Cardiology in the Young

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Risk factors for development of obesity in an ethnically diverse CHD population

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Original Article

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Abstract

Objectives: Previous cross-sectional studies have demonstrated obesity rates in children with CHD and the general paediatric population. We reviewed longitudinal data to identify factors predisposing to the development of obesity in children, hypothesising that age may be an important risk factor for body mass index growth. *Study design:* Retrospective electronic health records were reviewed in all 5–20-year-old CHD patients seen between 2011 and 2015, and in age-, sex-, and race/ethnicity-matched controls. Subjects were stratified into aged cohorts of 5–10, 11–15, and 15–20. Annualised change in body mass index percentile (BMI%) over this period was compared using paired Student's t-test. Linear regression analysis was performed with the CHD population. *Results:* A total of 223 CHD and 223 matched controls met the inclusion criteria for analysis. Prevalence of combined overweight/obesity did not differ significantly between the CHD cohort (24.6–25.8%) and matched controls (23.3–29.1%). Univariate analysis demonstrated a significant difference of BMI% change in the age cohort of 5–10 (CHD +4.1%/year, control +1.7%/year, $p = 0.04$), in male sex (CHD +1.8%/year, control −0.3%/year, $p = 0.01$), and status-post surgery (CHD 2.03%/year versus control 0.37%, $p = 0.02$). Linear regression analysis within the CHD subgroup demonstrated that age 5–10 years (+4.80%/year, $p < 0.001$) and status-post surgery (+3.11%/year, $p = 0.013$) were associated with increased BMI% growth. *Conclusions:* Prevalence rates of overweight/obesity did not differ between children with CHD and general paediatric population over a 5-year period. Longitudinal data suggest that CHD patients in the age cohort 5–10 and status-post surgery may be at increased risk of BMI% growth relative to peers with structurally normal hearts.

In the last 20 years, the age-adjusted rate of deaths of children aged >1-year-old attributable to CHD has been steadily declining.[1] This has resulted in an increased CHD patient population, estimated at 1 million children under the age of 18 living with CHD in the United States in 2010.[2] Meanwhile, in the general school age and adolescent populations, the prevalence of childhood obesity has more than doubled in the last 30 years.[3] Childhood and adolescent obesity increases the risk of adult obesity as well as risk factors for cardiovascular disease, including hypertension, type 2 diabetes, and hyperlipidaemia.[4,5]

Although children with CHD have specific risks of failure to thrive in infancy, many studies across the United States have demonstrated that the rates of obesity in the paediatric CHD population do not differ from the general paediatric population.[6,7] As individuals with a history of CHD increasingly survive into adulthood, the prevalence of obesity in adults with CHD is rising as well.[8,9]

To-date, most of the published research have reviewed cross-sectional data to compare prevalence of obesity in CHD patients with the general paediatric population and also the obesity rates within the CHD population by type and severity of lesion.[6,7] Although these investigations have demonstrated the risk of obesity in children with CHD, they have not used longitudinal data to identify trends in this patient population or to identify ages at which the risk of obesity develops. Only two recently published studies have investigated the longitudinal data. One of them demonstrated a positive correlation between body mass index z-scores and age in six major cardiac lesions, but did not identify specific age ranges associated with body mass index growth.[10] Another recent investigation characterised long-term body mass index trends in age-stratified cohorts patients who have undergone Fontan repair, but this data is not applicable to the general paediatric CHD population.[11]

We conducted a retrospective longitudinal study to examine the prevalence of overweight and obesity in an ethnically diverse population of children with CHD and also to investigate the risk factors, including age cohorts, associated with paediatric body mass index growth.

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Materials and methods

The study protocol was approved by the Icahn School of Medicine at Mount Sinai Institutional Review Board. Using Epic Software (Epic Systems Corporation, Verona, WI, United States of America) retrospective chart review was performed on all patients aged 5–20 years with an International Classification of Diseases, Ninth Revision visit code for a congenital heart lesion seen by the Mount Sinai Division of Pediatric Cardiology (New York, United States of America) between 2011 and 2015. Age, sex, race/ethnicity, height, weight, body mass index, BMI percentile (BMI%), form of CHD, surgical history, and other clinical characteristics were recorded from the electronic health record. Patients were excluded if they had diagnoses of genetic syndromes.

A control cohort of patients without CHD was randomly generated from the same electronic database of all patients seen within Mount Sinai Hospital for routine health maintenance between 2011 and 2015, matching for age, sex, and race/ethnicity in a 1:1 case–control ratio.

Patients–control pairs were only included in the data analysis if both the CHD patients and paired control had at least two body mass index data points between 2011 and 2015. Subjects were stratified into aged cohorts 5–10, 11–15, and 16+ based on their age in 2015.

BMI% was collected from the electronic health record, as calculated based on Commission for Disease Control body mass index-for-age standards,[12] and were classified as underweight if BMI < 5%, overweight if BMI ⩾ 85% and <95%, and obese if BMI ⩾ 95%.[13,14] CHD severity was classified as "simple", "moderate", or "complex" as per the Bethesda Conference criteria.[15]

Annual prevalence rates of overweight/obese were defined as percentages of the respective cohort with a body mass index data point in that year. BMI% change was calculated as the difference between the subject's most recent BMI% data point and their initial BMI% data point, divided by the number of years between these two data points. Transition to overweight/obese status was defined as samples whose initial recorded BMI% was <85% and whose most recent BMI% was ⩾ 85%. Transition from underweight to normal weight was defined as samples whose initial recorded BMI% was <5% and whose most recent BMI% was >5%.

Normally distributed continuous variables were assessed using paired sample t-test. Dichotomous variables were compared using Pearson's χ^2-test. Dichotomous paired variables were compared using McNemar's test. Linear regression analyses were performed using BMI% change as the dependent variable and age cohort, sex, race/ethnicity, CHD severity, and surgical status as independent variables. A p-value <0.05 was considered statistically significant. Statistical analyses were performed with IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp. Released 2013, Armonk, NY, United States of America).

Results

Of the 712 records reviewed, 446 met the inclusion criteria of at least two BMI% data points collected between 2011 and 2015, of which 223 were CHD and 223 were age-, sex-, and race/ethnicity-matched controls. In aggregate, this group was slightly more male, predominantly fell in the 5–10-year-old bracket at the end of the study period, and was ethnically diverse (Table 1). The CHD sample had a balanced mix of simple (34.1%), moderate (32.2%), and complex (33.6%) disease complexity, and a significant majority (83.4%) had undergone at least one lifetime cardiac intervention, either surgery or cardiac catheterisation.

When cross-tabulated by age cohort, it was found that the 5–10-year-old CHD cohort comprised more simple complexity patients, the 11–15-year-old CHD cohort comprised more males and whites, and the 16+-year-old CHD cohort comprised more post-surgical moderate complexity patients. None of these differences were statistically significant.

Mean BMI% between 2011 and 2015 was normally distributed and did not statistically differ between the CHD sample and paired controls (Table 1). Prevalence of combined overweight/obesity did not differ significantly between the CHD cohort and matched controls, ranging over the 5-year period from 24.6 to 25.8% in the CHD cohort and 23.3–29.1% in the control group. Rates of more severe obesity (as defined by BMI% > 100) ranged between 0.5 and 2.3% in the CHD cohort and 0 and 3.1% in the control group. Rates of underweight did not differ significantly between the CHD and control samples between 2011 and 2015. There was no statistically significant difference in the number of individuals who transitioned to overweight or obese in the 5-year period: 22 (9.9%) of the CHD sample and 16 (7.2%) of the controls ($p = 0.781$); nor was there a statistically significant difference in the number of individuals who transitioned from underweight to normal BMI% in the 5-year period: 22 (9.9%) of the CHD sample and 15 (6.7%) of the controls ($p = 0.310$). The 22 CHD subjects who transitioned to overweight or obese, in comparison to the overall CHD cohort, were more predominantly male (68%), age 5–10 (59%), white (45%), and simple (41%) or complex (41%) complexity. The annualised BMI% change in these 22 subjects was +8% per year.

Paired t-test between CHD samples and their paired controls demonstrated that there was nearly a significant difference in BMI % change per year over the 5-year period (CHD +1.5%/year, control 0.3%/year, $p = 0.055$) (Fig 1). When stratified by age cohort, sex, race/ethnicity, CHD complexity, and surgical status, a significant difference of BMI% change per year was seen in the age 5–10 cohort (CHD +4.1%/year, control +1.7%/year, $p = 0.04$), in male sex (CHD +1.8%/year, control −0.3%/year, $p = 0.01$), status-post surgical status (CHD 2.03%/year versus control 0.37%, $p = 0.02$), and nearly significant in moderate complexity CHD (CHD +0.6%/year, control −1.5%/year, $p = 0.06$). Mean BMI% change was not significantly different in any other subgroup analysis. When assessed within the CHD subjects alone, the only significant difference in BMI% change per year was seen when stratified by age cohort, with an increased BMI% change per year seen in the 5–10 age cohort when compared with the other two age cohorts (age 5–10: +4.1%/year, age 11–15: +0.1%/year, $p = 0.002$; age 5–10: +4.1%/year, age 16+: −0.9%/year, $p < 0.001$). Mean BMI% change did not differ significantly within the CHD subjects with any other subgroup analysis.

Multivariate linear regression analysis within the CHD subgroup demonstrated that age 5–10 and status-post surgical status were associated with increased BMI% growth, +4.80%/year ($p < 0.001$) and +3.11%/year ($p = 0.013$), respectively (Table 2). No other variables within the model were statistically significant.

Discussion

This is the first longitudinal study to assess risk factors for increased BMI in children with CHD. From this large, ethnically

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Table 1. Demographic and clinical characteristics of the study and control cohorts.

Parameter	CHD sample (n = 223)	Control sample (n = 223)	p-value
Age 5–10	95 (42.6%)	95 (42.6%)	1.00
Age 11–15	64 (28.7%)	64 (28.7%)	1.00
Age 16+	64 (28.7%)	64 (28.7%)	1.00
Male	126 (56.5%)	126 (56.5%)	1.00
White	78 (36.4%)	78 (36.4%)	1.00
Black	35 (16.4%)	35 (16.4%)	1.00
Asian	20 (9.3%)	78 (36.4%)	1.00
Hispanic	73 (34.1%)	73 (34.1%)	1.00
Other race/ethnicity	8 (3.7%)	8 (3.7%)	1.00
Simple CHD	76 (34.1%)		–
Moderate CHD	72 (32.2%)		–
Complex CHD	75 (33.6%)		–
Status post surgery	150 (68.8%)		–
Status post catheterisation	31 (14.2%)		–
No intervention	37 (17.0%)		–
BMI% 2011	49.1 ± 3.3	54.6 ± 3.6	0.25
BMI% 2012	53.5 ± 3.0	55.0 ± 3.0	0.72
BMI% 2013	53.7 ± 2.6	56.0 ± 2.7	0.57
BMI% 2014	53.8 ± 2.7	56.7 ± 2.5	0.39
BMI% 2015	57.0 ± 2.6	58.5 ± 2.5	0.66
Overweight/obese 2011	34 (24.6%)	37 (29.1%)	0.39
Overweight/obese 2012	39 (24.7%)	53 (28.8%)	0.27
Overweight/obese 2013	46 (25.8%)	55 (28.5%)	0.47
Overweight/obese 2014	43 (24.7%)	44 (23.3%)	1.00
Overweight/obese 2015	52 (25.0%)	41 (27.0%)	1.00

Categorical variables are expressed as n (%) and continuous variables as mean ± SE. p-values were calculated by Pearson's χ^2-test for categorical variables, McNemar's test for paired categorical variables, and paired t-test for continuous variables
BMI% = Body mass index percentile

diverse sample, we made several important observations, such as prevalence rates of overweight and obesity did not differ significantly between CHD patients and matched healthy controls over a 5-year period; there was an association between the 5 and 10-year-old cohort and increased BMI% growth, and surgically corrected CHD is also associated with increased BMI% growth.

This data adds additional support to the observations made in the last 15 years regarding the prevalence of overweight and obesity in the CHD children.[6,7] This study found that the prevalence rate of combined overweight and obesity in CHD patients not to be dissimilar from their matched healthy controls. The CHD cohort's combined overweight and obesity prevalence rates of 24.6–25.8% within each year of the study were not dissimilar from rates reported in CHD populations in other prominent studies.

This longitudinal study design permits the calculation of annualised body mass index growth rates. There was no significant difference in the transition from underweight to normal weight between the CHD and matched healthy controls, nor were there differences between the mean body mass index or combined overweight and obesity prevalence over the 5-year period. Therefore, one can conclude that the BMI% increase seen in the CHD sample relative to their matched controls does not reflect a relatively underweight CHD population "catching up" to the appropriate weights of their matched healthy controls.

We identified the age cohort of 5–10-year-old as a risk factor of increased BMI% growth, with an effect size measured at 4.80% per year. Tamayo et al reported increasing age as a risk factor for increased body mass index z-score.[10] No previous study has reported specific ages associated with increased body mass index growth. Literature in the field of paediatric obesity has demonstrated that in obese children, a decrease in body mass index-standard deviation score of 0.25–0.5 over 1 year significantly improves atherogenic profile, insulin resistance, and

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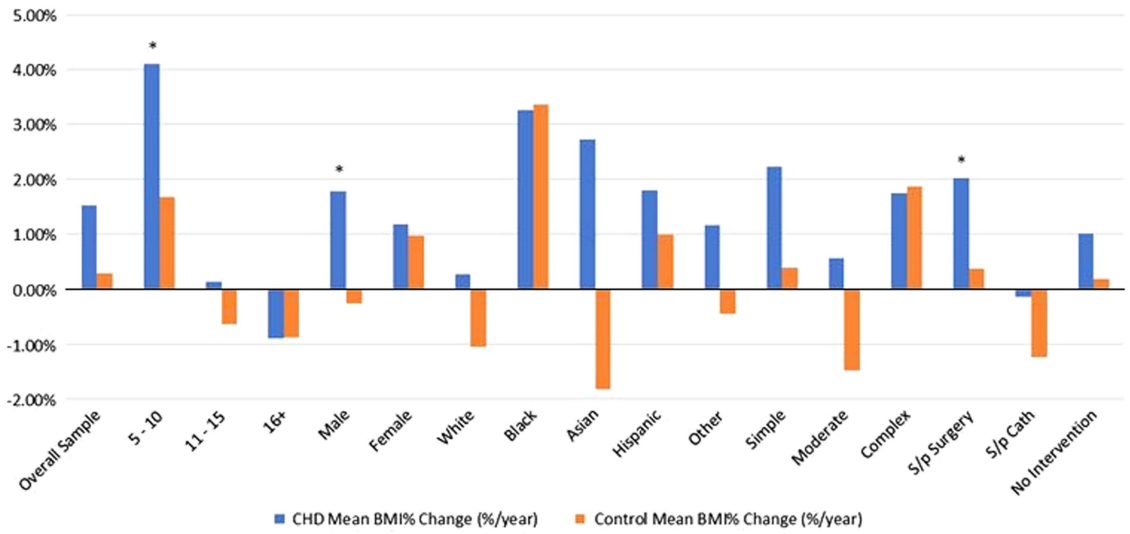

Figure 1. Mean BMI% change per year, CHD sample versus paired controls, stratified by overall sample and demographic subgroups. *denotes p-value < 0.05.

Table 2. Multivariate linear regression within CHD subgroup.

	Effect size, BMI% growth per year	p-value
Age 5–10	4.800	**<0.001**
Age 11–15	1.122	0.37
Male	0.842	0.38
Black	1.978	0.16
Asian	2.148	0.21
Hispanic	1.458	0.19
Simple	2.307	0.11
Moderate	−0.171	0.89
S/p surgery	3.110	**0.013**

S/p = status-post

hypertension.[16,17] Converted to a percentile basis from a baseline z-score of 2.0, this implies that a 1.73–4.40% percentile decrease is associated with improved cardiovascular risk factors. The age range of 5–10 may therefore be a critical time to provide weight management counselling to children and parents.

Status-post surgery was appreciated as a risk factor for increased BMI% growth. Similarly, Tamayo et al demonstrated weight z-scores increasing over time following complete surgical repair.[10] Many previous studies have implicated that this association may be related to exercise restrictions that may be incorrectly placed on the surgically corrected child.[18–20] Given that children and adolescents with CHD and their parents have been shown to have a poor knowledge of the appropriate level of exercise activity,[21,22] our data suggest that there may be an efficacy to more consistent exercise counselling with this patient population.

Our study is limited by its retrospective design and reliance on the accuracy and availability of coded information within the electronic health record. Therefore, important data was unable to be collected, including parental obesity, socio-economic status, patient-reported exercise tolerance, echocardiographic data, or exercise capacity testing. Though our data is longitudinal, it is limited to a 5-year period and may miss the trends seen over a longer period of time. In addition, our sample is drawn from a single academic centre in New York City. Although our patient demographics may differ from nationwide characteristics, it has afforded our study with significant ethnic diversity not seen in previous longitudinal studies.

Conclusions

In this retrospective longitudinal study, prevalence rates of combined overweight and obesity did not differ between CHD and general paediatric populations over a 5-year period. Longitudinal data suggest that age 5–10 and status-post surgery CHD patients may be at increased risk of BMI% growth relative to peers with structurally normal hearts. A higher power prospective study could further elicit these relationships.

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Conflicts of interest. None.

References

1. Benjamin EJ, Blaha MJ, Chiuve SE, et al. Heart Disease and Stroke Statistics-2017 update: a report from the American Heart Association. Circulation 2017; 135: e146–e603.
2. Gilboa SM, Devine OJ, Kucik JE, et al. Congenital heart defects in the United States: estimating the magnitude of the affected population in 2010. Circulation 2016; 134: 101–109.
3. Ogden CL, Carroll MD, Lawman HG, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988–1994 through 2013–2014. J Am Med Assoc 2016; 315: 2292–2299.
4. The NS, Suchindran C, North KE, Popkin BM, Gordon-Larsen P. Association of adolescent obesity with risk of severe obesity in adulthood. J Am Med Assoc 2010; 304: 2042–2047.

https://doi.org/10.1017/S1047951118001889 Published online by Cambridge University Press

5. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS. The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. Pediatrics 2005; 115: 22–27.
6. Pinto NM, Marino BS, Wernovsky G, et al. Obesity is a common comorbidity in children with congenital and acquired heart disease. Pediatrics 2007; 120: e1157–e1164.
7. Shustak RJ, McGuire SB, October TW, Phoon CK, Chun AJ. Prevalence of obesity among patients with congenital and acquired heart disease. Pediatr Cardiol 2012; 33: 8–14.
8. Lerman JB, Parness IA, Shenoy RU. Body weights in adults with congenital heart disease and the obesity frequency. Am J Cardiol 2017; 119: 638–642.
9. Zaidi AN, Bauer JA, Michalsky MP, et al. The impact of obesity on early postoperative outcomes in adults with congenital heart disease. Congenit Heart Dis 2011; 6: 241–246.
10. Tamayo C, Manlhiot C, Patterson K, Lalani S, McCrindle BW. Longitudinal evaluation of the prevalence of overweight/obesity in children with congenital heart disease. Can J Cardiol 2015; 31: 117–123.
11. Chung ST, Hong B, Patterson L, Petit CJ, Ham JN. High overweight and obesity in Fontan patients: a 20-year history. Pediatr Cardiol 2016; 37: 192–200.
12. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. Vital Health Stat 11 2002; 246: 1–190.
13. Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. The Expert Committee on clinical guidelines for overweight in adolescent preventive services. Am J Clin Nutr 1994; 59: 307–316.
14. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. Pediatrics 1998; 102: E29.
15. Warnes CA, Williams RG, Bashore TM, et al. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to develop guidelines on the management of adults with congenital heart disease). Developed in Collaboration with the American Society of Echocardiography, Heart Rhythm Society, International Society for Adult Congenital Heart Disease, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. J Am Coll Cardiol 2008; 52: e143–e263.
16. Reinehr T, Lass N, Toschke C, Rothermel J, Lanzinger S, Holl RW. Which amount of BMI-SDS reduction is necessary to improve cardiovascular risk factors in overweight children? J Clin Endocrinol Metab 2016; 101: 3171–3179.
17. Reinehr T, Andler W. Changes in the atherogenic risk factor profile according to degree of weight loss. Arch Dis Child 2004; 89: 419–422.
18. Pasquali SK, Marino BS, Pudusseri A, et al. Risk factors and comorbidities associated with obesity in children and adolescents after the arterial switch operation and Ross procedure. Am Heart J 2009; 158: 473–479.
19. Stefan MA, Hopman WM, Smythe JF. Effect of activity restriction owing to heart disease on obesity. Arch Pediatr Adolesc Med 2005; 159: 477–481.
20. Cohen MS. Clinical practice: the effect of obesity in children with congenital heart disease. Eur J Pediatr 2012; 171: 1145–1150.
21. Kendall L, Parsons JM, Sloper P, Lewin RJ. A simple screening method for determining knowledge of the appropriate levels of activity and risk behaviour in young people with congenital cardiac conditions. Cardiol Young 2007; 17: 151–157.
22. Cheuk DKL. Parents' understanding of their child's congenital heart disease. Heart 2004; 90: 435–439.

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