Association of parents’ physical activity and weight status with obesity and metabolic risk of their offspring

Associação do status de atividade física e peso dos pais com os indicadores de obesidade e risco metabólico dos filhos

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Abstract  Our aim was to analyze the joint association of parental characteristics and offspring obesity indicators with metabolic risk in adolescents. A cross-sectional study was carried out with 972 adolescents and their parents. We observed that overweight adolescents who have a normal weight mother show lower metabolic risk in comparison with their counterparts with overweight mothers. In conclusion, mother’s weight status moderates the relationship between offspring obesity indicators and metabolic risk in adolescents.

Key words Pediatric obesity, Parents, Metabolic syndrome

Resumo  Nosso objetivo foi analisar a associação combinada entre características dos pais e indicadores de adiposidade dos filhos com o risco metabólico em adolescentes. Foi realizado estudo transversal com 972 adolescentes e seus pais. Observamos que adolescentes com sobrepeso que possuem mãe com peso normal apresentaram menor risco metabólico em comparação com seus pares com mães que apresentam sobrepeso. Concluímos que o status de peso da mãe modera a relação entre indicadores de obesidade e risco metabólico dos adolescentes.

Palavras-chave Obesidade infantil, Pais, Síndrome metabólica
Introduction

Alongside the technological revolution, and the emergence of the epidemiological transition, chronic diseases have become the most common cause of death worldwide. The prevalence of chronic diseases, such as hypertension and type II diabetes, is high in adults and has been increasing in young populations, especially in overweight and obese children and adolescents. Combating early-onset obesity in childhood may be a fruitful way of reducing child morbidity and chronic disease risk in adulthood, but it is currently one of the main challenges of public health.

The determinants of obesity are multifactorial and may include diverse influences from infections, or ambient temperature affecting thermoregulation, to intrauterine and intergenerational factors. There are, however, some recognized pathways for heightened obesity and metabolic risk, including high-energy diets, low physical activity levels, socioeconomic and genetic factors. Parental influences on youth health outcomes are being studied, such as associations between maternal physical activity level during gestation and offspring health in the short-term and associations between parental obesity and long-term cardiovascular risk in children when middle-aged. Thus, it appears plausible that parental characteristics can influence childhood obesity propensity via a combination of behavioral and biological pathways.

In the British Birth Cohort Study, Cooper et al. found a positive association between parental obesity with cardiovascular risk factors in their adult offspring. Moreover, Khanolkar et al. found relationships between parental behavioral and biological risk factors with children’s metabolic risk using a Swedish sample. Nonetheless, to the best of our knowledge, no study has investigated the relationship between parental biology and behavior with offspring obesity and metabolic risk in a low to middle income country.

Countries with a different economic and social structure, such as Brazil, for example, which is marked by deep social inequalities, which may be factors that limiting the access to physical exercise and healthier living habits, especially for people with low income. Therefore, given that broader social determinants could explain health behaviors and metabolic risk in low to middle income countries, it is suppose that the relationship between parental biology/behavior and obesity and metabolic risk of their offspring could be different from high-developed countries. Thus, our aim was to analyze the joint association of parental health characteristics (weight status and physical activity) and offspring obesity indicators with metabolic risk in adolescents from Southern Brazil.

Methods

Sample

This was a cross-sectional study conducted in adolescents aged between 10 and 16 years-old who were enrolled in a project from public schools at Londrina/PR, Brazil in 2011, named “Prevalence of metabolic syndrome and cardiovascular risk factors in adolescents from Londrina”. Recruitment to the study was performed in two stages. First, all public schools in the city were separated into regions (north, south, east, west and center) and two schools were randomly selected from each region to participate in the study. Second, classes within schools were randomly selected, and all students within those classes received an invitation to the study. Students who were concurrently using prescription medicine, who were undergoing treatment for an illness, or who failed to return a signed parent consent form were all excluded. In total, 1,395 adolescents were successfully recruited, but due to incomplete data especially from parental variables, total sample ranged between 719 (paternal data) and 972 adolescents. Additional information about sampling process is previously published. The local ethics committee approved all of the study’s procedures, which adhered to the principles of the Declaration of Helsinki.

Metabolic risk

Blood pressure was measured on the right arm after a rest period of ten minutes using automatic apparatus (OMRON – HEM-742) that has been validated in adolescents. Two measurements interspersed with a rest interval of two minutes were made and the arithmetic mean of values was calculated. If the difference between measurements was greater than 10 mmHg for systolic (SBP) and diastolic blood pressure (DBP) then a third measurement was performed. In this case, the two closest values were used to inform the mean.

Laboratory tests to estimate fasting glucose, triglycerides and high-density lipoprotein cholesterol (HDL-C) were conducted by trained as-
sensors of the biochemistry laboratory from the university hospital of Londrina State University. In the school, blood samples were collected after a 12 hour fast from the antecubital vein, either in serum tubes (no anticoagulant) or for the determination of glucose a tube containing an anticoagulant fluoride preservative. Tubes were centrifuged at 3,000 rpm for 5 minutes at 4ºC to separate plasma and serum. Analyzes were performed immediately following separation of materials using a biochemical auto analyzer (Dimension RxL Max – Siemens Dade-Behring).

A continuous metabolic risk score was derived by summing z-scores for triglycerides, fasting glucose, HDL-C (with opposite sign) and the arithmetic mean of systolic and diastolic blood pressures ((calculation: triglycerides z + fasting glucose z + [-1*HDL-C z] + [{SBP z + DBP z}/2]) /4)12. We did not include waist circumference in the score, because of the relation with body mass index, as we previously showed13.

Adolescents’ obesity indicators

Central adiposity was ascertained by measurement of waist circumference between the lowest rib and the upper edge of the iliac crest to the nearest 0.1cm and classified according Katzmarzyk et al.14. Three measurements were collected for each participant and the median was used. Subscapular and triceps skinfold thicknesses were measured, according to the Boileau15 recommendations, by a trained evaluator using a calibrated (to a precision of 0.5 mm) Lange caliper. The technical error of measurement (TEM) was 4.8% and 3.5% for subscapular and triceps skinfolds, respectively, and from measurements the percentage of body fat was estimated and classified according Williams et al.16. Body mass index (BMI, kg/m²) was calculated from measured weight and height, both of which exhibited TEMs10 and classified according Onis et al.17.

Parents’ variables

Maternal and paternal BMI were calculated using self-reported values for weight and height in response to questionnaires that were answered by each parent. Previous studies have reported good validity of self-reported weight and height in Brazilian adults18. Parents were categorized as overweight if BMI was 25 kg/m² or more19. Physical activity levels were self-reported using the Baecke questionnaire, which captures information regarding about exercise practice and time of practice (Baecke et al., 1988) as well as physical activity in transportation, leisure and occupational domains, through Likert scales. Parents that reported themselves to be physically active for more than 120 min/week (moderate to vigorous physical activity at least for four months) were classified as active. The questionnaires were delivered to the parents through the adolescents.

Covariates

Somatic maturation was used as biological maturity indicator. Specifically, age at peak height velocity (PHV) was estimated by combining anthropometric measures with a published, commonly implemented, prediction equation20. We have previously described the specific details of this method21. Socioeconomic status (SES) was assessed by means of the Brazilian Criterion for Economic Classification instrument22, which provides a score based on the education of the household leader and ownership of certain possessions. We classified participants from classes C to E as with low socioeconomic status.

Statistical analysis

Descriptive statistics are presented with median, interquartile range and frequencies. Kolmogorov-Smirnov test was used to verify data distribution. Once some variables did not present a normal distribution, we used Mann-Whitney’s test and chi-square to compare boys against girls. The Spearman’s rank-order correlation was used to verify the relationship of covariates, adolescents’ adiposity indicators and parents’ variables with the adolescents’ metabolic risk score. The biserial correlation coefficient was used to obtain the correlation between the nominal variables and the metabolic risk score.

Subsequently, we calculated the estimated marginal means of metabolic risk score and tested if associations between adolescent adiposity (BMI, waist circumference and body fat; each modelled separately) and metabolic risk were modified by parental weight or physical activity status using Generalized Estimating Equations (GEE). GEE has been proposed for non-normally distributed data with advantages to fit models and to generate population-level parameters. Further details can be found elsewhere23. Evidence for effect moderation was considered statistically significant when the \( p \)-interaction term was < 0.0524, adjusted for sex, chronological age, socioeconomic status, and age of PHV. All analy-
ses were conducted using the software Stata 15.1 (StataCorp. College Station, TX, USA).

Results

Descriptive statistics for the sample are presented in Table 1. Boys were older and had a later estimated age at PHV than girls. With regards to metabolic risk factor characteristics, boys had a larger waist circumference, higher SBP, higher fasting glucose levels, and lower HDL-C than girls. In opposite, girls had higher total percentage body fat, marginally higher DBP, and higher triglyceride concentrations. There were no sex differences in paternal BMI or the proportion of parents classified as “active”.

Table 2 shows correlations of covariates, adolescents and parents' level variables with metabolic risk score. We observed significant (p < 0.05) and directly (+) correlations of chronological age, all three adolescents’ adiposity indicators and maternal BMI and metabolic risk score. Age of the peak height velocity was inversely correlated to the outcome.

Estimated marginal means of metabolic risk score for joint groups of adolescent obesity indicators and parental characteristics (physical activity and weight status) as well as the interaction

Table 2. The Spearman's rank-order correlation between adolescents and parents' levels variables with metabolic risk score (Londrina/PR, 2011).

| Covariates                          | Rho     | p-value |
|-------------------------------------|---------|---------|
| Sex (male/female)                   | 0.003   | 0.936   |
| Chronological age, years            | 0.095   | 0.003   |
| Age of peak height velocity, years  | -0.066  | 0.039   |
| Socio-economic status (classes)     | 0.057   | 0.100   |
| Adolescents' level BMI, kg/m²       | 0.188   | < 0.001 |
| Waist circumference, cm             | 0.175   | < 0.001 |
| Body fat, %                         | 0.137   | < 0.001 |
| Parents' level                      |         |         |
| Maternal BMI, kg/m²                 | 0.109   | 0.003   |
| Paternal BMI, kg/m²                 | 0.060   | 0.137   |
| Maternal physical activity (active/inactive) | 0.003   | 0.936   |
| Paternal physical activity (active/inactive) | 0.011   | 0.769   |

Note: BMI, body mass index; Parents was classified as active when reported more than 120 min/week of moderate to vigorous physical activity (at least for four months). The biserial correlation coefficient was used to obtain the correlation between the nominal variables and the metabolic risk score.

Source: Authors.

Table 1. General characteristics of the sample according to sex (Londrina/PR, 2011).

|                        | Boys (n = 408) | Girls (n = 564) | P    |
|------------------------|---------------|-----------------|------|
| Chronological age, years | Median (IQR)  | Median (IQR)    |      |
| Age of peak height velocity, years | 13.0 (2.2)    | 12.6 (2.2)      | 0.011|
| BMI, kg/m²              | 19.0 (4.5)    | 19.2 (4.8)      | 0.280|
| Waist circumference, cm | 66.5 (10.3)   | 64.3 (9.1)      | < 0.001|
| Body fat, %             | 16.9 (10.2)   | 25.3 (6.2)      | < 0.001|
| Systolic blood pressure, mmHg | 111 (16)    | 110 (13)        | 0.011|
| Diastolic blood pressure, mmHg | 62 (12)    | 64 (10)         | < 0.001|
| Fasting glucose, mg/dl  | 90.0 (8.0)    | 89.0 (8.0)      | < 0.001|
| HDL-C, mg/dl            | 50.0 (16.0)   | 50.0 (15.0)     | 0.192|
| Triglycerides, mg/dl    | 52.5 (33.0)   | 60.0 (37.0)     | < 0.001|
| Maternal BMI, kg/m²     | 25.5 (6.6)    | 25.7 (6.3)      | 0.586|
| Paternal BMI, kg/m²     | 26.0 (5.2)    | 25.9 (4.9)      | 0.506|
| Maternal physical activity, % active | 22.8        | 22.0            | 0.718|
| Paternal physical activity, % active | 22.3        | 23.2            | 0.560|
| Low socio-economic status, % | 65.5    | 68.3            | 0.332|

Note: IQR, interquartile range; BMI, body mass index; parents was classified as active when reported more than 120 min/week of moderate to vigorous physical activity (at least for four months).

Source: Authors.
terms are presented in Table 3. We observed significant interactions \( (p < 0.05) \) only of maternal BMI with adolescent BMI, waist circumference and body fat in the prediction of metabolic risk score. Values of metabolic risk score were significantly higher among adolescents with high adiposity (in all three indicators) who had an overweight mother compared to those who had normal weight mothers. However, adolescents with elevated adiposity (all three indicators) who had normal weight mother showed similar metabolic risk score compared to those with normal weight.

Discussion

Our aim was to analyze the joint association of parental characteristics (weight status and physical activity) and offspring obesity indicators with

Table 3. Estimated marginal means (EMM) of metabolic risk score for joint groups of adolescent obesity indicators and parental characteristics (physical activity and weight status) as well as the interaction terms (Londrina/PR, 2011).

| Indicator          | EMM    | CI 95%                | Interactions      | Wald   | p      |
|--------------------|--------|-----------------------|-------------------|--------|--------|
| **Body mass index**|        |                       |                   |        |        |
| Normal + active mother | -0.050 | -0.141 to 0.042       | BMI vs maternal PA | 1.465  | 0.226  |
| Normal + inactive mother | -0.107 | -0.153 to -0.062      |                   |        |        |
| Overweight + active mother | 0.204  | 0.045 to 0.363        |                   |        |        |
| Overweight + inactive mother | 0.275  | 0.178 to 0.371        |                   |        |        |
| Normal + active father  | -0.457 | -0.154 to 0.063       | BMI vs paternal PA | 0.669  | 0.414  |
| Normal + inactive father  | -0.088 | -0.138 to -0.039      |                   |        |        |
| Overweight + active father  | 0.216  | 0.028 to 0.0403       |                   |        |        |
| Overweight + inactive father  | 0.273  | 0.172 to 0.374        |                   |        |        |
| Normal + normal weight mother | -0.102 | -0.162 to -0.042      | BMI vs maternal BMI | 7.291  | 0.007  |
| Normal + overweight mother  | -0.086 | -0.144 to -0.027      |                   |        |        |
| Overweight + normal weight mother | 0.073  | -0.064 to 0.210       |                   |        |        |
| Overweight + overweight mother | 0.347  | 0.244 to 0.450        |                   |        |        |
| Normal + normal weight father  | -0.030 | -0.099 to 0.039       | BMI vs paternal BMI | 0.279  | 0.597  |
| Normal + overweight father  | -0.095 | -0.158 to -0.033      |                   |        |        |
| Overweight + normal weight father | 0.269  | 0.099 to 0.439        |                   |        |        |
| Overweight + overweight father | 0.263  | 0.152 to 0.374        |                   |        |        |
| **Waist circumference**    |        |                       |                   |        |        |
| Normal + active mother  | -0.043 | -0.132 to 0.046       | WC vs maternal PA | 0.809  | 0.369  |
| Normal + inactive mother  | -0.098 | -0.143 to -0.053      |                   |        |        |
| High + active mother  | 0.250  | 0.073 to 0.426        |                   |        |        |
| High + inactive mother  | 0.300  | 0.193 to 0.403        |                   |        |        |
| Normal + active father  | -0.054 | -0.156 to 0.048       | WC vs paternal PA | 0.183  | 0.669  |
| Normal + inactive father  | -0.073 | -0.121 to -0.025      |                   |        |        |
| High + active father  | 0.347  | 0.132 to 0.562        |                   |        |        |
| High + inactive father  | 0.271  | 0.162 to 0.379        |                   |        |        |
| Normal + normal weight mother  | -0.098 | -0.157 to -0.039      | WC vs paternal BMI | 7.340  | 0.007  |
| Normal + overweight mother  | -0.068 | -0.125 to -0.011      |                   |        |        |
| High + normal weight mother  | 0.073  | -0.071 to 0.216       |                   |        |        |
| High + overweight mother  | 0.375  | 0.260 to 0.491        |                   |        |        |
| Normal + normal weight father  | -0.029 | -0.096 to 0.038       | WC vs paternal BMI | 0.026  | 0.871  |
| Normal + overweight father  | -0.073 | -0.134 to -0.012      |                   |        |        |
| High + normal weight mother  | 0.315  | 0.134 to 0.497        |                   |        |        |
| High + overweight father  | 0.252  | 0.130 to 0.374        |                   |        |        |
metabolic risk in adolescent offspring. We identified that the relationship between adolescent obesity indicators and metabolic risk is stronger compared to the parental indicator, however maternal BMI moderates this association between adolescent adiposity and metabolic risk. In other words, among overweight/high adiposity adolescents, those who have an overweight mother tend to have worst metabolic profile compared with their counterparts. For other way, adolescents with elevated adiposity indicator who have normal weight mothers showed similar metabolic risk score than normal weight adolescents.

The negative impacts of obesity during childhood and adolescence are largely studied. Childhood obesity seems to be associated with several outcomes, since psychological, as a lower social acceptance and higher risk of depression\(^25\), up to cardiovascular risk factors\(^5\). Thus, a better comprehension of correlates of childhood obesity could culminate in the elaboration of more robust strategies of interventions for this outcome.

Among several determinants of obesity in childhood, genetic and environmental factors have been extensively mentioned. In fact, studies have shown that parents’ obesity seems to influence obesity indicators in adolescents through different paths\(^26\). The environmental path can be due to the transference of lifestyle behaviors of parents to their children or even by the importance given for the parents of a healthy lifestyle\(^27\). Parents can influence behaviors of their children, as feed behavior, physical activity and sedentary behaviors, which contributes with obesity indicators of adolescents\(^27,28\). Our findings did not show correlation between parental physical activity and adolescents’ metabolic risk score, suggesting other potential pathways.

We found that overweight adolescents had higher metabolic risk score, however, those who had a normal weight mother, even being overweight, had lower metabolic risk in comparison of their counterparts (overweight) with overweight mothers. There may be several ways to understand how mothers’ weight status changed the relation between obesity indicators and metabolic risk in adolescents. One possible approach would be an indirect way (as well as for obesi-

### Table 3

Estimated marginal means (EMM) of metabolic risk score for joint groups of adolescent obesity indicators and parental characteristics (physical activity and weight status) as well as the interaction terms (Londrina/PR, 2011).

|          | EMM  | CI 95%          | Interactions | Wald | p   |
|----------|------|-----------------|--------------|------|-----|
|          |      |                 |              |      |     |
| % fat    |      |                 |              |      |     |
| Normal + active mother | -0.041 | -0.131 to 0.048 | % fat vs maternal PA | 0.209 | 0.647 |
| Normal + inactive mother | -0.086 | -0.132 to -0.041 |              |      |     |
| High + active mother | 0.228 | 0.061 to 0.396 |              |      |     |
| High + inactive mother | 0.234 | 0.132 to 0.336 |              |      |     |
| Normal + active father | -0.052 | -0.157 to 0.052 | % fat vs paternal PA |      |     |
| Normal + inactive father | -0.063 | -0.111 to -0.015 |              | 0.083 | 0.773 |
| High + active father | 0.288 | 0.087 to 0.489 |              |      |     |
| High + inactive father | 0.240 | 0.132 to 0.348 |              |      |     |
| Normal + normal weight mother | -0.093 | -0.153 to -0.032 | % fat vs maternal BMI | 8.611 | 0.003 |
| Normal + overweight mother | -0.064 | -0.120 to -0.007 |              |      |     |
| High + normal weight mother | 0.033 | -0.096 to 0.163 |              |      |     |
| High + overweight mother | 0.346 | 0.233 to 0.460 |              |      |     |
| Normal + normal weight father | -0.028 | -0.098 to 0.042 | % fat vs paternal BMI | 0.007 | 0.935 |
| Normal + overweight father | -0.064 | -0.124 to -0.003 |              |      |     |
| High + normal weight father | 0.275 | 0.113 to 0.437 |              |      |     |
| High + overweight father | 0.230 | 0.101 to 0.359 |              |      |     |

Note: Adjusted by sex, chronological age, socioeconomic status and age of peak height velocity. CI: confidence interval; PA: physical activity. BMI: body mass index; PA: physical activity; WC: waist circumference. Metabolic risk score (adolescent) as outcome. EMMs represent predicted means of metabolic risk score considering all variables included in the models.

Source: Authors.
ty), through a behavioral influence, in this case, adolescents tend to adopt parent’s behaviors and consequently, develop biological risk factors as obesity and higher metabolic risk\textsuperscript{28}. Other path of interference of factors related with parents, is direct, through genetic determination\textsuperscript{29}. In this case, several cardiovascular risk factors are influenced by genetic load of parents\textsuperscript{29-31}. Moreover, an interesting finding was that mothers, but not fathers, moderated the relationship between obesity indicators and metabolic risk in adolescents. This fact can occur due to the local culture\textsuperscript{32}. In our case, probably the most part of fathers works outside home and, consequently, mothers had more contact with their children\textsuperscript{33}. However, it is important to highlight that due to the cross-sectional design adopted in our study, we cannot determine causality between parental and adolescent characteristics.

To the best of our knowledge, this is the first study investigating the relationship between parent’s health characteristics and adolescent’s metabolic outcomes in the context of low to middle income countries. Socioeconomic status is one of the strongest predictors of obesity. The lack of knowledge about healthy lifestyles, such as the amount and types of food to be consumed or the importance of active behaviors, can be associated to this. Families of lower socioeconomic status tend to present poorer eating and active habits\textsuperscript{34}. Also, products such as fruits and vegetables can be expensive in the budget of the poorest families\textsuperscript{35}, and the availability of fruits and vegetables is also lower in these countries. In this way, increasing the ease of acquiring fruits and vegetables can be a way to increase their consumption, and access to the knowledge of a healthy diet and the practice of physical activity through public policies.

Other practical implications can be inferred from the present findings. Firstly, given the association between maternal weight status and adolescent metabolic risk, family approach could be an effective target of interventions. Engagement of parents on childhood obesity interventions for prevention and/or treatment seems to have greater effect on the adolescents’ body fat\textsuperscript{36,37}. Moreover, family interventions should be focused in the parent that pass the greater time with their offspring, given our results concerning the greatest association between mother’s characteristics and offspring than father’s characteristics and offspring. Thus, the best way to deliver these interventions should be investigated.

Study limitations include that parental stature, body mass, and physical activity were all self-reported. Reassuringly, good agreement between self-reported and directly measured stature and body mass has previously been reported in Brazilian adults\textsuperscript{18}. Objective physical activity measurement is growing in Brazil, but subjective indicators are still more common and we accept that they can introduce bias\textsuperscript{38}. In addition, given the instability of weight status during the lifespan, no information about “trajectories” of parents’ weight status was obtained. Nonetheless, strengths of this investigation include availability of data in more than 900 Brazilian adolescents, which permitted metabolic risk calculation, and the analyses were adjusted for important confounding factors including chronological age, socioeconomic status and biological maturation.

**Conclusion**

Adolescents with elevated waist circumference, weight status and sum of skinfolds have higher metabolic risk. However, among them, those who have a normal weight mother show lower metabolic risk in comparison with their counterparts with overweight mothers. Future studies can analyze these relationships longitudinally (causal relationships), as well as unlock the possible mechanisms of interaction between the factors.
Collaborations

AO Werneck: conceptualization, formal analysis, validation, writing – original draft, visualization.
DRP Silva: conceptualization, validation, writing original draft, investigation, visualization.
ECM Silva: validation, writing – original draft, visualization.
P Collins and D Ohara: validation, visualization, writing – review and editing.
RA Fernandes and DS Barbosa: validation, visualization, writing – review and editing.
ERV Ronque and ES Cyrino: validation, visualization, writing – review and editing.

Acknowledgements

The authors thank Alessandra Okino, Jair Oliveira and Danielle Venturini for research support.
Crisieli Tomeleri, Mariana Carmelossi and Sandra Kawaguti for acquisition of data.
Coordenação de Aperfeiçoamento de Pessoal de Nivel Superior (CAPES/BRAZIL) for scholarships (DRP Silva) and Conselho Nacional de Desenvolvimento Cientifico e Tecnologico (CNPq/BRAZIL) for funding the project (483867/2009-8) and for scholarship of scientific initiation (AO Werneck) and productive research (ERV Ronque, ES Cyrino and RA Fernandes).

Funding

Conselho Nacional de Desenvolvimento Cientifico e Tecnologico – CNPq/BRAZIL (483867/2009-8).

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