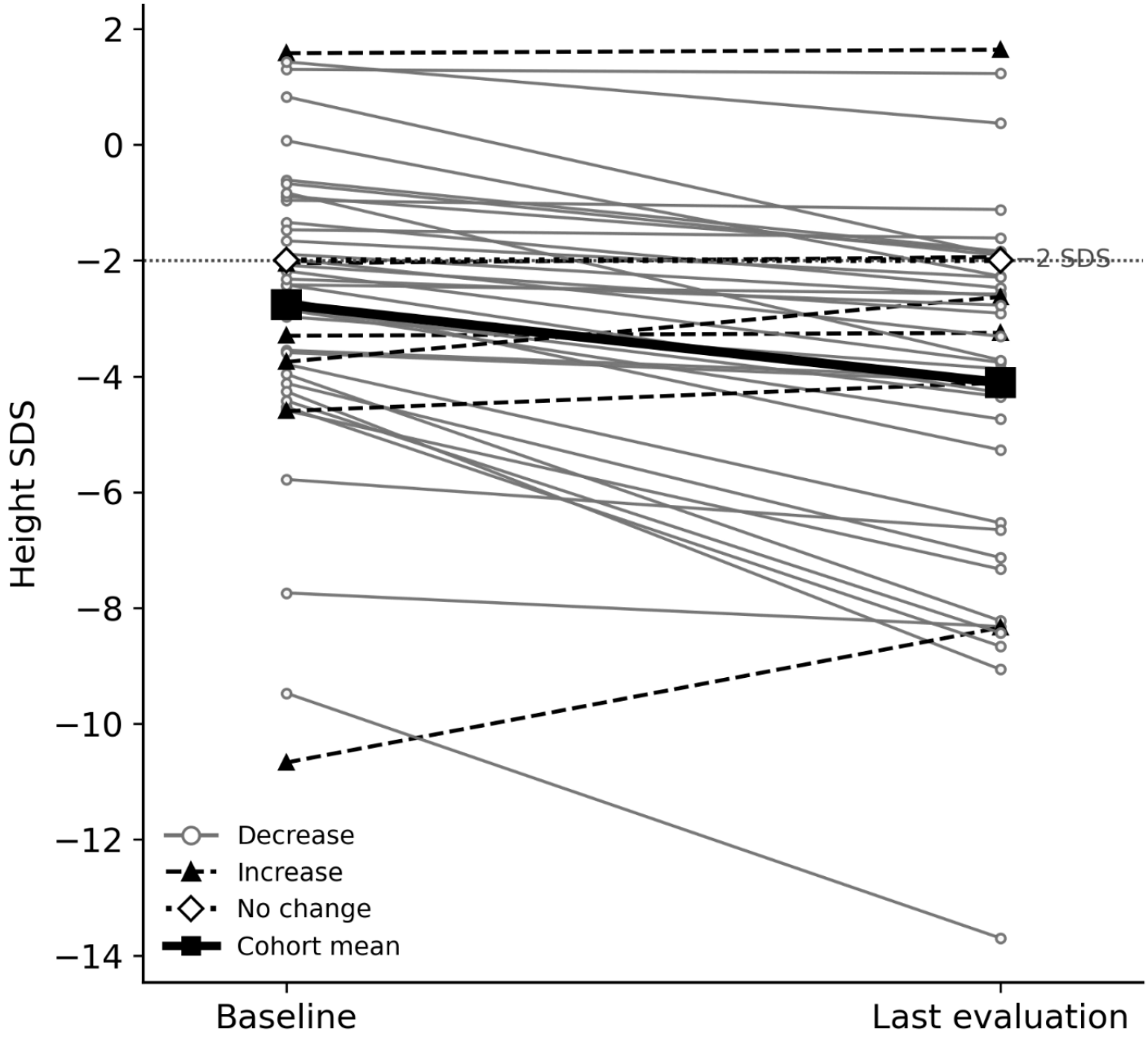


Figure 1

Individual change in height SDS between baseline and last evaluation (n = 42). Red, decrease; green, increase; gray, no change. The black line marks the cohort mean and the dashed line the −2 SDS threshold.

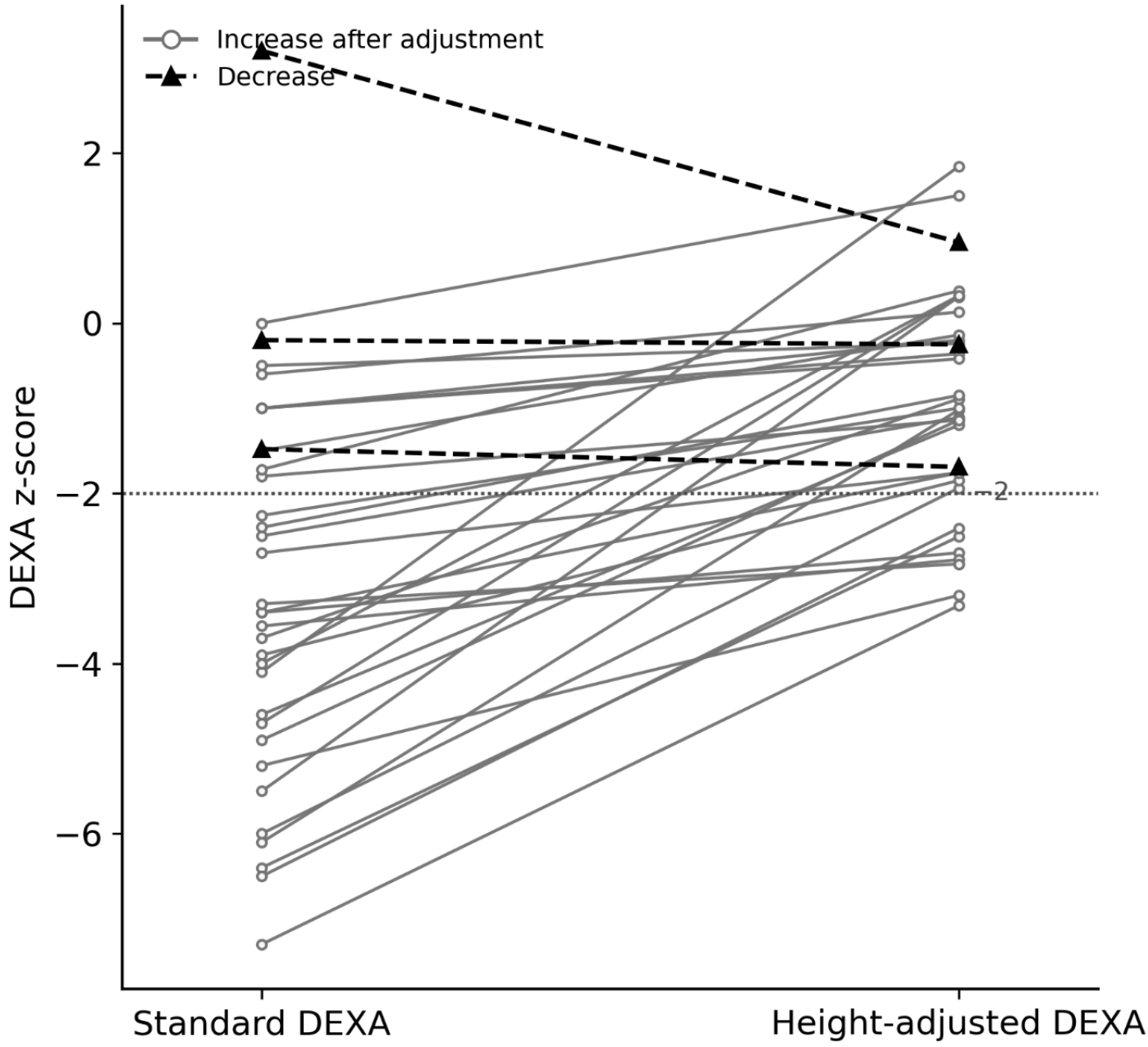


Figure 2

Individual comparison of standard and height-adjusted DEXA z-scores (n = 34). Green, increase; red, decrease. The dashed line marks the −2 threshold.

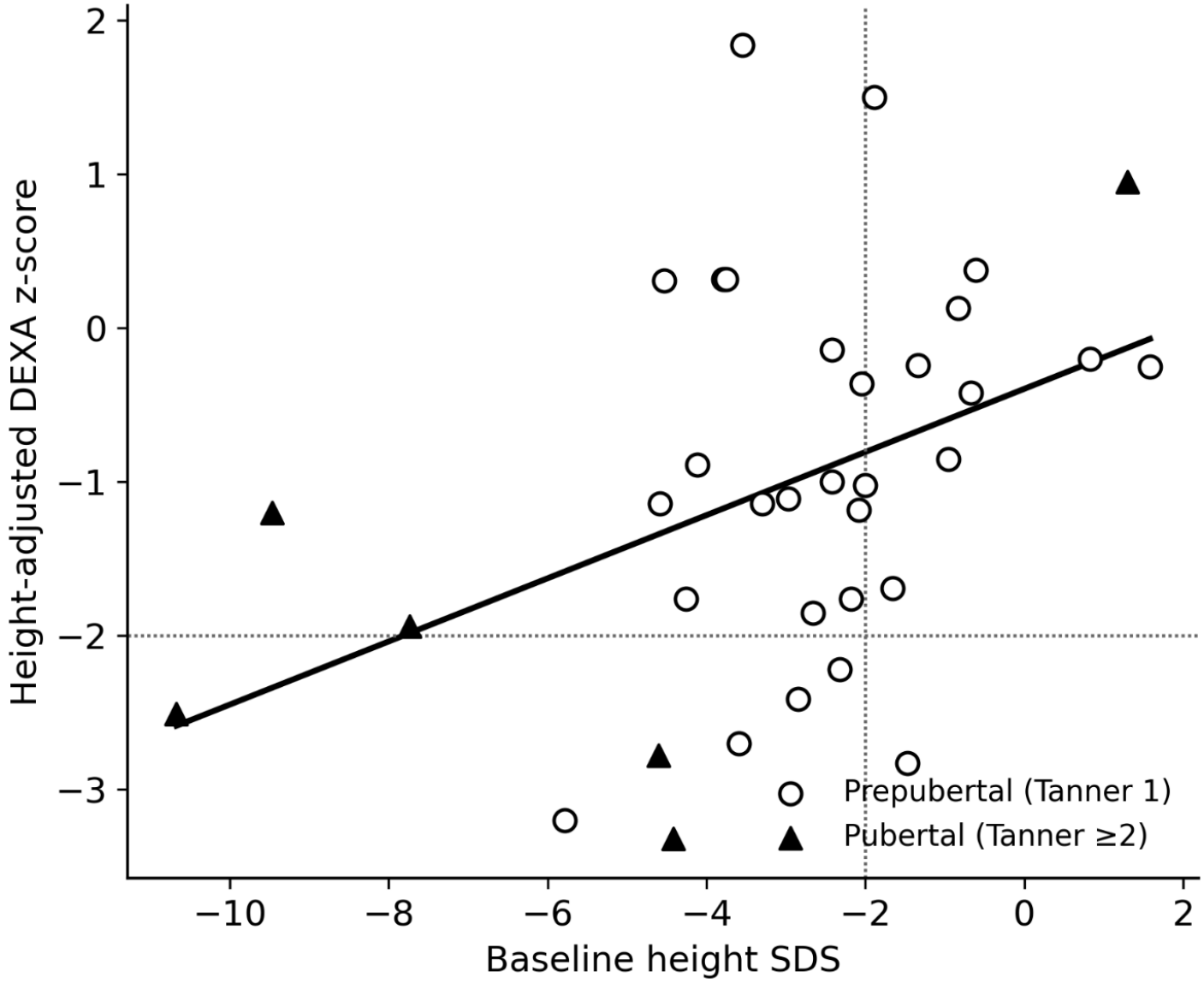


Figure 3

Relationship between baseline height SDS and height-adjusted DEXA z-score (n = 36). The red line shows the linear regression and the dashed lines the −2 thresholds.