MEDICAL SCIENCES

OPEN ACCESS

GROWTH PATTERNS IN DUCHENNE MUSCULAR DYSTROPHY

Iulia RODOMAN[1,2], Victoria SACARA[2], Ina PALII[1,3]

[1] Cardiology Unit, Institute of Mother and Child, Chisinau, Republic of Moldova
[2] Laboratory of Human Molecular Genetics, Institute of Mother and Child, Chisinau, Republic of Moldova
[3] Department of Pediatrics, *Nicolae Testemitanu* State University of Medicine and Pharmacy, Chisinau, Republic of Moldova

Corresponding author: Iulia RODOMAN, e-mail: iulia.rodoman@gmail.com

https://doi.org/10.38045/ohrm.2026.2.05 CZU: 616.8-056.76:616.74-009.54-053.2

ABSTRACT

Introduction Duchenne muscular dystrophy (DMD) is a genetic disorder that significantly affects growth and development, characterised by progressive degeneration of skeletal and cardiac muscles, typically beginning in early childhood, between 2 and 5 years old.

Purpose of the study To assess longitudinal growth parameters (height, weight, BMI) in children with genetically confirmed DMD compared with healthy age- and sex-matched controls.

Materials and methods A total of 100 children were included: 50 with DMD and 50 controls. Anthropometric data were collected at 3 follow-up visits over a 12-month period and analysed using Microsoft Excel (Office 365) and StatTech v.4.6.3.

Results Children with DMD exhibited impaired growth compared with controls: lower height (median 1.25 m [1.10–1.44] vs 1.56 m [1.37–1.68]; p<0.001), lower height z-score (–0.95±1.57 vs 0.42±1.41; p<0.001), and lower height percentile (16.0 vs 63.7; p<0.001). Body weight was also lower (27.7 kg [18.5–34.0] vs 46.0 kg [29.3–58.0]; p<0.001), with reduced weight z-score (–0.61 vs 0.57; p=0.003) and percentile (26.9 vs 71.6; p=0.003). BMI was lower in the DMD group (15.9 vs 18.4 kg/m^2; p=0.007), whereas BMI z-scores and percentiles did not differ significantly (p>0.05). Growth velocity declined over time (height increase of 0.03 m from Visit 1 to Visit 2 vs 0.01 m from Visit 2 to Visit 3; p<0.01). Among patients with DMD stratified by duration of deflazacort treatment, the >12-month subgroup showed the greatest growth gains, although differences between subgroups were not statistically significant.

Conclusions Children with DMD had a delayed growth compared with their peers, with progressive slowing over time. Regular auxological monitoring is essential, and further studies are needed to clarify the effects of corticosteroid therapy.

Keywords Duchenne muscular dystrophy, children, growth patterns.

PATTERNUL DE CREȘTERE ÎN DISTROFIA MUSCULARĂ DUCHENNE

Introducere Distrofia musculară Duchenne (DMD) este o afecțiune genetică cu impact major asupra creșterii, provocând degenerarea progresivă a mușchilor scheletici și cardiaci, care debutează în copilăria timpurie – la 2-5 ani.

Scopul Evaluarea longitudinală a creșterii (înălțime, greutate și IMC) la copiii cu DMD comparativ cu martori sănătoși, potriviți după vârstă și sex.

Materiale și metode Studiul a inclus 100 copii: 50 pacienți cu DMD și 50 martori. Datele antropometrice au fost colectate la 3 vizite pe parcursul a 12 luni și analizate cu Office365 Excel și StatTech v.4.6.3.

Rezultate Comparativ cu lotul martor, copiii cu DMD au prezentat creștere semnificativ afectată: talie mai mică (mediana 1,25 m [1,10–1,44] vs 1,56 m [1,37–1,68]; p<0.001), scor z al taliei mai redus (–0,95±1,57 vs 0,42±1,41; p<0.001) și percentilă mai joasă (16,0 vs 63,7; p<0.001). Greutatea a fost, de asemenea, mai mică (27,7 kg [18,5–34,0] vs 46,0 kg [29,3–58,0]; p<0,001), cu scor z redus (–0,61 vs 0,57; p=0.003) și percentilă inferioară (26,9 vs 71,6; p=0.003). IMC a fost mai mic la DMD (15,9 vs 18,4 kg/m^2; p=0.007), însă scorurile z/percentilele IMC nu au diferit (p>0.05). Viteza de creștere s-a redus (0,03 m V1–V2 vs 0,01 m V2–V3; p<0,01). Subgrupul cu durata de tratament cu deflazacort >12 luni a înregistrat cele mai mari îmbunătățiri, deși diferențele între subgrupuri nu au atins semnificație statistică.

Concluzii Copiii cu DMD prezintă întârziere de creștere comparativ cu semenii, cu încetinire progresivă în timp. Monitorizarea auxologică regulată este esențială, iar studiile suplimentare sunt necesare pentru clarificarea efectului corticosteroizilor.

Cuvinte-cheie Distrofia musculară Duchenne, copii, pattern de creștere.

INTRODUCTION

Duchenne muscular dystrophy (DMD) is one of the most common types of muscular dystrophy, caused by mutations in the *DMD* gene on the X chromosome, and affecting approximately 1 in 3,500–5,000 male births worldwide. The lack of dystrophin, a protein essential for maintaining muscle strength and stability, leads to progressive muscle damage and weakness (1,2). This condition predominantly affects boys and typically manifests in early childhood with progressive muscle weakness that initially involves the pelvic and thigh muscles and later the additional muscle groups, ultimately leading to respiratory and cardiac failure (3).

Patients with DMD commonly exhibit altered growth trajectories that complicate clinical management. Boys with DMD show significant deviations in growth patterns compared with their peers, characterised by reduced growth velocity and developmental delays, particularly between 2 and 12 years of age (4). Although height may fall within normal ranges at around 2 years of age, a progressive decline in growth velocity is typically observed in later childhood (5). Children with DMD also frequently experience metabolic complications, including obesity, which may further affect their health status and growth patterns (6).

The use of glucocorticoids to manage symptoms of DMD has been associated with impaired linear growth. Although glucocorticoid therapy can delay the loss of ambulation and prolong motor function, it may also contribute to growth suppression and delayed puberty in a substantial proportion of patients (4,7,8). Several studies have shown that long-term corticosteroid therapy is associated with adverse effects on bone health and increased body weight, further complicating growth trajectories in affected individuals (5). However, recent evidence suggests that growth hormone therapy may improve growth velocity in prepubertal boys with DMD without adversely affecting neuromuscular function (9).

Growth velocity in boys with DMD has been reported to improve with appropriate therapeutic interventions. Some studies have shown that, prior to growth hormone therapy, growth velocity was recorded at 0 cm/year, increasing to a maximum of 7.8 cm/year following treatment (10). Nevertheless, height-for-age z-scores in these patients generally continue to decline, highlighting the persistent challenges in achieving normal growth trajectories despite therapy (11).

Research also highlights the importance of multidisciplinary management in tackling the many symptoms and complications of DMD, requiring coordinated care from specialists across multiple disciplines (12). Early diagnosis and timely intervention are essential for optimizing growth and functional outcomes, facilitating access to appropriate therapies and supportive services (13). Continuous monitoring of growth parameters—including height, weight, and body mass index—is essential for identifying potential treatment-related adverse effects and ensuring comprehensive care for affected children (14).

MATERIALS AND METHODS

This longitudinal observational study with a control group was conducted at the Paediatric Cardiology Clinic of the PMHI Institute of Mother and Child, Chișinău, Republic of Moldova. The study included 50 children with DMD, aged 1 year to 17 years, 11 months, and 29 days, from both urban and rural areas. Patients were consecutively enrolled during scheduled outpatient evaluations between November 2017 and February 2023.

The purpose of the study was to assess longitudinal growth (height, weight, and BMI) in children with genetically confirmed Duchenne muscular dystrophy and to compare anthropometric parameters with those of healthy age- and sex-matched controls. To achieve the purpose of the study, the following objectives were defined: to evaluate auxological parameters (height, body weight, and body mass index), including standardized indicators (z-scores and percentiles), in children with Duchenne muscular dystrophy compared with healthy peers; to assess longitudinal growth dynamics and growth velocity by analyzing shifts in anthropometric parameters across three follow-up visits over a 12-month period ; and to investigate the potential influence of glucocorticoid therapy (deflazacort) on growth patterns based on the duration of corticosteroid exposure.

The research comprised several stages.

The 1st stage included a total number of 100 paediatric participants : 50 patients with a genetically confirmed diagnosis of Duchenne muscular dystrophy (DMD) forming the study group, and 50 age- and sex-matched children with no known chronic pathologies serving as the control group.

Inclusion criteria for the study group were an age range of 1 to 17 years and 11 months, a DMD diagnosis confirmed via molecular genetic testing, informed consent from parents or legal guardians, and written assent from participants aged 14 years or older. The control group was selected based on demographic matching (age and sex) and a confirmed healthy status (classified as Health Level I or II), while adhering to the same consent and assent protocols as the study group. Exclusion criteria for both cohorts included pre-existing cardiac pathology, significant comorbidities potentially impacting cardiac function, an inadequate echocardiographic acoustic window, or the absence of required consent/assent. Participants were enrolled consecutively from eligible inpatient admissions and outpatient referrals during the study period.

During the 2nd stage, all participants underwent a standardized clinical examination. Particular emphasis was placed on collecting anthropometric data, including height, weight, and body mass index (BMI). Body weight was recorded using calibrated scales to the nearest 0.1 kg. Height was measured with a stadiometer and expressed in meters to one decimal place. BMI was calculated using the standard formula of weight divided by the square of height (kg/m^2). To allow for age- and sex-adjusted comparisons, anthropometric indices were further standardized by calculating z-scores and percentiles based on reference values. Additionally, data regarding deflazacort therapy, including dosage and duration, were recorded. Participants were then stratified according to their cumulative glucocorticoid exposure

In the 3rd stage of the study, the collected data were introduced into an electronic database. Statistical analysis was performed using Office 365 Microsoft Excel and StatTech version 4.6.3. Descriptive statistics included means, standard deviations, medians, interquartile ranges (IQR), z-scores, and percentiles. Group comparisons were performed using the Mann–Whitney U test for non-normally distributed variables and Student's t-test for normally distributed data. Categorical variables were analyzed using χ^2 tests with Cramér's V effect size. Repeated comparisons across visits were assessed using paired tests (paired t-test or Wilcoxon), and intergroup comparisons by ANOVA where appropriate. Statistical significance was defined as $p \leq 0.05$.

Subsequently, in the 4th stage, practical conclusions and evidence-based recommendations were formulated based on the study findings. All participants were included only after receiving an individual explanation of the study objectives and procedures. No financial incentives were provided, and participation involved no costs for the families.

RESULTS

Auxological Analysis

To evaluate differences in anthropometric parameters between the study and control groups, absolute values and standardized indicators—specifically z-scores and percentiles for height, weight, and BMI—were compared using appropriate statistical methods (Fig. 1).

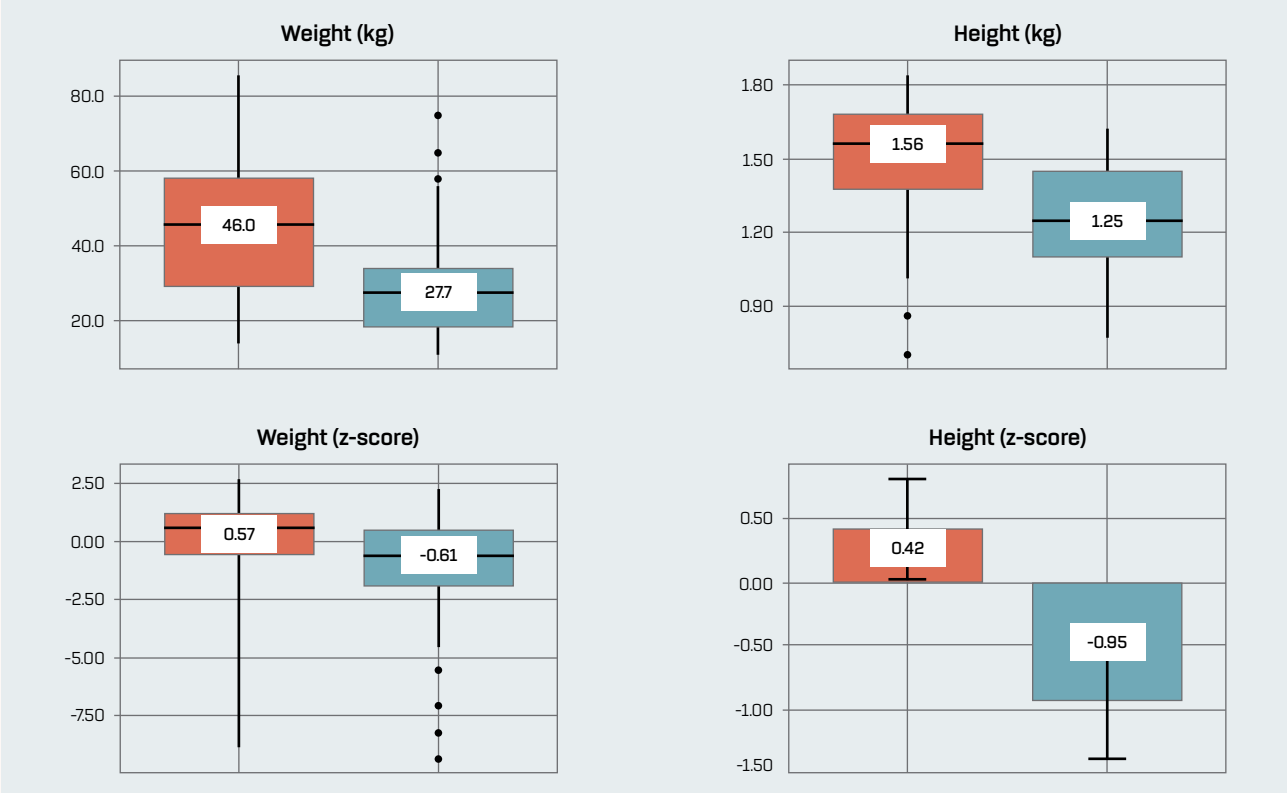

Figure 1. Boxplot comparison of body weight, height, and corresponding z-scores between the control group (red) and the DMD group (turquoise).

Height. Upon the first visit, the median height in the DMD group was 1.25 m (Q_1–Q_3: 1.10–1.44 m), significantly lower than the control group 1.56 m (Q_1–Q_3: 1.37–1.68 m; $p < 0.001$, $U = 596.5$). The mean height z-score was –0.95 ± 1.57 in the DMD group versus 0.42 ± 1.41 in the control group ($p < 0.001$; $t = 4.584$). The height percentile was also significantly lower in the DMD group (median 16.0; Q_1–Q_3: 3.84–59.4) compared to controls (median 63.7; Q_1–Q_3: 25.8–90.8; $p < 0.001$). These data highlighted a significant deficiency in physical development in children with DMD. Categorical analysis of height revealed marked disparities between the groups ($\chi^2 = 22.64$; $df = 4$; V Cramér = 0.48; $p < 0.001$), with a higher incidence of low stature in the DMD group (32.0%) relative to controls (8.0%). Normal stature was recorded in 40.0% of DMD patients versus 50.0% of controls, whereas tall and very tall stature were exclusively observed in the control group.

Weight. Children with DMD had significantly lower body weight (median 27.7 kg; Q_1–Q_3: 18.5–34.0) compared to control group (46.0 kg; Q_1–Q_3: 29.3–58.0; $p < 0.001$; $U = 663$). The z-score for weight was also lower (median –0.61; Q_1–Q_3: –1.88 to 0.49 vs. 0.57; Q_1–Q_3: –0.53 to 1.17; $p = 0.003$). Similarly, the weight percentile was lower in DMD patients (median 26.9 vs. 71.6 in controls; $p = 0.003$), indicating a delayed weight gain and growth restriction.

Body Mass Index. The median BMI was significantly lower in the DMD group (15.9 kg/m²; Q_1–Q_3: 14.6–19.5) than in the control group (18.4 kg/m²; Q_1–Q_3: 16.1–20.7; p = 0.007). However, BMI z-scores and percentiles showed no significant differences between the groups (p > 0.05), suggesting relatively similar distribution patterns once adjusted for age and sex. Analysis of BMI categories likewise revealed no significant overall disparities between the two cohorts (χ^2 = 3.89; df = 3; V Cramér = 0.2; p = 0.274). However, when stratified by disease stage, the distribution of BMI categories differed significantly (χ^2 = 22.88; df = 12; V Cramér = 0.39; p = 0.029). Underweight status was most prevalent during the presymptomatic, late ambulatory, and late non-ambulatory stages of the disease.

Analyses of growth pattern through 3 visits

To assess physical growth dynamics, body weight, height, and BMI were recorded at three follow-up visits conducted over 6- and 12-month intervals. Violin plots (Fig. 2) illustrate the distribution density of these parameters across the three evaluation time points. An observed rightward shift in median values, accompanied by changes in distribution morphology, reflects an active growth process consistent with established pediatric developmental trends within this cohort.

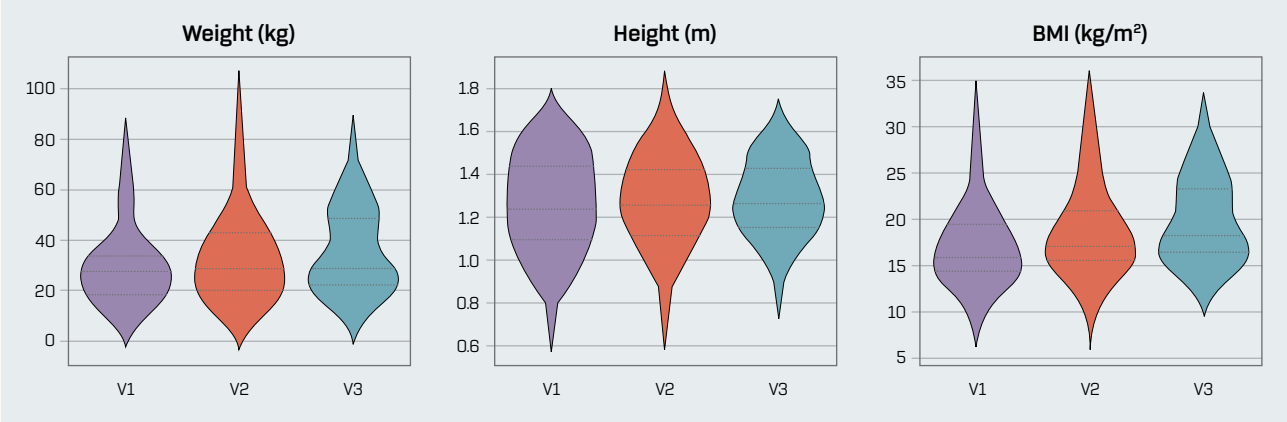

Figure 2. Violin plot distribution of body weight (kg), height (m), and BMI (kg/m²) across three follow-up visits (V1–V3), with V1 shown in purple, V2 in red, and V3 in green.

Height demonstrated a modest upward trend, with the mean increasing from 1.277 ± 0.218 m at visit 1 to 1.291 ± 0.200 m (visit 2) and 1.297 ± 0.175 m (visit 3). The median rose from 1.245 m to 1.27 m, with a slight narrowing of the range, indicating growing homogeneity in height across the cohort.

Body weight showed a progressive increase over time. At the first visit, the mean weight was 29.19 ± 14.30 kg (median: 27.7 kg; range: 10.9–75.0 kg). At the second visit, the mean increased to 33.62 ± 17.61 kg, and at the third visit to 35.15 ± 15.63 kg, with a stable median of 29.0 kg for the latter two. The interquartile range increased from 18.5–34.0 kg (visit 1) to 22.25–48.75 kg (visit 3), suggesting increased variability in weight gain among subjects.

Body Mass Index exhibited a clear upward trend across the three visits: 17.08 ± 4.44 kg/m² at visit 1, 18.92 ± 4.86 kg/m² at visit 2, and 19.79 ± 4.30 kg/m² at visit 3. The median values increased from 15.92 to 18.32 kg/m², and the interquartile range expanded from 14.55–19.55 to 16.47–23.35 kg/m², reflecting an increase in the variability of nutritional status among the participants over time.

Growth Velocimetry

To further examine growth dynamics, absolute and relative changes in weight, height, and BMI between Visits 1–2 and 2–3 were evaluated using paired t-tests. The results, as illustrated in Figure 3, demonstrated statistically significant changes across all assessed parameters.

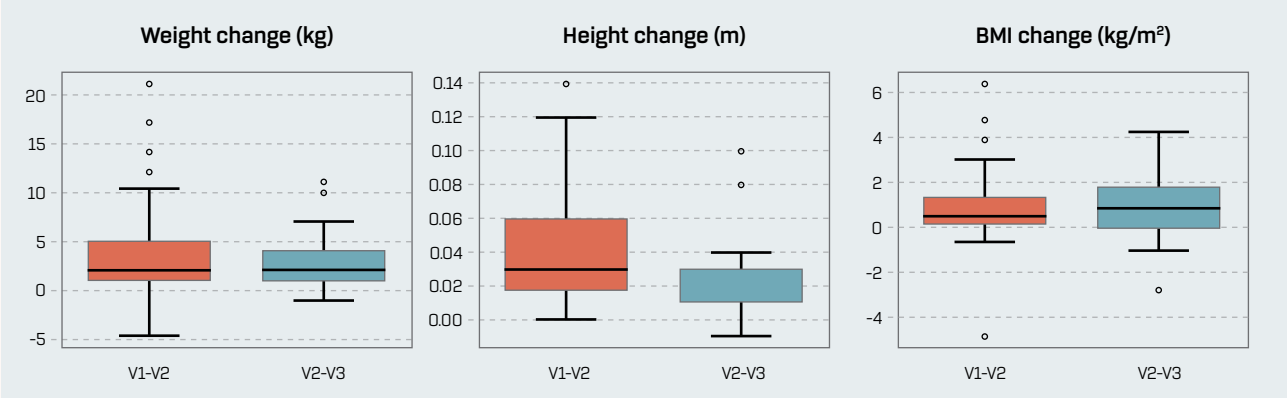

Figure 3. Growth velocimetry: distribution of absolute changes in body weight (kg), height (m), and body mass index (BMI, kg/m²) between consecutive visits (V1–V2 and V2–V3), presented as boxplot diagrams.

Weight gain velocity decreased from a median of 2.0 kg between visits 1–2 to 2.0 kg in visits 2–3, with lower mean and variability in the second period. Height growth also slowed significantly, with the median change decreasing from 0.03 m to 0.01 m. BMI changes were more variable and nonlinear, suggesting a disproportionate progression between height and weight in some subjects. Overall, these data reflect a physiological transition toward slower growth in the later observation period.

Impact of deflazacort exposure on growth

To assess the potential impact of deflazacort exposure on growth, patients were stratified into four groups based on the cumulative duration of corticosteroid use: 0 – no exposure, 1 – short-term use (≤6 months), 2 – intermediate use (7–12 months), and 3 – long-term use (>12 months). Longitudinal changes in height, weight, and BMI were evaluated across three visits (Fig. 4).

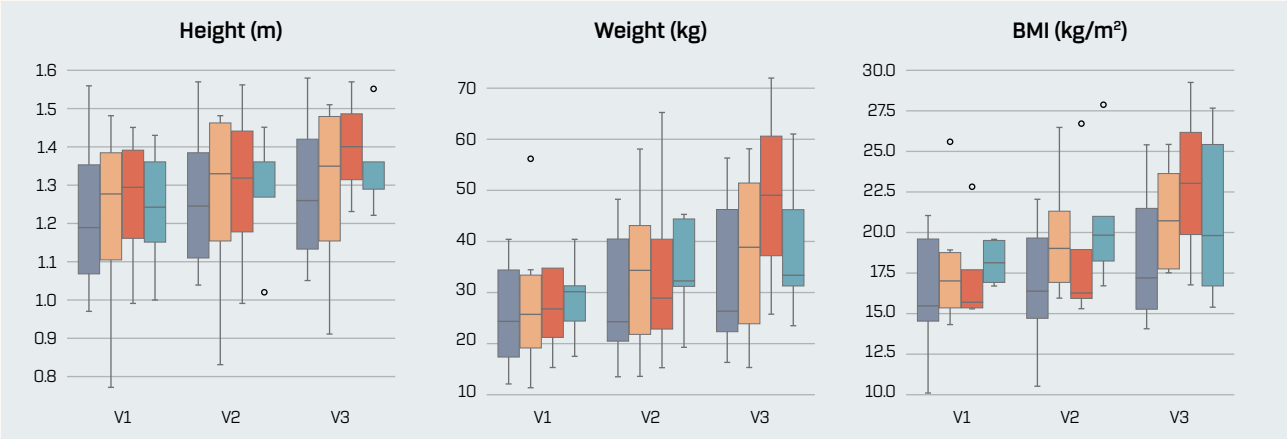

Figure 4. Impact of deflazacort exposure on growth patterns. Boxplots of height (m), weight (kg), and BMI (kg/m²) across three visits, stratified by deflazacort exposure duration: no use (blue), 0–6 months (orange), 7–12 months (red), and >12 months (green).

Height. At baseline (Visit 1), mean height was comparable across groups, ranging from 1.21 m (Group 0) to 1.26 m (Group 1), with no statistically significant difference ($p = 0.978$). All groups demonstrated incremental height increases over the study period. The most substantial gain was observed in Group 3 (long-term deflazacort), which increased from 1.24 m at Visit 1 to 1.34 m at Visit 3. While height gains remained relatively consistent between Visits 1 and 2 (mean change: 0.04–0.05 m), Group 3 exhibited the highest mean increase of 0.068 ± 0.083 m between Visits 2 and 3. However, this trend did not reach statistical significance ($p = 0.142$). ANOVA revealed no significant intergroup differences in height at any follow-up visit (Visit 2, $p = 0.993$; Visit 3, $p = 0.824$).

Weight. At Visit 1, mean weight varied from 25.4 kg (Group 2) to 29.0 kg (Group 1), with no significant group differences ($p = 0.899$). Over time, all groups experienced weight gain. By Visit 3, Group 3 exhibited the highest mean weight (38.8 kg), corresponding to a total gain of 10.4 kg. Between Visits 1 and 2, the largest mean increase was observed in Group 3 (+5.8 kg, SD = 5.76; $p = 0.028$), while Group 2 recorded more modest gains (+3.11 kg, $p < 0.001$). ANOVA did not detect significant group differences at Visits 2 or 3 ($p = 0.752$ and $p = 0.831$, respectively).

Body Mass Index. At baseline, mean BMI ranged from 16.57 kg/m² (Group 2) to 18.20 kg/m² (Group 3). All groups exhibited increases in BMI over time. The most notable rise occurred in Group 3, which reached a mean of 21.02 kg/m² by Visit 3, characterized by a pronounced gain of +2.54 units between Visits 1 and 2. In contrast, Group 2 showed the most stable BMI progression (0.38 units between Visits 1 and 2, 1.37 units between Visits 2 and 3). ANOVA results for BMI were not statistically significant across groups at any time point (Visit 1 $p = 0.684$; Visit 2 $p = 0.729$; Visit 3 $p = 0.762$). These findings suggest that while longitudinal gains in anthropometric parameters were observed across all groups, the most pronounced increases—particularly in height and BMI—occurred in patients with >12 months of deflazacort exposure. However, the lack of consistent statistical significance limits the ability to draw definitive conclusions regarding a direct causal effect.

DISCUSSIONS

The present study provides a longitudinal analysis of growth trajectories in children with Duchenne muscular dystrophy, stratified by cumulative deflazacort exposure. These findings have revealed that all patient groups, regardless of GC exposure, experienced positive growth trajectories in height, weight, and BMI over the study period.

In a similar analysis, Stimpson et al. analyzed the growth patterns in boys with DMD using longitudinal anthropometric data from 598 patients with a total of 2604 observations, demonstrating that daily corticosteroid treatment was associated with significant height stunting compared with glucocorticoid-naïve patients. In particular, boys receiving daily deflazacort showed a mean annual height change approximately 0.25 standard deviations below reference growth values, highlighting the potential consequence of prolonged corticosteroid therapy on linear growth (5). These findings also confirmed that boys with DMD generally exhibit lower height trajectories compared with the general paediatric population. In contrast to some prior reports, the results of this study indicated that patients with prolonged deflazacort use (>12 months) showed the most pronounced increases, particularly in height and BMI, although their anthropometric parameters remained lower compared with healthy controls.

Clinical studies indicate that deflazacort administration can result in statistically significant increases in height when compared to other corticosteroids, such as prednisolone. In a study by Singhal et al. children receiving deflazacort demonstrated an average height increase of 2.13 ± 0.50 cm, significantly higher than the 1.44 ± 0.45 cm observed with prednisolone ($p = 0.03$) (15). This finding supports the hypothesis that deflazacort may be more favourable for promoting growth in paediatric populations with conditions such as idiopathic nephrotic syndrome. Such data may provide indirect supportive evidence relevant to DMD cohorts. Furthermore, longitudinal evidence indicates that patients treated with deflazacort exhibit superior overall growth patterns compared to those receiving prednisolone. However, it is important to note that both therapies have well-documented side effects, including growth retardation (5).

Regarding BMI, evidence suggests that deflazacort may lead to less weight gain than prednisolone. Guglieri et al. noted that while both daily prednisolone and deflazacort resulted in slowing growth, the weight gain associated with prednisolone was significantly higher (16). This difference is crucial, as excessive weight gain can complicate the clinical picture in patients with DMD, suggesting that deflazacort may provide a more balanced therapeutic profile, potentially supporting growth while limiting excessive weight gain. Such confirmatory findings were presented in studies where long-term use of deflazacort improved growth markers over time. For example, Marden et al. observed that deflazacort therapy was associated with maintained or improved functional outcomes, suggesting a positive correlation between functional capacity and growth in boys with DMD (17). Conversely, Levine et al. reported that while patients receiving deflazacort generally had better clinical outcomes, prolonged corticosteroid therapy could still result in stunted growth when compared to children not on corticosteroids (18).

Deflazacort has shown potential to improve growth parameters such as height and BMI. However, its effectiveness should be evaluated in comparison with prednisolone, while also considering potential adverse effects, in order to optimise treatment for children with Duchenne muscular dystrophy and other paediatric neuromuscular disorders. Variability in growth responses associated with different corticosteroid regimens may influence clinical decision-making and supports an individualised approach to patient management.

LIMITATIONS OF THE STUDY

The relatively small sample size may increase the risk of type II error (false-negative findings), potentially masking true differences between groups. Additionally, the single-centre design may limit external validity and the generalisability of the findings. Other limitations include the lack of hormonal evaluation (e.g., growth hormone axis, pubertal status) and the absence of bone age assessment to objectively document growth delay. Furthermore, the absence of consistent statistical significance across some comparisons has highlighted the need for larger multicentred cohorts and well-designed prospective studies to better clarify the impact of deflazacort on growth trajectories in DMD patients.

CONCLUSIONS

1. Longitudinal data demonstrate that while children with DMD increase in height and weight over time, their growth remains delayed compared with that of their peers. The findings underscore a higher prevalence of short stature and underweight status in the DMD group, with significant differences in both absolute and standardized anthropometric measures.
2. Analysis of growth velocity further revealed a deceleration of growth in height and weight between the second and third visits, reflecting a progressive slowing of growth as the disease advances.
3. Regular monitoring of auxological parameters in DMD remains essential for individualized clinical management.
4. Further longitudinal studies with larger cohorts are required to clarify the dose–response relationship between corticosteroid duration and growth outcomes and to identify modifiable clinical factors that may help optimize growth and nutritional status in this vulnerable population.

CONFLICT OF INTEREST The authors declare no conflicts of interest.

FUNDING STATEMENT The study was conducted as part of a PhD's research project within the Doctoral School in Health Sciences of Nicolae Testemitanu State University of Medicine and Pharmacy.

ETHICAL APPROVAL Ethical approval was obtained from the Research Ethics Committee of the *Nicolae Testemițanu* State University of Medicine and Pharmacy (approval report no. 1 dated 27.11.2019).

REFERENCES

1. Babbs A, Chatzopoulou M, Edwards B, et al. From diagnosis to therapy in Duchenne muscular dystrophy. *Biochem Soc Trans*. 2020;48(3):813-821. https://doi.org/10.1042/BST20190282
2. Shih JA, Folch A, Wong BL. Duchenne Muscular Dystrophy: the Heart of the Matter. *Curr Heart Fail Rep*. 2020;17(3):57-66. https://doi.org/10.1007/s11897-020-00456-0
3. Wasilewska E, Małgorzewicz S, Sobierajska-Rek A, et al. Transition from Childhood to Adulthood in Patients with Duchenne Muscular Dystrophy. *Medicina (Kaunas)*. 2020;56(9):426. Published 2020 Aug 24. https://doi.org/10.3390/medicina56090426
4. Wang B, Zhou L, Li S, et al. Height development and multiple bone health indicators in children aged 2-12 years with Duchenne muscular dystrophy (DMD). *PLoS One*. 2025;20(1):e0316938. Published 2025 Jan 10. https://doi.org/10.1371/journal.pone.0316938
5. Stimpson G, Raquq S, Chesshyre M, et al. Growth pattern trajectories in boys with Duchenne muscular dystrophy. *Orphanet J Rare Dis*. 2022;17(1):20. Published 2022 Jan 24. https://doi.org/10.1186/s13023-021-02158-9
6. Weber DR, Hadjiyannakis S, McMillan HJ, Noritz G, Ward LM. Obesity and Endocrine Management of the Patient With Duchenne Muscular Dystrophy. *Pediatrics*. 2018;142(Suppl 2):S43-S52. https://doi.org/10.1542/peds.2018-0333F
7. Bowden SA, Connolly AM, Kinnett K, Zeitler PS. Management of Adrenal Insufficiency Risk After Long-term Systemic Glucocorticoid Therapy in Duchenne Muscular Dystrophy: Clinical Practice Recommendations. *J Neuromuscul Dis*. 2019;6(1):31-41. https://doi.org/10.3233/JND-180346
8. McCarrison S, Denker M, Dunne J, et al. Frequency of Delayed Puberty in Boys with Contemporary Management of Duchenne Muscular Dystrophy. *J Clin Res Pediatr Endocrinol*. 2024;16(4):458-465. https://doi.org/10.4274/jcrpe.galenos.2024.2024-2-18
9. Lavi E, Cohen A, Dor T, Tsabari R, Zangen D. Growth Hormone Therapy for Children With Duchenne Muscular Dystrophy and Glucocorticoid Induced Short Stature. *J Endocr Soc*. 2021;5(Suppl 1):A715. Published 2021 May 3. https://doi.org/10.1210/jendso/bvab048.1455
10. Lavi E, Cohen A, Libdeh AA, Tsabari R, Zangen D, Dor T. Growth hormone therapy for children with Duchenne muscular dystrophy and glucocorticoid induced short stature. *Growth Horm IGF Res*. 2023;72-73:101558. https://doi.org/10.1016/j.ghir.2023.101558
11. Rutter MM, Collins J, Rose SR, et al. Growth hormone treatment in boys with Duchenne muscular dystrophy and glucocorticoid-induced growth failure. *Neuromuscul Disord*. 2012;22(12):1046-1056. https://doi.org/10.1016/j.nmd.2012.07.009
12. Paganoni S, Nicholson K, Leigh F, et al. Developing multidisciplinary clinics for neuromuscular care and research. *Muscle Nerve*. 2017;56(5):848-858. https://doi.org/10.1002/mus.25725
13. Bushby K, Finkel R, Birnkrant DJ, et al. Diagnosis and management of Duchenne muscular dystrophy, part 2: implementation of multidisciplinary care. *Lancet Neurol*. 2010;9(2):177-189. https://doi.org/10.1016/S1474-4422(09)70272-8
14. Welch TR. Growth in Duchenne muscular dystrophy. *J Pediatr*. 2013;163(6):1537-1539. https://doi.org/10.1016/j.jpeds.2013.10.037
15. Singhal R, Pandit S, Dhawan N. Deflazacort Versus Prednisolone: Randomized Controlled Trial in Treatment of Children With Idiopathic Nephrotic Syndrome. *Iran J Pediatr*. 2015;25(2):e510. https://doi.org/10.5812/ijp.510
16. Guglieri M, Bushby K, McDermott MP, et al. Effect of Different Corticosteroid Dosing Regimens on Clinical Outcomes in Boys With Duchenne Muscular Dystrophy: A Randomized Clinical Trial. *JAMA*. 2022;327(15):1456-1468. https://doi.org/10.1001/jama.2022.4315
17. Marden JR, Freimark J, Yao Z, Signorovitch J, Tian C, Wong BL. Real-world outcomes of long-term prednisone and deflazacort use in patients with Duchenne muscular dystrophy: experience at a single, large care center. *J Comp Eff Res*. 2020;9(3):177-189. https://doi.org/10.2217/cer-2019-0170
18. Levine H, Goldfarb I, Katz J, et al. Pulmonary function tests for evaluating the severity of Duchenne muscular dystrophy disease. *Acta Paediatr*. 2023;112(4):854-860. https://doi.org/10.1111/apa.16653

Date of receipt of the manuscript: 20.05.2025

Date of acceptance for publication: 08.03.2026

Iulia Rodoman, WoS Researcher ID: AAI-2287-2021, SCOPUS ID: 57468128500

Victoria Sacara, WoS Researcher ID: Y-3880-2018, SCOPUS ID: 57205301327

Ina Palii, WoS Researcher ID: AAI-2319-2021