Overweight and obesity in children with congenital heart disease: combination of risks for the future?

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Abstract

Background: Children who have unhealthy lifestyles are predisposed to develop hypertension, dyslipidemia and other complications. The epidemic of obesity is also affecting children with congenital heart disease. The aim of this study is to estimate the prevalence of obesity and describe associated risk factors, including family history in children with congenital heart disease.

Methods: A cross-sectional study with 316 children and adolescents with congenital heart disease seen in an outpatient clinic of a reference hospital. Collected sociodemographic data included family history of chronic disease, dietary habits, laboratory tests (total cholesterol, HDL and LDL/cholesterol, triglycerides, fasting glucose, CRP, hematocrit and hemoglobin), and anthropometric assessment. Anthropometric data of the caregivers was self-reported.

Results: The prevalence of excess weight was 26.9%. Altered levels of total cholesterol were observed in 46.9%, of HDL in 32.7%, LDL in 23.6% and of triglycerides levels in 20.0%. A higher frequency of family history of obesity (42.6%; p = 0.001), dyslipidemia (48.1%; p = <0.001), diabetes (47.4%; p = 0.002), hypertension (39.2%; p = 0.006) and ischemic disease (43.7%; p = 0.023), as well as significantly higher values of triglycerides (p = 0.017), glycemia (p = 0.004) and C-reactive protein (p = 0.002) were observed among patients with excess weight.

Conclusion: The presence of modifiable risk factors and the variables associated to excess weight in this population was similar to that described in the literature for children without congenital disease. As these children already present the risks associated to heart disease, it is particularly important to promote a healthy lifestyle in this group.

Keywords: Child, Adolescent, Congenital heart disease, Overweight, Ischemic disease

Background

During the last three decades, there has been a considerable increase in the prevalence of obesity in children and adolescents (4–18 year-old) worldwide [1-3]. Children and adolescents with unhealthy lifestyles are predisposed to develop hypertension, dyslipidemia and other complications [4]. These factors, as well as physical inactivity, may track into adulthood [5] and increase the risk of chronic diseases such as atherosclerosis [1].

The epidemic of obesity is also affecting children with congenital heart disease (CHD). More than one quarter of this population is already overweight [6,7]. Two main causes have been described: physical activity restrictions and interventions for weight gain in infancy, when many lesions cause undernutrition [5]. These interventions often include consumption of increased calories and foods with high fat and sodium content [8,9]. Although nutritional requirements and physical functional capacity change as these children grow older and their heart lesions are successfully treated, the inappropriate dietary
behaviors and physical inactivity are frequently maintained across childhood [10]. The family frequently influences these unhealthy behaviors, both directly, restricting physical activity, for example, and indirectly, by setting an unhealthy model. When parents are obese, as one example, the risk of obesity in their children is increased [11-14].

Therefore, the objective of the present study was to estimate the prevalence of overweight, obesity and associated physical activity habits, passive smoking, glycemia and lipids in children with congenital heart disease. We also sought to investigate cardiovascular risk factors present in children’s families.

Methods

We conducted a cross-sectional study of 316 patients with congenital heart disease, aged between 2 and 18 years, and receiving outpatient care at the Pediatric Cardiology Outpatient Clinic of a referral hospital between September 2010 and March 2013. The study protocol was approved by the Institutional Research Ethics Committee of Instituto de Cardiologia do Rio Grande do Sul, Brazil (4470/2010).

Patients who had innocent murmur, clinical conditions that prevented anthropometric assessment (wheelchair users, malformation of the lower limbs, etc.), genetic syndromes or children without a diagnosis of structural heart disease were excluded from the study.

Data collection was performed according to a weekly list of patients scheduled for routine outpatient visits. Based on this list, the children’s guardians were contacted by telephone, and the patients were invited to participate in the study. Those who accepted to participate were asked to fast for 12 hours before laboratory tests. Patients who could not be reached by phone were invited to participate during the medical visit, and their laboratory tests were scheduled for another day.

All patients and guardians received information about the study and, after accepting to participate, signed the written consent form. Next, patients underwent collection of blood samples and anthropometric assessment. The participants’ caregivers present during data collection provided information about family risk factors and physical activity habits (International Physical Activity Questionnaire-IPAQ short version) [15]. Data were collected using a questionnaire administered by health professionals who attended two specific training sessions and received training updates regularly. After being assessed, participants who showed abnormal results were referred to multidisciplinary outpatient care for prevention and treatment of risk factors.

Weight was measured to the nearest 0.1 kg and height to the nearest centimeter using a Welmy electronic digital scale with stadiometer, with 200 Kg capacity, with the child standing, without shoes or heavy clothing. Nutritional status was based on body mass index (BMI), and classified using the software WHO Anthro and Anthro Plus. Cutoff points for underweight/normal weight (<85th percentile) and excess weight (>85th percentile being overweight 85th−95th percentile and obesity > 95 percentile) for BMI values were used according to the WHO-2006/2007 [16].

Blood was collected by peripheral venous puncture after 12 h fasting. The hematocrit and hemoglobin were determined using whole blood collected with ethylenediaminetetraacetic acid (EDTA), in an automated analyzer (Coulter Act, Coulter, USA), Biochemical analysis of total cholesterol, LDL, HDL cholesterol and triglycerides were determined in serum obtained by centrifugation of blood samples, through enzymatic method on an automated analyzer (Selectra E, Vital Scientific, USA), using reagent kits and protocols according to instructions of the manufacturer. Levels of hs-CRP were determined in serum by nephelometry, using a Behring Nephelomefer 100 Analyzer (Dade Behring, USA).

Blood tests were considered abnormal according to the U.S. pediatric guidelines (2011) and the I Brazilian Guidelines for Prevention of Atherosclerosis in Childhood and Adolescence (2005): total cholesterol >170 mg/dL, HDL/cholesterol < 45 mg/dL, LDL/cholesterol > 110 mg/dL, triglycerides > 75 mg/dL (2–9 years) or > 90 mg/dL (10–18 years) [17], fasting glucose > 100 mg/dL, CRP > 0.30 mg/dL, hematocrit < 35%, and hemoglobin < 11.0 g/dL [18].

Sample size was estimated as 250 children and adolescents, based on the prevalence of obesity observed in a previous study [19], with absolute error margins ranging from 3% to 6% with a confidence level of 95%.

Data were stored and analyzed using the computer program SPSS, version 17.0. The prevalence rates were expressed as percentages with 95% confidence intervals. The association between risk factors was assessed using the chi-square test or Fischer’s exact test. Differences between the groups with and without risk factors were evaluated using the Student t test or Mann–Whitney test for continuous variables and the chi-square test or Fisher’s exact test for categorical variables (gender, total cholesterol, HDL/cholesterol, LDL/cholesterol, triglycerides, hematocrit, hemoglobin, glucose, BMI percentile). Poisson multiple logistic regression analysis was adjusted for family history (obesity, dyslipidemia, diabetes, hypertension, and ischemic heart disease), mother’s nutritional status, both parents’ nutritional status, and adolescents’ age. Statistical significance was set at p-value ≤ 0.05.

This report is presented as suggested by the STROBE statement: guidelines for reporting observational studies [20].

Results

A total of 341 patients were interviewed, but 25 did not collect blood and were excluded from analysis, resulting
in 316 participants. Most participants were male (55.7%), Caucasian (81.6%) and aged between 6 and 11 years (43.7%). The majority had been born at term (83.2%) and had acyanotic congenital heart disease (81.1%). The proportion of passive smoking was reported to be 43.7% (Table 1).

Family history of cardiovascular risk factors included excess weight in 44.3%, dyslipidemia in 53.8%, diabetes in 49.7%, arterial hypertension in 83.2%, and ischemic disease in 52.2% (Table 1).

The prevalence of excess weight (BMI ≥ 85th percentile) was 26.9%; of these, 17.4% were overweight (BMI > P85 ≤ P95) and 9.5% were obese (BMI > P95). Excess weight was more common among boys (60%). In the group of 6–11 years old, 34.1% presented with excess weight (p = 0.009). The group of acyanotic congenital heart disease showed 27.7% of overweight, while in patients with cyanotic lesions the proportion was 23.3% (Table 2).

Regarding physical activity classification, children and adolescents with excess weight were very active in 20%, active in 36.5% and irregularly active in 40%, while eutrophic children were very active in 19.1%, active in 38.7% and irregularly active in 35.7% (p = 0.802).

There were 165 mothers (52.2%), and 92 fathers (29.1%) with excess weight. Mothers’ and both parents’ excess weight was significantly associated with children’s excess weight (p = 0.003 and 0.049, respectively). The Prevalence Ratio of an excess weight mother to have an excess weight child was 1.24 (CI 1.08-1.43).

As shown in Table 3, the excess weight group had more often a positive family history (first degree relative) for obesity (p = 0.002), dyslipidemia (p < 0.001), diabetes (p = 0.005), hypertension (p = 0.010), and ischemic disease (p = 0.040). The prevalence ratio for excess weight in children was 1.92 (CI 1.22 ± 3.02, p = 0.005) when the mother had excess weight and 1.74 (CI 1.15 ± 2.62; p = 0.009) when there was a positive family history for dyslipidemia.

Table 4 presents the laboratory tests results, showing that 32.7% had low HDL, 18.4% had high total cholesterol, 11.4% had high LDL, and 32.0% had increased triglycerides. The excess weight group had significantly higher triglycerides (p = 0.017), glucose (p = 0.004), and C-reactive protein (p = 0.002).

Discussion
The present study reports a high prevalence of excess weight in children and adolescents with congenital heart disease. Additionally, we observed a high frequency of excess weight in parents and a positive family history for chronic non-transmissible diseases.

The prevalence of overweight and obesity in children with congenital heart disease was similar to that described in the literature for children with non-congenital disease [19,21] In a population of patients with congenital heart disease in the U.S., researchers found a prevalence of more than 25% of obese and overweight children [22]. However, in a study published six years ago, the excess weight rate of a population of children and adolescents in Belgium was 7.6% [11].

In Brazil, the high prevalence of excess weight in children and adolescents in general has been a reason for concern, because other associated risk factors for ischemic heart disease, such as hypertension, glucose

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**Table 1 General characteristics of the population**

| Variables                              | n = 316 (%) |
|----------------------------------------|-------------|
| Male                                   | 176 (55.7)  |
| White                                  | 258 (81.6)  |
| Age                                    |             |
| Preschool age (2–5 years)              | 67 (21.2)   |
| School age (6–11 years)                | 138 (43.7)  |
| Adolescents (12–18 years)              | 111 (35.1)  |
| Born at term                           | 263 (83.20) |
| Heart disease                          |             |
| Acyanotic                              |             |
| Ventricular Septal Defect (VSD)        | 76 (24.1)   |
| Atrial Septal Defect (ASD)             | 61 (19.3)   |
| Miscellaneous                          | 119 (37.7)  |
| Cyanotic                               |             |
| Tetralogy of Fallot                    | 43 (13.6)   |
| Pulmonary Atresia                      | 6 (1.9)     |
| Miscellaneous                          | 11 (3.4)    |
| Father’s educational level             |             |
| Elementary school                      | 191 (68.0)  |
| High school                            | 79 (28.1)   |
| Incomplete/Complete higher education   | 11 (3.9)    |
| Mother’s educational level             |             |
| Elementary school                      | 184 (60.7)  |
| High school                            | 94 (31.0)   |
| Incomplete/Complete higher education   | 25 (8.3)    |
| Number of siblings                     |             |
| Only child                             | 67 (21.2)   |
| Siblings                               | 249 (78.8)  |
| Positive family history for            |             |
| Excess weight                          | 140 (44.3)  |
| Dyslipidemia                           | 170 (53.8)  |
| Diabetes                               | 157 (49.7)  |
| Hypertension                           | 263 (83.2)  |
| Heart disease/ischemia                 | 165 (52.2)  |
| Presence of smokers in the household   | 138 (43.70) |
intolerance, dyslipidemia, and physical inactivity have emerged [7,23-27].

The presence of modifiable risk factors for ischemic heart disease in this population, such as an abnormal lipid profile (high total cholesterol/LDL/triglycerides, low HDL) and excess weight may lead individuals with congenital heart disease to have a combination of risks that may persist into adulthood [4,28]. These modifiable risk factors have been well discussed in the literature about children without heart disease [2,21].

The presence of chronic diseases in families of patients with congenital heart disease is an additional risk factor for ischemic heart disease in this population, such as an abnormal lipid profile (high total cholesterol/LDL/triglycerides, low HDL) and excess weight may lead individuals with congenital heart disease to have a combination of risks that may persist into adulthood [4,28]. These modifiable risk factors have been well discussed in the literature about children without heart disease [2,21].

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In our study, approximately half of children and adolescents were irregularly active or sedentary. In many cases, physical activity may be limited by the parents anxiety [35].

Passive smoking was detected in almost half of the population studied, a rate much higher than in a survey conducted over the past decade, in which more than 25% of children lived with at least one smoking parent. Exposure to secondhand smoking in children causes higher rates of pneumonia, ear infections, sudden infant death syndrome, asthma, and other negative health effects [36]. In addition, children’s airways are more vulnerable, suffering dramatically with the effects of secondhand smoking [37]. Children exposed to tobacco smoke at a young age are more likely to become smokers and continue the cycle of smoking in adulthood [38].

It is important to consider that factors present since the children’s conception may contribute to “programming” of disease in adult life [39,40]. The quality of the mother’s nutrition during pregnancy may affect the fetal

### Table 2 Distribution of general characteristics of the population according to the BMI classification of individuals with congenital heart disease

| Variables                  | Total 316 (%) | Underweight/Normal weight 231 (%) | Excess weight 85 (%) | PR   | CI (95%) | p       |
|----------------------------|---------------|-----------------------------------|----------------------|------|----------|---------|
| Male                       | 176 (55.7)    | 125 (54.1)                        | 51 (60)              | 1.20 | 0.84-1.74| 0.321   |
| Only child                 | 67 (21.2)     | 49 (21.2)                         | 18 (21.2)            | 1.0  | 0.65-1.55| 0.99    |
| Preterm birth              | 49 (15.5)     | 31 (13.4)                         | 18 (21.2)            | 1.53 | 1.01-2.32| 0.046   |
| Age                        |               |                                   |                      |      |          |         |
| Preschool age (2–5 years)  | 67 (21.2)     | 57 (24.7)                         | 10 (11.8)            | 1.0  | -        | -       |
| School age (6–11 years)    | 138 (43.7)    | 91 (39.4)                         | 47 (55.3)            | 2.29 | 1.24-4.23| 0.008   |
| Adolescent (12–18 years)   | 111 (35.1)    | 83 (35.9)                         | 28 (32.9)            | 1.71 | 0.89-3.27| 0.104   |
| Congenital heart disease   |               |                                   |                      |      |          |         |
| Cyanotic                   | 60 (19)       | 46 (19.9)                         | 14 (16.5)            | 0.84 | 0.51-1.37| 0.48    |
| Acyanotic                  | 256 (81)      | 185 (80.1)                        | 71 (83.5)            | 1.0  | -        | -       |

PR: prevalence ratio; CI: confidence interval.

### Table 3 Family history of obesity and chronic diseases according to the BMI categories of individuals with congenital heart disease

| Variables                        | Total 316 (%) | Underweight/Normal weight 231 (%) | Excess weight 85 (%) | PR   | CI (95%) | P       |
|----------------------------------|---------------|-----------------------------------|----------------------|------|----------|---------|
| 1st-degree relative with obesity| 68 (21.5)     | 39 (16.9)                         | 29 (34.1)            | 1.26 | 1.07-1.49| 0.002   |
| dyslipidemia                     | 52 (16.5)     | 27 (11.7)                         | 25 (29.4)            | 1.25 | 1.08-1.45| <0.001  |
| diabetes                         | 38 (12.0)     | 20 (8.6)                          | 18 (21.2)            | 1.16 | 1.03-1.3 | 0.005   |
| hypertension                     | 74 (23.4)     | 45 (19.5)                         | 29 (34.1)            | 1.22 | 1.04-1.44| 0.010   |
| heart disease/ischemic disease   | 32 (10.1)     | 18 (7.8)                          | 14 (16.5)            | 1.1  | 0.99-1.22| 0.040   |

PR: prevalence ratio; CI: confidence interval.
The present study has some limitations that deserve to be mentioned. Possible confounding biases may be related to memory bias and underreporting of information by the respondents. Cross-sectional designs do not allow causal inferences or detailed evaluation of sequences of events. Despite these limitations, to the best of our knowledge, this is one of the largest series of patients with congenital heart disease evaluated for these risk in Brazil or other developing countries.

Conclusions
The obesity epidemic also affects children and adolescents with congenital heart disease. In this population, factors inherent to the heart disease can be added to other traditional risk factors for the development of ischemic heart disease in the future. Changes in the lifestyle are necessary to change these risk factors and its comorbidities in the adult life of these people who are living longer.

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References
1. Kelishadi R, Hashempour M, Sheikh-Heidar A, Ghareh-Samani S: Changes in serum lipid profile of obese or overweight children and adolescents following a lifestyle modification course. AHA Atheroscler 2012, B143–148.
2. Gupta N, Goel K, Shah P, Misra A: Childhood obesity in developing countries: epidemiology, determinants, and prevention. Endocr Rev 2012, 33:48–70.
3. Organization WH: Facts and figures on childhood obesity. 2014. http://www.who.int/dietphysicalactivity/end-childhood-obesity/facts/en/ Accessed in 06/26/2014.
4. Moons P, Deyk KF, Deroog D, Toot E, Buds W: Prevalence of cardiovascular risk factors in adults with congenital heart disease. Eur J Cardiovasc Prev Rehabil 2006, 13:612–616.
5. Muller-Riemenschneider F, Nocon M, Willich SN: Prevalence of modifiable cardiovascular risk factors in German adolescents. Eur J Cardiovasc Prev Rehabil 2010, 17:204–210.
6. Shustak RJ, McGuire SL, October TW, Phoon CK, Chun AJ: Prevalence of obesity among patients with congenital and acquired heart disease. Pediatr Cardiol 2012, 33:8–14.
7. Cohen MS: Clinical practice: the effect of obesity in children with congenital heart disease. Eur J Pediatr 2012, 171:1145–1150.
8. Hansen SR, Dorup I: Energy and nutrient intakes in congenital heart disease. Acta Paediatr 1993, 82:166–172.
9. Ratanachu-Ek S, Pongdara A: Nutritional status of pediatric patients with congenital heart disease: Pre- and post cardiac surgery. J Med Assoc Thai 2011, 94(Suppl 3):S133–S137.
10. Moola F, Fusco C, Kirsh JA: The perceptions of caregivers toward physical activity and health in youth with congenital heart disease. Qual Health Res 2011, 21(2):278–291. United States.
11. Massin MM, Hövels-Gürich H, Seghaye MC: Atherosclerosis lifestyle risk factors in children with congenital heart disease. Eur J Cardiovasc Prev Rehabil 2007, 14:349–351.
12. Martinez-Quintana E, Rodriguez-Gonzalez F, Nieto-Lago V, Novoa FJ, Lopez-Rios L, Riano-Ruiz M: Serum Glucose and Lipid Levels in Adult Congenital Heart Disease Patients. Metabolism. United States. 2010 Blevick Inc; 2010.1642–1648.
13. Yoshinaga M, Takahashi H, Shinomiya M, Miyazaki A, Kurihara Y, Ichiha F: Impact of having one cardiovascular risk factor on other cardiovascular risk factor levels in adolescents. J Atheroscler Thromb 2010, 17:1167–1175.
14. Murrin CM, Kelly GE, Tremblay RE, Kelleher CC: Body mass index and height over three generations: evidence from the lifeways cross-generational cohort study. BMC Public Health 2012, 12:61.
15. Pardini R: Validation of the international physical activity questionnaire (ipaq version 6): Pilot Study in Brazilian young adults. Rev Bras Ciên e Mov 2001, 9:45–51.
16. De Onis M, World Health Organization, Dept. of Nutrition for Health and Development: Who Child Growth Standards. Methods and Development Length/Height-for-age, Weight-for-age, Weight-for-Length, Weight-for-Height and Body Mass Index-for-age. Geneva: 2006.
17. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. Pediatrics 2011, 128(Suppl 5):S213–S256.
18. Back Giuliano Ide C, Caramelli B, Pelanda L, Duncan B, Mattos S, Fonseca FH, Sociedade Brasileira de Cardiologia: I diretriz de prevenção da aterosclerose na infância e na adolescência. Arq Bras Cardiol 2005, 85:54–536.

Table 4 Laboratory tests according to the BMI categories of individuals with congenital heart disease

| Variables         | Underweight/Normal weight mean ± SD | Excess weight mean ± SD | p    |
|-------------------|-------------------------------------|-------------------------|------|
| Cholesterol       | 148.1 ± 25.6                        | 154 ± 32.2              | 0.106|
| HDL               | 50.3 ± 11.3                         | 48.9 ± 12.2             | 0.352|
| LDL               | 83.3 ± 21.7                         | 89.3 ± 27.8             | 0.053|
| Triglycerides     | 72.6 ± 34.3                         | 83.7 ± 38.8             | 0.007|
| Glucose           | 87 ± 9.5                            | 90.6 ± 9.0              | 0.004|
| C-reactive protein| 0.1 ± 0.1                           | 0.2 ± 0.2               | 0.002|
| Hematocrit        | 39.1 ± 4.1                          | 38.8 ± 3.4              | 0.523|
| Hemoglobin        | 13.3 ± 1.9                          | 12.9 ± 1.5              | 0.099|

SD: standard deviation.

metabolism and the child’s taste and attitudes towards food [41]. Along the life course, these factors interact with family habits and childhood risks to compose different health and disease pathways [14].

Abbreviations
BMI: Body mass index; CHD: Congenital heart disease; DBP: Diastolic blood pressure; EDTA: Ethylenediaminetetraacetic acid; SBP: Systolic blood pressure.

Competing interests
The authors declare that they have no competing interests.

Authors’ contributions
SMB: Substantial contributions to conception and design, collection of data, acquisition of data, analysis, interpretation of data and drafting the article. CSG, DSS: Participation in collection of data, acquisition and analysis of data. CCC, ROP: contributions to conception and design, analysis, interpretation of data, revising the article critically for important intellectual content. LCP: Substantial contributions to conception and design, acquisition of funding, analysis, interpretation of data, drafting the article, revising and drafting the article critically for important intellectual content; and final approval of the version to be published. All authors read and approved the final manuscript.
Overweight, obesity and other risk factors for IHD in Brazilian schoolchildren. Public Health Nutr 2009, 12(5):710–715. England.

Elm E, Altman D, Egger M, Pocock S, Gotzsche P: the strengthening of reporting of observational studies in epidemiology (STROBE) statement: Guidelines for reporting observational studies. 2007, 370:1453–1457.

Messiah SE, Arheart KL, Nazario RA, Hlaing WM, Lipschultz SE, Miller TI, BMI, waist circumference, and selected cardiovascular disease risk factors among preschool-age children. Obesity (Silver Spring) 2012, 1942–1949. United States.

Pinto NM, Marino BS, Wernovsky G, de Ferranti SD, Walsh AZ, Laonde M, Hyland K, Dunn SO, Cohen MS. Obesity is a common comorbidity in children with congenital and acquired heart disease. Pediatrics 2007, 120:e1157–e1164.

Giowinski B, Urban M, Koup A: Cardiovascular risk factors in children with obesity, hypertension and diabetes: Lipoprotein(a) levels and body mass index correlate with family history of cardiovascular disease. Eur J Pediatr 2010, 161:511–518.

Update on the 1987 task force report on high blood pressure in children and adolescents: A working group report from the national high blood pressure education program. National high blood pressure education program working group on hypertension control in children and adolescents. Pediatrics 1996, 98:649–658.

Pall D, Kiss I, Karona E: Importance of ambulatory blood pressure monitoring in adolescent hypertension. Kidney Blood Press Res 2012, 35(2):129–134. Switzerland: Basel.

Tremblay MS, LeBlanc AG, Kho ME, Saunders TJ, Larouche R, Colley RC, Goldfield G, Connor Gorber S: Systematic review of sedentary behaviour and health indicators in school-aged children and youth. Int J Behav Nutr Phys Act 2011, 9:88. England.

Rivera IR, Silva MA, Silva RD, Oliveira BA, Carvalho AC: Physical inactivity, tv-watching hours and body composition in children and adolescents. Arq Bras Cardiol 2010, 95:159–165.

Massin M: the cardiovascular risk in children with congenital heart disease. Arch Mal Coeur Vaiss 2007, 100(5):448–453. France.

Pasquali SK, Marino BS, Pudusseri A, Wernovsky G, Paridon SM, Walker SA, Cohen MS: 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.

Costa Silva Zemdegs J, Barreto Corli S, De Castro Coelho L, Duarte Pimentel G, Toyomi Hirai A, Sachs A: Lipid profile and cardiovascular risk factors among first-year brazilian university students in são paulo. Nutr Hosp 2011, 26:553–559.

Dwivedi S, Aggarwal A: Central obesity, hypertension and coronary artery disease: the seed and soil hypothesis. World J Cardiol 2011, 3:40–42.

Roche SL, Silverides CK: Hypertension, obesity, and coronary artery disease in the survivors of congenital heart disease. Can J Cardiol 2013.

Sonneveld KR, Riffas-Shiman SL, Kleinman KP, Gortmaker SL, Gillman MW, Taveras EM: Associations of obesogenic behaviors in mothers and obese children participating in a randomized trial. Obesity (Silver Spring) 2012, 20:1449–1454.

Camargo AP, Barros Filho Ade A, Antonio MA, Giglio JS: the non perception of obesity can be an obstacle to the role of mothers in taking care of their children. Cien Saude Colet 2013, 18:323–333.

Pemberton VL, McCrindle BW, Barkin S, Daniels SR, Barlow SE, Binnis HJ, Cohen MS, Economos C, Faith MS, Gidding SS, Goldberg CS, Kavey RE, Longmuir P, Rocchini AP, Van Horn L, Kaltman JR: Report of the national heart, lung, and blood institute’s working group on obesity and other cardiovascular risk factors in congenital heart disease. Circulation 2010, 121:1153–1159.

Hipple B, Nabi-Burza E, Hall N, Regan S, Winicoff JP: Distance-based training in two community health centers to address tobacco smoke exposure of children. BMC Pediatr 2013, 13:613.

Coelho SA, Rocha SA, Jong LC: Consequências do tabagismo passivo em crianças. Cien Cuid Saude 2012, 11:294–301.

Pandey AKP, Pandey S, Blaha MI, Agaston A, Feldman T, Ozer M, Santos RD, Budoff MJ, Blumenthal RS, Nasis K: Family history of coronary heart disease and markers of subclinical cardiovascular disease: Where do we stand? Atherosclerosis 2013, 228(2):285–294.

Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, Steer C, Sherriff A: Early life risk factors for obesity in childhood: Cohort study. BMJ 2005, 330:1357.

Vickers MH: Developmental programming of the metabolic syndrome - critical windows for intervention. World J Diabetes 2011, 2:137–148.

Heenwagen MJ, Miller MR, Barbour LA, Friedman J: Maternal obesity and fetal metabolic programming: a fertile epigenetic soil. Am J Physiol Regul Integr Comp Physiol 2010, 299:R711–R722.

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