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Height and weight trajectories are associated with submaximal and maximal exercise capacity in children with congenital heart defects

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Abstract

Children with congenital heart defects (CHD) are often short/lightweight relative to peers. Limited growth, particularly height, may reflect energy deficits impacting physical activity. Latent class analyses of growth from birth and Bruce treadmill exercise data retrospectively identified for height, weight, and body mass index z-scores growth trajectories. Linear regression models examined exercise parameters by growth trajectory, adjusting for age/sex/CHD severity. A total of 213 children with CHD (39% female, 12.1 ± 2.9 years) achieved 85.8 ± 10.1% of the predicted peak heart rate. Peak heart rate among children whose height was consistently below average (class 1) was 15.2 ± 4.9 beats/min lower than children with other height trajectories. These children also attained a lower percentage of predicted peak heart rate. Children whose weight ($p = 0.03$) or body mass index ($p < 0.001$) z-score increased throughout childhood had significantly lower exercise duration (mean difference 1–2 min) than children whose growth trajectories were stable or declined. Children with above-average weight or an increasing body mass index also used a higher percentage of their heart rate reserve at each submaximal exercise stage. A very low height z-score trajectory is associated with decreased exercise capacity that may increase the risk for morbidities associated with a sedentary lifestyle. Future studies should examine potential mechanisms for the observed height deficits, such as an inadequate energy supply that could impact physical activity participation, congestive heart failure, cyanosis, pubertal stage, supplemental feeding history, or familial growth patterns. Prospective studies examining growth in relation to objective measures of daily physical activity are required.

Introduction

Physical activity enables childhood growth/development.[1] Young children learn through physical movement, and activity is correlated with quality of life.[2] Children with congenital heart disease (CHD) can be less active than their peers,[3–5] even when their diagnosis has minimal sequelae and they have similar exercise capacity.[3,6–8] Studies identify a small proportion (<10%) of children with even complex CHD who successfully achieve the physical activity recommended for optimal health,[9–11] suggesting decreased activity is not an inevitable consequence of CHD. Hypoactive lifestyles have been identified as important,[3] as have parental overprotection[3,12] and children's physical activity self-efficacy.[13]

Growth deficit occurs in approximately 20% of children with CHD.[14,15] Infants with left-to-right shunts can require 35% more calories/day than peers.[16] Tachycardia, work of breathing, increased myocardial muscle mass, and heightened catecholaminergic state are suggested causal factors.[17] Coupled with increased energy demands, these children often experience feeding difficulties (e.g. gastroesophageal reflux, breathlessness) and inadequate nutritional intake.[18] Dietary counselling, supplements, and feeding strategies are employed to avoid the failure-to-thrive scenarios associated with poor outcomes.[18] There is often "catch-up" weight gain but not necessarily normalised height[17] after surgical CHD treatment, particularly after cardiopulmonary bypass exposure.[19] Decreased length/height occurs among two-thirds of infants/toddlers with CHD.[17] Increased weight for height[17] is common. Birth head circumference is associated with neurodevelopmental outcomes in childhood[20] and adolescence[21] with measurable growth deficits observed at 10 years of age.[15]

Adequate energy stores are necessary for physical activity. Childhood play requires energy expenditure rates 3–6 times higher than resting values.[22] Theoretically, growth deficits resulting from energy imbalances would limit energy stores available for active lifestyles. Understanding

how energy imbalance (underweight or overweight) relates to submaximal and maximal exercise capacity among children with CHD would inform family activity counselling by clinicians, enable effective active lifestyle intervention development, and identify children at increased risk for inactive lifestyle morbidities. This study evaluated relationships between submaximal and peak exercise capacity and height, weight, and body mass index growth trajectories among children with CHD.

Methods

Study data were collected retrospectively from medical charts among paediatric CHD patients completing a Bruce treadmill exercise protocol.[23] Height and weight were extracted for clinic visits prior to and at the exercise test. Latent class analyses identified homogeneous patient groups with similar height, weight, and body mass index growth trajectories. Linear regression models examined growth trajectory and exercise parameter relationships, adjusting for age, sex, and CHD severity. The Research Ethics Board of the Children's Hospital of Eastern Ontario (#11/168X) approved the protocol.

Participants

All patients with CHD who had completed a Bruce treadmill exercise protocol, with or without direct measures of cardiorespiratory function, at our institution were identified. Genetic or other medical conditions affecting growth, not performing the Bruce treadmill protocol, or an exercise test report indicating inadequate effort (as per the supervising cardiologist) were exclusion criteria. CHD diagnoses were classified[24] as simple, moderate or severe, or functional single ventricle (diagnostic details in Supplementary 1).

Data management

Peak heart rate, percentage of predicted maximal heart rate for age, and total exercise time were extracted from the exercise test reporting system (CASE 8000; GE Medical Systems, Milwaukee, Wisconsin). For each Bruce protocol stage attempted, heart rate, time, speed, and grade were recorded.

Three to 20 height and weight measures, from birth to exercise test age, were extracted from the medical record, and body mass index was calculated (weight(kg)/height^2(m)). Growth measures were converted to World Health Organization z-scores.[25] Biological sex and primary cardiac diagnosis were also recorded.

Statistical analyses

Descriptive statistics described the study population. Growth z-scores did not differ by sex ($p > 0.37$), so all participants were analysed together. Finite mixture modelling is a statistical tool that identifies unobserved or "latent" patterns within research observations.[26] The best probabilistic clustering of cases within the series growth measurements is determined from maximum likelihood estimates. The optimal number of classes had the minimum Akaike, Bayesian, and sample-size-adjusted Bayesian information criteria. Each class has a homogenous within-group pattern, and members are distinct from all other groups.[26] Separate latent class analyses (finite mixture modelling with categorical latent outcome) with graphical representations were completed for the height, weight, and body mass index growth z-scores. Coded variables identified three growth trajectory classes per participant. Children whose growth trajectory was not within an identified class were not excluded from that growth parameter. Separate linear regression models analysed growth trajectory relationships with peak (peak heart rate, total exercise duration, percentage of predicted maximum heart rate) and submaximal (heart rate and percentage of heart rate reserve during Bruce treadmill stages 1 or 2) exercise parameters. All models were adjusted for age, sex, and CHD severity. Statistical significance was alpha < 0.05. Analyses were performed in SPSS (version 29) or R (version 4.2.1) software. We used the *lcmm* R package to conduct the latent class growth analysis.

Results

Participants

The 213 participants (83 female sex (39.0%), gender unavailable in medical records) had simple ($n = 31$, 14.6%), moderate ($n = 73$, 34.3%), complex ($n = 85$, 39.9%), or functional single ventricle ($n = 24$, 11.3%) CHD diagnoses. All lesions had been treated/repaired as medically appropriate with most (162/213 (76.1%)) having surgical interventions, 17 (8.0%) having catheterisation interventions, and 34 (16.0%) having no previous intervention. Growth trajectories were calculated from 8.9 ± 4.2 hospital visits.

The mean age at the exercise assessment was 12.1 ± 2.9 years (range 6–18 years). Children with complex CHD were repaired at a significantly younger age (0.6 ± 1.3 years) than all other groups (3.0 ± 4.5 to 4.5 ± 5.0 years, $p < 0.001$). The age of repair was unknown for 34 participants (16.0%). Participants achieved 85.8 ± 10.1% of the maximal heart rate predicted for age.[27] Children with moderate CHD achieved a higher ($p = 0.02$) percentage of predicted maximal heart rate (87.6 ± 9.1%) than those with a single ventricle (80.8 ± 12.1%). Those treated by catheterisation achieved a higher ($p = 0.01$) percentage of maximal heart rate (91.3 ± 7.1%) than participants requiring surgery (84.6 ± 10.3%, single ventricle patients excluded). The percentage of predicted maximal heart rate achieved increased with older age (0.5 ± 0.2% increase/year, $p = 0.04$) but was not related to sex ($p = 0.17$) or age of repair ($p = 0.57$).

Latent class analyses

Four distinct z-score trajectories were identified for height (Figure 1), weight (Figure 2), and body mass index (Supplementary 2). Height trajectories differed by exercise test age and weight trajectories differed by age of repair, but growth trajectories were not associated with sex, CHD severity, or type of repair (Supplementary 3). The growth classes were generated through the latent class analysis and therefore reflect the distribution of "groups" of participants with similar growth trajectories. Not all possible growth trajectories were represented by groups of study participants.

Height class 1 ($n = 18$ (10.5%) was below-average height at all ages. Class 2 ($n = 61$ (35.7%) was low-average height at all ages. Class 3 ($n = 21$ (12.3%)) was taller at birth but declined to average height by adolescence. Class 4 ($n = 71$ (41.5%)) was average height at all ages.

Weight class 1 ($n = 55$ (32.0%)) was low-average weight at birth that gradually increased. Class 2 ($n = 102$ (59.3%)) was normal weight at all ages. Class 3 ($n = 10$ (5.8%)) was low-average weight at birth, increased to high average in childhood, and then declined in adolescence. Class 4 ($n = 5$ (2.9%)) was below average at birth that rapidly increased to above average in adolescence.

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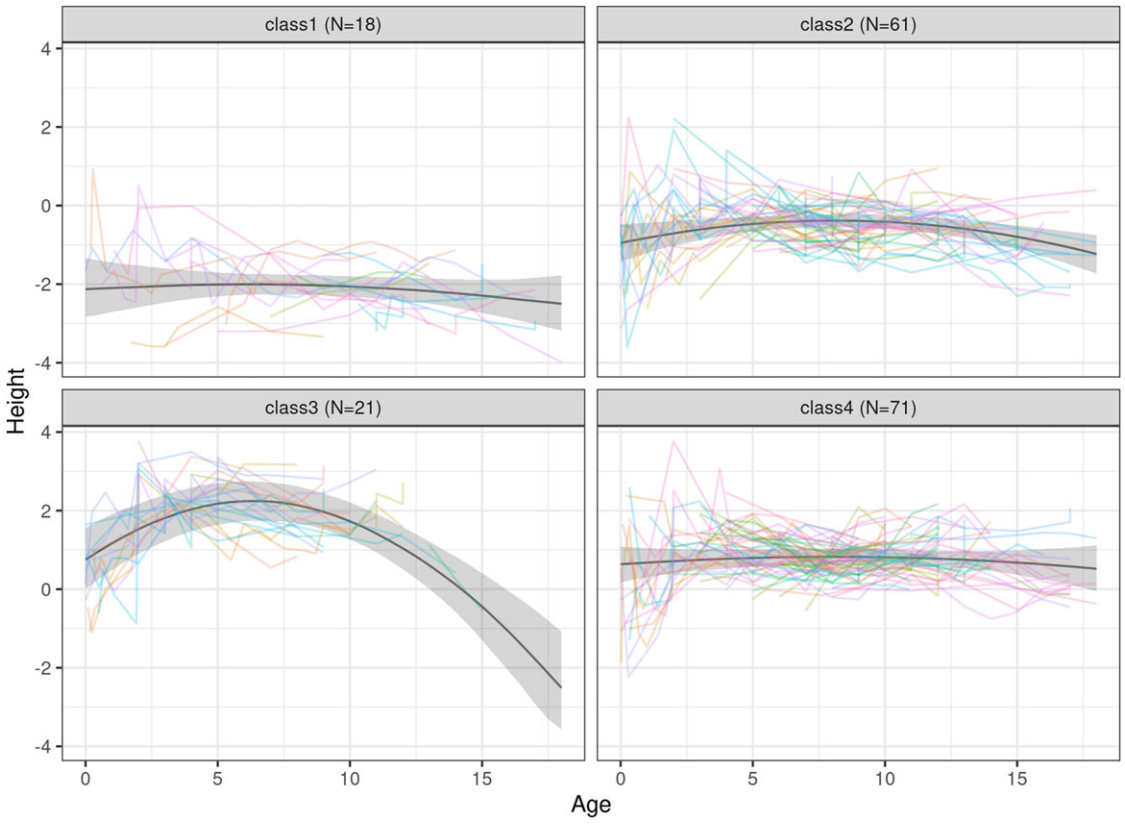

Figure 1. Height z-score trajectory classes.

A normal body mass index at all ages was class 1 ($n = 80$ (47.3%)). Class 2 ($n = 20$ (11.8%)) was normal at birth but declined to low average in adolescence. Class 3 ($n = 41$ (24.3%)) was normal at birth but gradually increased. Class 4 ($n = 28$ (16.6%)) changed from below average at birth to above average by early adolescence.

Growth trajectory and peak exercise capacity

Peak exercise heart rate was associated with height trajectory ($p = 0.02$) and CHD severity ($p < 0.01$, Table 1). Consistently short children (class 1) achieved a lower peak heart rate (15.2 ± 4.9 beats/min). Although singleventricle patients had lower peak heart rates ($p \leq 0.01$), there was no interaction between height trajectory and CHD complexity ($p = 0.38$). Consistently short children (class 1) also achieved a lower percentage of predicted peak heart (7.6 ± 2.4% lower, $p = 0.01$). The percentage of predicted peak heart achieved increased with exercise test age (0.68% per year, $p < 0.01$). Single ventricle patients achieved a lower percentage of predicted peak heart rate ($p \leq 0.01$, Table 2), but there was no height trajectory–CHD complexity interaction ($p = 0.36$). Children whose weight ($p = 0.03$) or body mass index ($p < 0.001$) z-score increased throughout childhood had shorter exercise durations (mean difference: 1–2 min) than children whose trajectory was stable or declined.

Growth trajectories and submaximal exercise capacity

Children with very low (class 1, 132 ± 12 bpm, $p = 0.02$) or low-average (class 2, 139 ± 20 bpm, $p = 0.03$) height had lower heart rates during stage 2. Children who rapidly gained weight (class 4: mean difference: 15.6 ± 8.1%-21.5 ± 8.0%, $p = 0.01$) or whose body mass index increased rapidly (class 4: mean difference: 9.3 ± 3.9%-17.5 ± 4.7%, $p < 0.001$) used a higher proportion of heart rate reserve in stage 2.

Weight ($p = 0.12$), height ($p = 0.14$), and body mass index ($p = 0.60$) trajectories, adjusted for exercise test age, sex, and CHD severity, were not associated with stage 1 heart rate. Children who gained weight faster than expected (class 4: mean difference: 11.4 ± 6.5-15.1 ± 5.5%, $p = 0.03$) or with a rapidly increasing body mass index (class 4, 45.5 ± 12.6%, $p < 0.01$) used a higher proportion of heart rate reserve.

Discussion

Among 213 children with CHD, growth trajectories were related to measures of peak and submaximal exercise capacity, adjusting for age, sex, and diagnosis severity. Height deficits were the primary focus since energy deficits affect weight first and only affect height when energy deficits are extreme.[28] Children whose height was consistently below expected values (class 1) achieved lower peak heart rates and percentages of predicted peak heart rates, suggesting limitations for vigorous exercise. Paradoxically, these children had lower stage 2 heart rates, typically suggestive of higher cardiorespiratory fitness and sufficient capacity for activity with peers. Vigorous exercise deficits may reflect physiological limitations only relevant during maximal effort or limited motivation for vigorous exercise or peak effort symptoms (e.g. shortness of breath, muscle fatigue).

Weight and body mass index trajectories confirmed the negative impact of increasing obesity on exercise capacity recognised among children with/without CHD.[29,30] Children whose weight or body mass index increased rapidly throughout

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Table 1. Bruce treadmill peak heart rate associated with height trajectory and disease severity in children with congenital heart disease

Variable	Beta ± standard rror	Significance	Partial eta squared
Intercept	158.6 ± 8.1	<0.001	0.70
Male sex	5.2 ± 3.0	0.09	0.02
CHD severity[4]			
Simple CHD[1]	14.2 ± 5.9	0.02	0.03
Moderate CHD[2]	16.8 ± 4.9	<0.001	0.07
Complex CHD[3]	15.5 ± 4.9	0.002	0.06
Age at exercise eest	0.5 ± 0.5	0.31	0.01
Stable height trajectory			
Below average (class 1)	−15.2 ± 4.9	0.003	0.06
Low average (class 2)	−2.1 ± 3.3	0.53	0.002
Tall to average (class 3)	1.6 ± 4.7	0.74	0.001

[1]Simple CHD = septal defects, patent ductus arteriosus, mitral valve prolapse.
[2]Moderate CHD = aortic valve stenosis/regurgitation, coarctation/interrupted arch, atrioventricular defect, tricuspid valve stenosis/regurgitation, Ebstein anomaly requiring valve repair (biventricular anatomy), coronary artery anomalies, mitral valve stenosis, restrictive ventricular septal defect with left ventricular dilatation, partial/total anomalous pulmonary venous connection, pulmonary stenosis, truncus arteriosus.
[3]Complex CHD = biventricular diagnoses of transposition of the great arteries, tetralogy of Fallot, coarctation with left and right ventricular dysfunction, tricuspid/pulmonary atresia.
[4]CHD severity = single ventricle patients were the reference group to which other CHD severity groups were compared. Single ventricle diagnoses were double outlet right/left ventricle, double inlet left ventricle, and hypoplastic left heart.

Table 2. Association of height trajectory with percentage of predicted peak heart rate achieved and severity of congenital heart disease

	Height trajectory			
CHD[1] severity	Class[6] 1 (n)	Class 2 (n)	Class 3 (n)	Class 4 (n)
Simple[2]	83.5 ± 6.4 (2)	83.3 ± 12.7 (7)	89.0 ± 1.4 (2)	87.3 ± 8.5 (10)
Moderate[3]	76.8 ± 12.4 (5)	88.0 ± 7.9 (20)	89.0 ± 5.9 (9)	89.2 ± 8.8 (28)
Complex[4]	83.6 ± 9.6 (9)	88.6 ± 6.4 (21)	84.4 ± 10.0 (9)	86.5 ± 8.4 (29)
Single ventricle[5]	71.0 ± 7.1 (2)	78.3 ± 13.1 (13)	95.0 (1)	83.0 ± 13.3 (4)
Total	80.3 ± 10.3	85.6 ± 10.1	87.3 ± 7.9	87.5 ± 8.9

[1]CHD = congenital heart disease.
[2]Simple CHD = septal defects, patent ductus arteriosus, mitral valve prolapse.
[3]Moderate CHD = aortic valve stenosis/regurgitation, coarctation/interrupted arch, atrioventricular defect, tricuspid valve stenosis/regurgitation, Ebstein anomaly requiring valve repair (biventricular anatomy), coronary artery anomalies, mitral valve stenosis, restrictive ventricular septal defect with left ventricular dilatation, partial/total anomalous pulmonary venous connection, pulmonary stenosis, truncus arteriosus.
[4]Complex CHD = biventricular diagnoses of transposition of the great arteries, tetralogy of Fallot, coarctation with left and right ventricular dysfunction, tricuspid/pulmonary atresia.
[5]Single ventricle CHD = double outlet right/left ventricle, double inlet left ventricle, hypoplastic left heart.
[6]Class = latent class analysis results.

childhood had reduced total exercise duration and utilised a higher proportion of heart rate reserve at each submaximal stage. These results align with research associating overweight/obesity among children with CHD with decreased exercise capacity.[31] Sedentary lifestyle health risks are particularly important for those with CHD, whose atherosclerosis risk is increased by the CHD lesion and treatment.[14] Contrary to previous research among those with CHD[32] and the general population,[33] the weight trajectories of only 6 and 3% of study participants were indicative of childhood or adolescent obesity, respectively. The reason for the under-representation of obese participants is unknown but may represent a bias in the selection of patients for the exercise testing protocol. It is possible that obese patients were less likely to be referred for exercising testing or that these patients completed a bicycle exercise protocol to minimise the impact of weight on energy consumption.

Approximately 10% of children in this study were very short at birth, and their short stature was consistent throughout childhood and adolescence. This proportion was four times higher than expected (i.e. due to hereditary short stature) but similar to data for children with CHD.[34] Decreased height occurs among children with single ventricle,[35] but in this study, height trajectory did not differ by CHD severity. This finding aligns with a large study ($n = 856$, children with CHD compared to matched controls),[34] which found decreased weight and length for height among children with simple, complex, and single ventricle CHD. Despite some catch-up growth after surgical repair, children with CHD remain smaller than peers at 3–4 years[34,36] or up to 10 years of age,[15] although differences become smaller and the proportion of children affected decreases.[34,36] In this study, genetic conditions did not account for the height deficit as such children were excluded. Additional research could elucidate the role of familial

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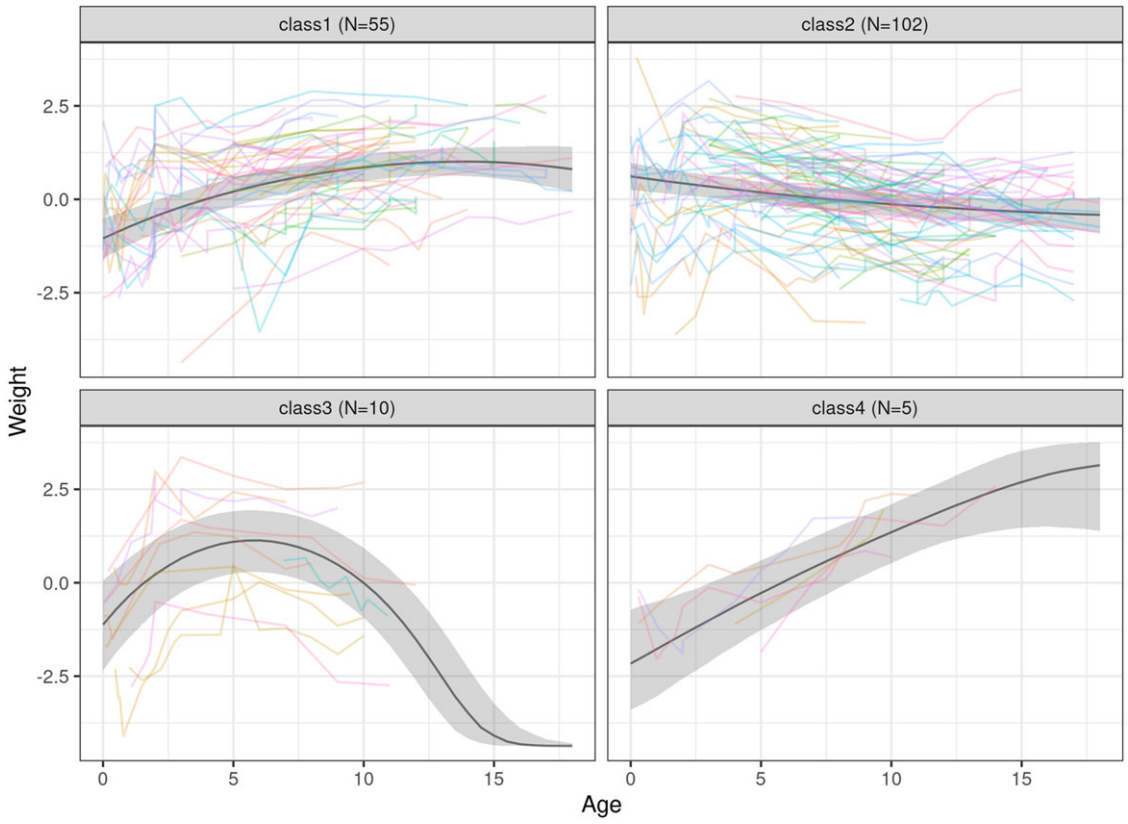

Figure 2. Weight z-score trajectory classes.

growth patterns[37] or CHD-related factors (e.g. nutritional support, surgical repair timing, heart failure, cyanosis, biochemical growth markers) impacting growth.[34]

Previous research has linked adolescent height z-score gains with higher peak exercise capacity among children with single ventricle CHD.[35] The authors suggested that lower height z-scores may be a marker for poor haemodynamics and higher mortality risk.[35] These study results suggest the height z-score trajectory and peak exercise link for children with CHD independent of diagnosis. If a decreased height z-score trajectory reflects a lower energy supply relative to the body's demand, the hypothesis that energy supply could limit physical activity via decreased exercise capacity would be supported. Such a mechanism, not investigated directly in this study, would align with growth-related morbidity/mortality risks in children with CHD.[14,15] Prospective research should investigate exercise capacity, growth, and physical activity changes over time.

It has been suggested that young children with CHD who require supplemental feeding often have an increased weight for height in adolescence,[17] perhaps due to established eating habits or developed preferences for high-calorie foods.[38] In this study, both negative height z-score trajectory and positive weight z-score trajectory were associated with decreased exercise capacity. Monitoring growth patterns through adolescence is recommended as 14% of patients with normal height z-scores prior to 5 years of age did not maintain their growth trajectory into adolescence. Since the limited change in height can signify extreme energy deficits,[28] a change in this growth parameter in the absence of a change to nutritional or physical activity patterns may indicate an increased energy demand from CHD sequelae. Such patients are also at risk of an adequate energy supply to support a healthy active lifestyle. Six percent of adolescents in this study experienced a dramatic decline in weight z-score during adolescence with 3% having a rapidly increasing weight z-score. Interventions to support a physically active lifestyle may be particularly important for children with these height or weight growth profiles.

Limitations

The retrospective study design limited available data and did not allow direct measures of daily physical activity. Nonmetabolic parameters (i.e. heart rate response, exercise duration) assessed exercise capacity with all patients performing the same exercise protocol utilised for published normative data.[39] Few patients had completed direct metabolic energy consumption measures, the gold standard for exercise capacity. Pubertal status or familial growth pattern data were not available. Whether observed growth patterns were related to CHD, genetic or other factors could not be determined. Despite the large range (6–18 years), age was not associated with peak exercise measures, indicating younger participants successfully completed the Bruce protocol. The large number of participants (>200) and results reflecting expected exercise capacity patterns (age, sex, CHD diagnosis) suggest the study population was not significantly biased by these limitations.

Bruce protocol exercise capacity was a proxy for physical activity, assuming that children with greater exercise capacity would be more active. The strong genetic component of exercise capacity[40] requires caution related to this assumption. Since daily physical activity, not cardiorespiratory fitness, is recommended for optimal health,[41] directly measuring changes in daily physical activity and growth over time is recommended.

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Conclusions

Children with CHD whose height z-score was consistently below expected values had limited capacity for vigorous exercise based on significantly lower peak exercise heart rates. These limitations were not related to CHD severity. Since growth limitations impact weight before height, consistently very low height z-score trajectories may reflect inadequate energy to meet demands, potentially impacting physical activity participation. In this study, 10% of children consistently had very low height z-score trajectories, 14% of patients with normal height z-scores prior to 5 years of age did not maintain their growth trajectory into adolescence, 6% experienced a dramatic decline in weight z-score during adolescence, and 3% had a rapidly increasing weight z-score. These results suggest that monitoring growth patterns through adolescence should be recommended. Interventions to support a physically active lifestyle may be particularly important for children with changing height or weight growth profiles. Prospective studies are required to understand growth limitation mechanisms (e.g. heredity, pubertal stage, feeding history, cyanosis, congestive heart failure) and the link to the physical activity associated with optimal mental and physical health.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S1047951125000253.

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Competing interests. The authors declare no competing interests.

Ethical standards. The study was conducted in accordance with the Declaration of Helsinki and approved by the Research Ethics Board of the Children's Hospital of Eastern Ontario (protocol code 11/168X, 21-Dec-2011). Patient consent was waived due to the use of only retrospective data extracted from medical charts.

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