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Heritability of biochemical and anthropometric parameter markers of cardiovascular risk: a study with twins

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Abstract. The present study has the objective of investigating the heritability of anthropometric and biochemical markers as predictors of cardiovascular risk in a sample with children, adolescents, and adults from the Northeast of Brazil, considering the scarcity of studies with this population, using the method of twins. *Material and Method.* The sample was composed of 88 twins, 52 children and adolescents and 36 adults. Stature, body mass, waist circumference, BMI, fasting glycemia, total cholesterol, HDL-C, LDL-C, and triglycerides were analyzed. Heritability analysis of the biochemical and anthropometric indicators was performed by means of structural equation modeling in the Mplus program, version 7.4. This analytical approach typically decomposes the phenotypic variance into three components: genetic (A), common or shared environmental (C), and non-shared environmental (E) - ACE model. *Results.* When testing the ACE model, the presented biochemical and anthropometric parameters did not demonstrate significant inherited components. The best adjustment for all these parameters was CE, indicating that the shared and non-shared components were stronger. *Conclusion.* By identifying the heritability of these parameters, it becomes possible to carry out specific intervention strategy planning. A lifestyle with practice of regular physical exercise and healthy habits may be able to modify the action of inherited components, responsible for the predisposition to obesity and cardiovascular risk. **Key words:** *twins, obesity, genetic inheritance, anthropometry, lipid profile, cardiovascular risk*.

Introduction

The growing number of cardiovascular disorders highlights the crucial need to investigate efforts to prevent and screen cardiovascular risk factors. Studies (1-3) show that a sedentary lifestyle has been associated with the accumulation of body fat, changes in biochemical parameters, and low cardiorespiratory capacity. In contrast, exercise has a positive effect on preventing disease and provides a number of health benefits, such as control of obesity, hypertension, diabetes mellitus, and hypercholesterolemia (3-5). The association between metabolic, nutritional, psychosocial, environmental, and cultural factors influences the development of this condition in genetically predisposed individuals (6).

Associations between genotype and phenotype are important to identify risk related to the development of chronic diseases such as Diabetes Mellitus type II, cardiovascular diseases, and metabolic syndrome (7, 8). Studies about hereditability (9) have shown that genetic and environmental factors contribute to the development of obesity and its associated phenotypes, in addition to which, it may be seen that the phenotypic variation of a trait could be attributed to genetic variation. A healthy lifestyle, with adequate diet and physical activity, positively influences a reduction in the body mass index, elevates HDL-cholesterol levels, lowers blood glucose, LDL-cholesterol, and triglyceride levels and, consequently, cardiovascular risk (10). However, it is necessary to understand the influence of heritability on these components, allowing health professionals a margin of intervention.

Heritability studies seek to observe hereditary and environmental contributions to biochemical, anthropometric, and functional markers as health risk markers (7, 11, 12), however this identification is specific and differentiated for each population, considering that ethnicity, age, and gender may be

determining variables in inherited and shared factors (7, 10). In this sense, the present study aims to investigate the heritability of anthropometric and biochemical markers, as predictors of cardiovascular risk, in a sample with children, adolescents, and adults from the Northeast of Brazil, considering the scarcity of this type of study with this population.

Material and Method

Participants. A total of 88 individuals participated in the study, of which 52 were children and adolescents (08 to 17 years of age), represented by 32 Monozygotic (MZ) (20 females and 12 males) and 20 Dizygotic twins (DZ) (12 females and 08 males), and 36 adults (18 to 28 years old), represented by 24 MZ (08 female and 16 male) and 12 DZ (06 female and 06 male), all volunteers and residents in the metropolitan region of the city of Natal / RN, Brazil (Table 1).

Table 1. Characteristics of participants (median or %) stratified by zygosity

Characteristic	Monozygotic twins (n = 17 pairs; n = 34 individuals)	Dizygotic twins (n = 11 pairs; n = 22 individuals)	p^1
Sex (% female)	58.8	54.5	0.752
Age (y)	14	18	0.256
Fasting glucose	76	71	0.098
Total cholesterol	159	148	0.927
HDL	40	50	0.055
LDL	101	73	0.461
Triglycerides	63.5	76	0.808
BMI	20.9	20.2	0.801
WC	68.3	67.5	0.980

Note: [1]Chi-square test and Mann-Whitney test for categorical and continuous variables, respectively.

Individuals diagnosed with diabetes and cardiovascular disease, physically disabled individuals, pregnant women, individuals on obesity-related medical treatment, or individuals with endogenous or secondary obesity (Down syndrome, Prader Willi syndrome, and hypothyroidism), as well as different sexes, pairs of twins in different pubertal stages, and pairs that did not share the same environment and presented disagreement in the practice of physical activity were excluded from the study.

The study was approved by the Research Ethics Committee of the University Hospital Onofre Lopes - CEP / HUOL, duly recognized by the National Ethics Committee under protocol 484/10, according to Resolution CNS196 / 96, according to the Declaration of Helsinki 1975 and Addendum of 2000.

Procedures. The research was divided into three moments: In the first, the selection of the pairs of twins in an intentional non-probabilistic manner was performed with the signing of the Informed Consent Term (TCLE) by the subjects or caregivers. After signing the TCLE, an anamnesis questionnaire was applied as a method of controlling the intra-pair physical activity of the twins and analyzing the health status of each twin.

The twins were then submitted to anthropometric evaluation. The anatomical landmarks and protocols for the anthropometric evaluation followed the standardization of the International Society for Advancement in Kinanthropometry (ISAK). For the analysis of body mass, a Filizola® 110 electronic scale was used. Stature was obtained by means of a stadiometer, Sanny®. For measurements of circumference, an anthropometric metal measuring tape, Sanny®, was used. The anthropometric indicators used as cardiovascular and obesity risk factors were waist circumference (WC) and Body Mass Index (BMI)(13).

The reference values used for the WC followed the Taylor cut-off points (14), for individuals aged 8 to 17 years and for adults (above 18 years), according to the classification of the World Health Organization (WHO)(15). The classification of BMI followed the WHO classification (17) for adults, and the tables of Cole (12, 16) for individuals aged 8 to 17 years.

Individuals participated in the evaluations wearing light clothes and barefoot, in a quiet room with a temperature between 22-24º C. No twin participated in any type of vigorous activity or consumed alcohol or caffeine for 24 hours prior to the tests. All were informed about the importance of adequate sleep the night before the procedure and were familiar with the research.

The pubertal stage of each pair of twins was evaluated using the Tanner stage self-assessment protocol (17). The determination of zygosity was performed through a zygosity questionnaire, applied to the mothers of the twins, validated by Peeters (18). On the third day, on a non-consecutive day to the physical evaluation, the subjects underwent venous puncture, after an 8 to 12 hour fast, to collect peripheral blood samples without anticoagulant (10 mL). Biochemical examinations were performed at the Integrated Laboratory of Clinical Analyzes, Faculty of Pharmacy, UFRN, following the routine established in this service. The lipid profile (LDL-c, HDL-c, Triglycerides and Total Cholesterol) and fasting glycemia, measured by the colorimetric/enzymatic method, using Labtest® Diagnostica SA kits, were analyzed. LDL-c was obtained by applying the Friedewald formula (19).

Statistical analysis. The descriptive analysis of the data was carried out in the program R 3.3.1 (20). The comparison of the MZ and DZ groups according to the study variables was performed using the chi-square test and the Mann-Whitney test for categorical and continuous data, respectively. Spearman's correlation was used to evaluate the correlation between twins of the same pair in the MZ and DZ groups. This comparison provides a preliminary indication of genetic influence, since a stronger correlation between MZ than between DZ suggests heritability of the trait analyzed.

Heritability analysis of the biochemical and anthropometric indicators was performed by means of structural equation modeling in the Mplus program, version 7.4 (21). This analytical approach typically decomposes the phenotypic variance into three components: genetic (A), common or shared environmental (C) and non-shared environmental (E) - ACE model (22). For the construction of this model, it is assumed that the genetic influence (A) is perfectly correlated between MZ (share 100% of their genes) and, between DZ, this correlation is considered to be 50%, (half of their genes). By definition, the influence of the shared environment (C) is considered perfectly correlated for MZ and DZ (100%) and that of the non-shared environment (E) is assumed to be uncorrelated in both groups (0%). The complete models (ACE) were compared with nested models in which components A, C, or both are considered equal to zero (models AE, CE, and E). Models with a low and non-significant chi-square value, with lower AIC (Akaike's information criterion) and lower RMSEA (Root-Mean-Square Error of Approximation) were considered to be better adjusted and more parsimonious. The estimates of parameters A, C, and E and their 95% confidence intervals (95% CI) were expressed as a percentage of the total phenotypic variance (a^2, c^2 and e^2). The estimation was performed by maximum likelihood and the 95% CI was extracted by the bootstrap method (1000 replications). Values of $p \leq 0.05$ were considered significant. The models were adjusted by potential confounders (gender and age).

Results

The strongest correlation presented among the MZ twins in Table 2, in all variables, with the exception of triglycerides, suggests heritability in the analyzed trait. However, this analysis is exploratory, being only an initial assessment that needs to be confirmed through ACE models.

Table 2. Twin-twin Spearman correlation coefficients (r) for biochemical and anthropometric indicators

Characteristic	**Monozygotic twins** (n = 17 pairs)	**Dizygotic twins** (n = 11 pairs)
Fasting glucose	0.732***	0.705*
Total cholesterol	0.866***	0.820**
HDL	0.891***	0.765**
LDL	0.883***	0.697*
Triglycerides	0.594*	0.900***
BMI	0.926***	0.891***
WC	0.932***	0.888***

Note: *p≤0.05; **p≤0.01; ***p≤0.001

Thus, in testing these more complex models, heritability was not significant. Therefore, the presented biochemical and anthropometric parameters did not demonstrate the significant inherited component (Table 3). The best fit for all these parameters was the CE, indicating that the shared and non-shared components were stronger, that is, they are variables with greater possibility for intervention. Another important factor is that in the ACE models it was possible to adjust for sex and age, controlling for the effect of these variables, thus providing a truer estimate.

Table 3. Estimates of genetic (a^2) and environmental (c^2 and e^2) components (with 95% confidence intervals) for biochemical and anthropometric indicators, with model fit statistics, adjusted for sex and age.

ACE model	Proportions of phenotypic variance explained						Model fit			
	a^2 (%)	*p*	c^2 (%)	*p*	e^2 (%)	*p*	AIC	RMSEA	X^2	*p*
Fasting glucose										
ACE	0.000 (-44.1; 44.1)	0.999	56.6 (16.9; 96.4)	0.005	43.4 (1.2; 85.5)	0.044	346.7	0.00 (0.00; 0.09)	12.12 (20)	0.912
AE	55.2 (8.9; 101.4)	0.019	-		44.8 (-1.4; 91.1)	0.057	347.3	0.00 (0.00; 0.13)	14.63 (21)	0.841
CE	-		**56.6 (19.0; 94.3)**	**0.003**	**43.4 (5.7; 81.0)**	**0.024**	**344.7**	**0.00 (0.00; 0.06)**	**12.12 (21)**	**0.936**
E	-		-		1 (1; 1)	0.999	353.6	0.06 (0.00; 0.23)	22.94 (22)	0.405
Total cholesterol										
ACE	0.5 (-19.5; 20.5)	96.2	85.9 (62.7; 109.1)	0.000	13.6 (1.7; 25.5)	0.025	484.7	0.00 (0.00; 0.06)	11.32 (20)	0.937
AE	86.8 (75.5; 98.1)	0.000	-		13.2 (1.9; 24.5)	0.022	491.5	0.00 (0.00; 0.22)	20.10 (21)	0.515
CE	-		**86.3 (73.8; 98.7)**	**0.000**	**13.7 (1.3; 26.2)**	**0.031**	**482.7**	**0.00 (0.00; 0.00)**	**11.32 (21)**	**0.956**
E	-		-		1 (1; 1)	0.999	518.9	0.30 (0.19; 0.41)	49.50 (22)	0.001
HDL										
ACE	24.8 (-21.6; 71.2)	0.295	56.3 (20.5; 92.0)	0.002	18.9 (-2.3; 40.1)	0.080	383.3	0.00 (0.00; 0.01)	10.88 (20)	0.949
AE	82.0 (60.8; 103.2)	0.000	-		18.0 (-3.2; 39.2)	0.097	383.7	0.00 (0.00; 0.10)	13.30 (21)	0.898
CE	**-**		**75.1 (59.2; 90.9)**	**0.000**	**24.9 (9.1; 40.8)**	**0.002**	**382.2**	**0.00 (0.00; 0.03)**	**11.76 (21)**	**0.946**
E					1 (1; 1)	0.999	402.5	0.20 (0.02; 0.32)	34.06 (22)	0.048
LDL										
ACE	27.6 (-21.0; 76.3)	0.265	61.2 (10.8; 111.6)	0.017	11.2 (1.0; 21.3)	0.031	481.4	0.00 (0.00; 0.08)	11.80 (20)	0.923
AE	88.9 (79.3; 98.6)	0.000	-		11.1 (1.4; 20.7)	0.025	482.8	0.00 (0.00; 0.15)	15.21 (21)	0.812
CE	-		**82.5 (65.6; 99.4)**	**0.000**	**17.5 (0.6; 34.4)**	**0.042**	**481.6**	**0.00 (0.00; 0.12)**	**14.01 (21)**	**0.869**
E	-		-		1 (1; 1)	0.999	509.8	0.27 (0.15; 0.38)	44.17 (22)	0.003
Triglycerides										
ACE	10.2 (-34.0; 54.5)	0.650	69.5 (23.6; 115.3)	0.003	20.3 (6.6; 34.0)	0.004	524.7	0.00 (0.00; 0.00)	7.95 (20)	0.992
AE	80.4 (67.2; 93.6)	0.000	-		19.6 (6.4; 32.8)	0.004	526.3	0.00 (0.00; 0.00)	11.57 (21)	0.950
CE	-		**77.6 (59.6; 95.5)**	**0.000**	**22.4 (4.5; 40.4)**	**0.014**	**522.9**	**0.00 (0.00; 0.00)**	**8.12**	**0.995**
E	-		-		1 (1; 1)	0.999	546.6	0.20 (0.00; 0.32)	33.88 (22)	0.050
BMI										
ACE	12.6 (-22.9; 48.1)	0.486	74.9 (38.7; 111.0)	0.000	12.5 (3.6; 21.5)	0.006	222.6	0.00 (0.00; 0.00)	7.63 (20)	0.994
AE	86.8 (78.3; 95.3)	0.000	-		13.2 (4.7; 21.7)	0.002	224.9	0.00 (0.00; 0.04)	11.88 (21)	0.943
CE	-		**85.2 (74.4; 95.9)**	**0.000**	**14.8 (4.1; 25.6)**	**0.007**	**221.2**	**0.00 (0.00; 0.00)**	**8.19 (21)**	**0.994**
E	-		-		1 (1; 1)	0.999	255.4	0.27 (0.15; 0.38)	44.38 (22)	0.003
WC										
ACE	0.000 (-23.4; 23.4)	0.999	85.4 (60.5; 110.3)	0.000	14.6 (4.4; 24.9)	0.005	317.1	0.00 (0.00; 0.00)	10.32 (20)	0.962
AE	85.3 (75.4; 95.1)	0.000	-		14.7 (4.9 ; 24.6)	0.003	322.5	0.00 (0.00; 0.19)	17.73 (21)	0.666
CE	-		**85.4 (74.2; 96.5)**	**0.000**	**14.6 (3.5; 25.8)**	**0.010**	**315.1**	**0.00 (0.00; 0.00)**	**10.32 (21)**	**0.974**
E	-		-		1 (1; 1)	0.999	349.7	0.28 (0.17; 0.40)	46.88 (22)	0.001

Note: a^2, additive genetic component; c^2, common or shared environmental component; e^2, unique or non-shared environmental component; -2LL, -2 log likelihood; AIC, Akaike's information criterion; CFI, comparative fit index; RMSEA, Root-Mean-Square Error of Approximation; X^2, chi-square test of model fit. Bold values indicate best models.

Discussion

This study presents new evidence on the environmental effects on the biochemical and anthropometric markers analyzed, from the ACE model(22). The variables were significantly correlated with the fact that these markers are modified or influenced by the environment, external, and shared factors.

Fasting glucose demonstrated greater influence of the shared component, indicating that the individual's life habits influence 56.6% of their mobilization. Given this, it is possible to generate positive changes induced by exercise, since regular use of muscles also prevents accumulation of intramuscular fat that may be related to the glucose metabolism (23). In the study of Leskinen, et al. (23), active twins had lower fasting blood glucose levels and Rottensteiner, et al. (24), observed that healthy adult twins, with a higher level of physical activity, presented glucose improvement in situations of homeostasis, regardless of the genetic component. Our finding contributes to confirming the importance of strategies to prevent or reduce the risk of type 2 diabetes (4, 5).

Total Cholesterol, HDL, and LDL behaved as characteristic markers of accumulation of environmental factors, ranging from 73.8% to 98.7% (total cholesterol); 59.2% to 90.9% (HDL); and 65.6% to 99.4% (LDL), demonstrating that these components can be modified independently of the inherited factors and that lifestyle characteristics are contributing factors for their modification. Corroborating our findings, Li, et al (25), found moderate to high contributions of the single environment for cholesterol and LDL phenotypes in Danish twins (approximately 51%), however, Chen, et al, (26), found higher heritability for HDL, LDL, and total cholesterol (from 49% to 62% of heritability influence). These differences found in heritability may be influenced by age, since our study presented a higher age gradient (8 to 28 years), while in the study of Chen, et al (26), the age range was 5 to 18 years. These differences can also be justified due to the method used, sample size, and sex of the sample, however in all these studies there are percentages of environmental influence and this is what the present study refers to, regardless of the inherited factors, these variables can be modulated by external factors.

Triglyceride also showed greater influence of the environment (59.6% to 95.5%), indicating that health behavior is an important factor behind the genetic effects. In this sense, we also want to highlight waist circumference, with influence of 74.2% to 96.5% from external factors. These two markers represent a simple clinical phenotype to identify patients with excess visceral fat and the related characteristics of metabolic syndrome. The level of triglycerides in plasma is a useful marker of visceral adiposity in the presence of a given waist circumference, which justifies this being an extremely important data set in determining cardiovascular risk in our study. This higher environmental influence of triglycerides and waist circumference was also found in other studies such as Li, et al (25) and Chen, et al, (26), who verified high contributions for the unique environment for triglycerides, as well as which, the environment may reduce the genetic variance of waist circumference in early adulthood, suggesting that heritability for abdominal obesity is minimized in physically active people (27).

Although some studies have shown that men of different ages have a higher incidence of visceral abdominal fat, justified by more frequent android obesity (28, 29, 30), our study demonstrated that in the anthropometric markers waist circumference and BMI there was a prevalence of environmental influence, regardless of age or gender. As also observed by Doornweerd, et al (31), where pairs of monozygotic twins discordant in BMI, exhibited exposure to environmental factors responsible for lifestyle factors more compromising to health.

Given this, we can also highlight that there was no difference in the model adjusted for gender and age, indicating that for any stage of life, interventions and incentives to adopt a healthy lifestyle are appropriate. Therefore, it was verified that the variables of the present study can be modulated by the influence of the environment, that is, the practice of physical exercises and an adequate diet can generate changes in these variables, regardless of heritability. In addition, these healthy lifestyle habits will aid in the prevention of metabolic disorders, maintaining glucose homeostasis, and decreasing obesity, risk of type 2 diabetes mellitus, and associated diseases (23). On the other hand, it is also possible that environmental factors may contribute to the aggregation of risk factors that characterize obesity and chronic degenerative diseases.

Conclusion

It was concluded that the anthropometric variables BMI and waist circumference and biochemical parameters (fasting glucose, total cholesterol, HDL, LDL, and triglycerides) suffered low influence from inherited components, with greater influence from the shared environment and the non-shared environment.

Through identification of the heritability of these parameters, related to cardiovascular risk, it becomes possible to carry out specific intervention strategy plans. The action of the environment can become a decisive agent in the severity of the repercussions for the health of the individual. Therefore, a lifestyle with regular physical exercise and healthy habits may be able to modify the action of the inherited components responsible for the predisposition to obesity and cardiovascular risk.

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