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Lifetime overweight and adult asthma: 1978/1979 Ribeirão Preto Birth Cohort, São Paulo, Brazil

Sobrepeso ao longo da vida e asma na idade adulta: Coorte de Nascimento de 1978/1979, Ribeirão Preto, São Paulo, Brasil

Sobrepeso a lo largo de la vida y asma en adultos: Cohorte de Nacimientos de 1978/1979 en Ribeirão Preto, São Paulo, Brasil

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Abstract

Studies focusing on obesity and asthma frequently consider the weight at a given time; thus, modeling pathways through lifetime overweight may contribute to elucidate temporal aspects in this relationship. This study modeled the pathways in the association of lifetime overweight with asthma in adult life, using data from the 1978/1979 Birth Cohort, Ribeirão Preto, São Paulo, Brazil (n = 2,063) at birth (baseline), school age (9/11 years) and adult age (23/25 years). A theoretical model was proposed to explore the effects of lifetime overweight on asthma in adult life analyzed by structural equation modeling. Parental obesity (SC – standardized coefficent$_{total}$ = 0.211, p < 0.001; SC$_{direct}$ = 0.115, p = 0.007) and overweight at school age (SC$_{total}$ = 0.565, p < 0.0001; SC$_{direct}$ = 0.565, p < 0.0001) were associated with overweight in adult life. Parental obesity (SC$_{direct}$ = 0.105, p = 0.047) and nutritional status at birth (SC$_{total}$ = -0.124, p = 0.009; SC$_{direct}$ = -0.131, p = 0.007) were associated with asthma in adult life. A higher "current adult socieconomic situation" was inversely associated to overweight (SC$_{direct}$ = -0.171, p = 0.020) and to asthma in adult life (SC$_{total}$ = -0.179, p = 0.041; SC$_{direct}$ = -0.182, p = 0.039). Parental obesity showed a transgenerational effect in weight, triggering to childhood and adulthood overweight. Parallel to underweight at birth, parental obesity was also a risk to asthma in adult life. While, the socioeconomic status in adult life protected from both, overweight and asthma.

Overweight; Life Cycle Stages; Structural Equation Modeling; Asthma

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Introduction

Asthma is a chronic disease that affects 235 million people all over the world [1]; and that is being consistently associated with other non-communicable chronic diseases (NCCD) [2] with which it appears to share physiopathological mechanisms that need to be better explored [3,4].

Obesity is an important risk factor for NCCD, which is being associated with asthma from birth to adulthood [5,6,7,8,9]. Overweight during childhood increases the risk to develop asthma during this phase of life [5,6,7,10,11] or in the future [8,12]. Overweight and obesity have also been associated with asthma in adults, with a dose-response effect on body mass index (BMI) [9].

Studies focusing on obesity and asthma frequently consider overweight at a given time [13,14,15,16]. Few prospective studies on the association between obesity and asthma have adjusted the model for birth variables using regression models [17,18].

The association between obesity and asthma involves a complex and temporal relationship between maternal prenatal weight, birth weight, childhood and even adulthood weight [19,20]. Structural equation modeling (SEM) is a useful tool for the study of epidemiological multicausality phenomena [21].

Modeling the structure between lifetime overweight and asthma may contribute to a better understanding of the temporal aspects and direct and indirect pathways present in this relationship. Thus, this study modeled the pathways in the association of lifetime overweight and asthma in adults in the 1978/1979 Ribeirão Preto Birth Cohort, São Paulo, Brazil, using SEM.

Method

Study design

This was a prospective cohort study entitled *From Perinatal Health to Young Adult Health: a Study of the 1978/1979 Birth Cohort conducted in the Hospitals of Ribeirão Preto, São Paulo*.

Ribeirão Preto is a city located 320km northeast from the state capital of São Paulo, in the Southeast region of Brazil, a wealthy industrialized region with a municipal human development index (M-HDI) of 0.626 in 1991 and 0.733 in 2000, occupying the sixth place in the ranking of the 645 municipalities in the State of São Paulo and 22nd place in the national ranking of the 5,561 municipalities of the country [22,23].

Participants and sample

The baseline information (first follow-up) for the 1978/1979 Ribeirão Preto cohort was obtained with 9,067 interviews held with mothers immediately after delivery, corresponding to 98% of the live newborns delivered at the eight maternities from Ribeirão Preto, from June 1, 1978 to May 31, 1979. The proportion of mothers who were discharged from the hospital before they could be interviewed was 2.5%, and less than 1% refused the interview. The newborns whose mothers did not reside in Ribeirão Preto (N = 2,094) on the date of delivery were excluded [24].

Thus, 6,973 newborns remained in the study, 6,827 of them being singletons and 146 being twins [24]. A total of 257 of the 6,827 singletons died during the first year of life [25] and 86 died by 20 years of age. Thus, 6,484 were left at 20 years of age [26].

During the second follow-up of this cohort in 1987/1989, 2,861 children (43.5% of the original cohort) were evaluated at 9-11 years of age, in private, state and municipal public schools [24].

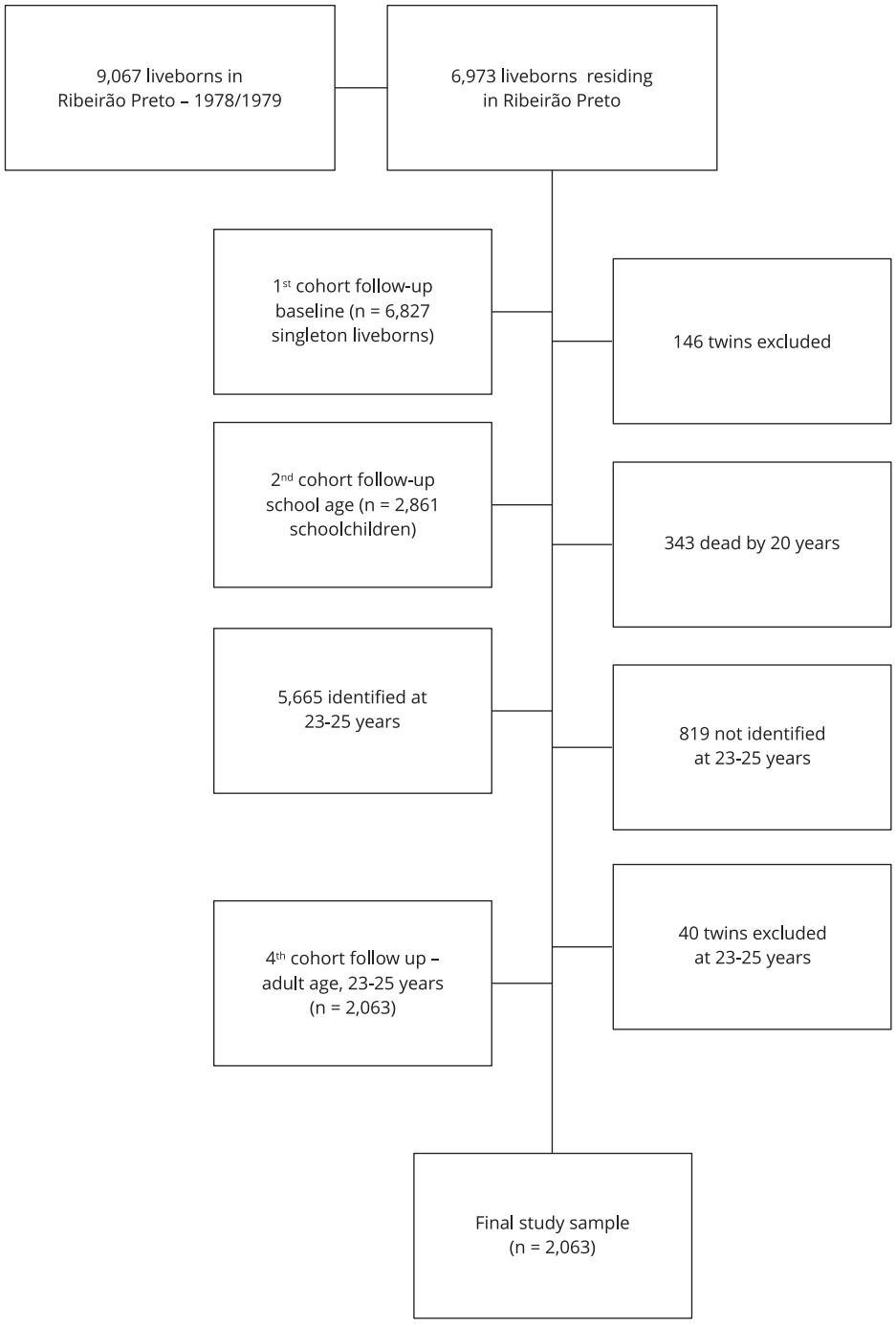
The third follow-up occurred in 1996/1997 and involved only males assessed at the time of recruitment for compulsory military service (n = 2,048) [24].

In the fourth follow-up, 2002-2004, one third of the cohort was sought, and 5,665 individuals among who were alive at 20 years of age were located. In this phase, 2,063 provided data for the questionnaires and for laboratory tests (Figure 1), corresponding to 31.8% of the original cohort [24].

Data for the participants were used for analysis of lifetime overweight and its association with adult asthma in the three follow-up periods of the cohort: baseline, 2nd follow-up (school age, 9-11

Figure 1

Flow diagram of the 1978/1979 Ribeirão Preto Birth Cohort, São Paulo, Brazil, 1978-2002/2004.



years) and 4th follow-up (adult age, 23-25 years). The 3rd follow-up was not used in the present study since it included only 18-year-old males.

Thus, a total participants at the 4th follow-up of the cohort were included. This sample size permitted us to estimate, for the main outcomes of interest, prevalence values within the 50% range with 1.8% relative precision, and prevalence values of about 10% with 1.1% relative precision, with a 95% confidence interval (95%CI). Methodological details regarding this cohort are available in previous studies [24,27].

Data collection

Data collection for the cohort included interviews, the application of structured questionnaires, anthropometric exams, examination of medical records, and the bronchial hyperresponsiveness test.

At birth (baseline), the following information was used: maternal schooling (years of study), maternal occupation, family income (in minimum wages), newborn sex (male and female), and birth weight (grams) and length (cm).

Birth weight and length were measured by trained personnel using appropriate instruments. The newborns were weighed without clothes on a baby weighing scale with 10g precision calibrated weekly. Length at birth was measured with the newborn lying in the supine position on a neonatometer [28] and the data were checked in order to obtain the newborns' weight per length score (z-score).

At school age, information was obtained about the students' current weight (kg) and height (meters) measured according to the recommended techniques [29] and obtained by duly trained personnel in order to determine the growth scores (z-score) of the BMI for age. At the time of weight measurement, students wore a school uniform and were instructed to remove shoes, socks, and to empty their pockets [29]. To measure the weight, the scales used in the schools were used when they were in use; the weight measurement was performed to the nearest 100g. When the school did not have a scale or it was defective, a scale was used by the Department of Child Care and Pediatrics, Faculty of Medicine of Ribeirão Preto, University of São Paulo specially assigned for this purpose [30].

To measure the students' height, we used anthropometers manufactured exclusively for the research, whose measurements were taken in centimeters, to the nearest 0.5cm [30]. The students were instructed to remove all props from their hair, shoes, and socks, and were placed in an upright, anatomical position, arms freely loosened along the trunk, looking straight into the horizon [29].

At adult age, the following information was obtained: adult's age (years), schooling (years of study), occupation, family income (minimum wages), current weight (kg), height (meters) and report of maternal and paternal obesity – history of parental obesity, by the participant.

Adult weight and height were measured by trained personnel according to standard techniques, with the subject barefoot and wearing light clothing [31]. Anthropometric measurements were obtained with a periodically calibrated Filizola precision scale and with an anthropometer standing against the wall for measurements in the orthostatic position [32].

Information about medical diagnosis of asthma, wheezing during the last 12 months, sensation of tightness in the chest and shortness of breath during the last 12 months was obtained by applying the *Respiratory Health Survey* [27] questionnaire containing questions on these symptoms and diagnosis.

Bronchial hyperresponsiveness was also measured by the methacholine bronchoprovocation test. The test was applied using methacholine chloride (Sigma, St. Louis, USA) diluted in phosphate buffered saline according to international criteria [33]. The procedure was carried out by a trained and experienced technician, a physiotherapist and a doctor at the laboratory of Pneumology Service of the University Hospital, Faculty of Medicine of Ribeirão Preto, University of São Paulo [27].

Data processing and statistical analysis

Due to the occurrence of losses on follow-up, the baseline and school phase variables were compared between the adults that attended or did not attend the 4th cohort follow-up, using chi-square test. It was observed that participants born to unskilled manual working mothers ($p = 0.001$) with 0 to 4 years of schooling ($p < 0.0001$), whose families had a monthly income of 0 to 1.9 minimum wages ($p < 0.0001$) attended less the 4th follow-up period of the study. On this basis, the sample was weighted

by calculating the probability of participants attending the 4th cohort follow-up as a function of the birth variables significantly associated in the chi-square test using a logistic regression model. The inverse of this probability of selection was then calculated and this variable was used to weigh the SEM estimates using the Stata software, version 14.0 (https://www.stata.com).

Latent variable

Latent variables result from the combination of various observed variables (indicator variable). The use of latent variables permits a better statistical estimate by representing the theoretical concepts in a more appropriate manner. Furthermore, in a latent variable only common variance shared by different indicators persists, resulting in the estimated effects free from the bias originated by measurement errors [34].

The indicators for the all latent variables were selected based on the convergent loadings (> 0.50) that formed this construct in exploratory factorial analyses (EFA). Model fit was assessed based on the following fit indices: (a) p-value < 0.05 in the chi-square test (χ^2); (b) p > 0.05 and upper 90% confidence interval limit < 0.08 for the Root Mean Square Error of Approximation (RMSEA); (c) Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) values > 0.95; and (d) Weighted Least Square Mean and Variance Adjusted (WLSMV) value < 1 [34].

Then, confirmatory factorial analysis (CFA) was carried out in order to verify the factors previously determined by EFA [34], adopting the same criteria used in the EFA, with the inclusion of discriminant validity, i.e., the correlations between the indicators should not be excessively high (> 0.90), since each indicator should measure a distinct aspect of the latent variable [34]. The modindices (modification index) command was used to obtain suggestions of changes in the proposed latent variable [21]. Exploratory and confirmatory factorial analyses were carried out using the Mplus software, version 7.0 (https://www.statmodel.com/).

• Latent variable: prenatal (baseline) socioeconomic situation

The latent variable "prenatal socioeconomic situation" (prenatal SES) consisted of the following variables: (a) maternal schooling (1 = 0 to 4 years; 2 = 5 to 8 years; 3 = 9 to 11 years, and 4 = 12 or more years of study); (b) maternal occupation (1 = unskilled manual worker; 2 = semi-skilled and skilled manual worker; 3 = non-manual worker, according to the International Standard Classification of Occupation (ISCO) [35]; (c) monthly family income based on the Brazilian national minimum wage in force during the 1978/1979 period, categorized as: 1 = 0 to 1.9 minimum wages; 2 = 2 to 2.9 minimum wages; 3 = 3 to 4.9 minimum wages; 4 = 5 or more minimum wages.

• Latent variable: current socioeconomic situation of the adult (4th cohort follow-up)

The latent variable socioeconomic situation of the adult (current adult SES) consisted of the following variables: (a) adult's schooling (1 = 0 to 4 years; 2 = 5 to 8 years; 3 = 9 to 11 years and 4 = 12 or more years of study); (b) adult's occupation (1 = unskilled manual worker; 2 = semi-skilled manual worker; 3 = skilled manual worker; and 4 = non-manual worker) [35]; (c) monthly family income based on the Brazilian national minimum wage in force during the 2002/2004 period, categorized as: 1 = less than 1 minimum wage; 2 = 1 to 2.9 minimum wages; 3 = 3 to 4.9 minimum wages; 4 = 5 to 9.9 minimum wages; and 5 = 10 or more minimum wages.

Observed variables indicating lifetime obesity

The following observed variables represent lifetime overweight: (a) 1st follow-up (baseline): weight per length z-score (categorized as: 0 = underweight, z-score < -2; 1 = normal weight, z-score ≥ -2 and ≤ +1, and 2 = overweight, z-score > +1) [36]; (b) 2nd follow-up (school age): BMI for schoolchild at age 9-11 years (categorized as: 0 = malnutrition, z-score < -2; 1 = normal weight, z-score ≥ -2 and ≤ +1, and 2 = overweight, z-score > +1) [36]; (c) 4th follow-up (adult age): adult's BMI (categorized as: 0 = malnutrition, < 18.5kg/m^2; 1 = normal weight, 18.5-24.99kg/m^2; and 2 = overweight, ≥ 25kg/m^2) [37] and report

of history of parental obesity by the participant (categorized as 0 = no report of parental obesity; 1 = yes, report of maternal or paternal obesity; or 2 = yes, report of both maternal and paternal obesity).

Outcome: asthma

Asthma was a continuous latent variable deduced from the correlation between five indicators in EFA: (a) self-report of a medical diagnosis of asthma (1 = yes or 0 = no); (b) presence of wheezing in the last 12 months (1 = yes or 0 = no); (c) measurement of bronchial hyperresponsiveness (categorized as: 1 = ≤ 4mg/mL to indicate bronchial hyperresponsiveness or obstructive pattern with compatible spirometry or 0 = > 4mg/mL, absence of bronchial hyperresponsiveness); (d) sensation of chest tightening sometimes when waking up during the last 12 months (1 = yes or 0 = no); (e) shortness of breath during the day at rest over the last 12 months (1 = yes or 0 = no); shortness of breath during the night over the last 12 months (1 = yes or 0 = no). From these indicators, those with convergent loads in EFA (above 0.50) were selected. This latent variable was evaluated in CFA, adopted according to the measures analyzed previously.

Proposed theoretical model

In the proposed theoretical model, the "prenatal SES" would be a more distal determinant (exogenous latent variable), exerting its effects on the development of asthma outcome and on all the remaining dependent variables of the model. Parental obesity, as a proxy indicator of the interaction of genotypic-phenotypic components in the history of obesity in the family environment, potentially affects the other variables related to overweight (at birth, schoolchild and adult life). In a temporal sequence, lifetime overweight variables (parental obesity, overweight at birth and schoolchild and adult overweight) may be associated with asthma in adult life. Finally, as a more proximal determinant, "current adult SES" would be associated with an increased risk of overweight and asthma during adulthood (Figure 2).

Structural equation modeling (SEM)

SEM is an epidemiological tool used to test hypotheses on relationships between latent variables (non-observed variables) and observed variables, permitting the analysis of a set of structural equations [38].

The WLSMV estimator, theta parameterization for the control of residual variances [38], was used in SEM, in addition to weighting the estimates by loss of follow-up. To determine whether the model showed good fit, the same estimates previously described for CFA were considered [21,34]. The chi-square, degrees of freedom and p-value were evaluated but were not adopted as parameters for model fitting due to their sensitivity to sample size.

The command for the verification of modification indices (modindices) was used to test if suggestions could improve model fit. Modindices values higher than 10 were used to generate a new model [21], as long as these pathways were plausible from a theoretical viewpoint.

The effect of observed variables and latent variables on the outcomes were analyzed as standardized coefficients (SC), being considered significant when p-value < 0.05. The total, direct and indirect effects were estimated [38].

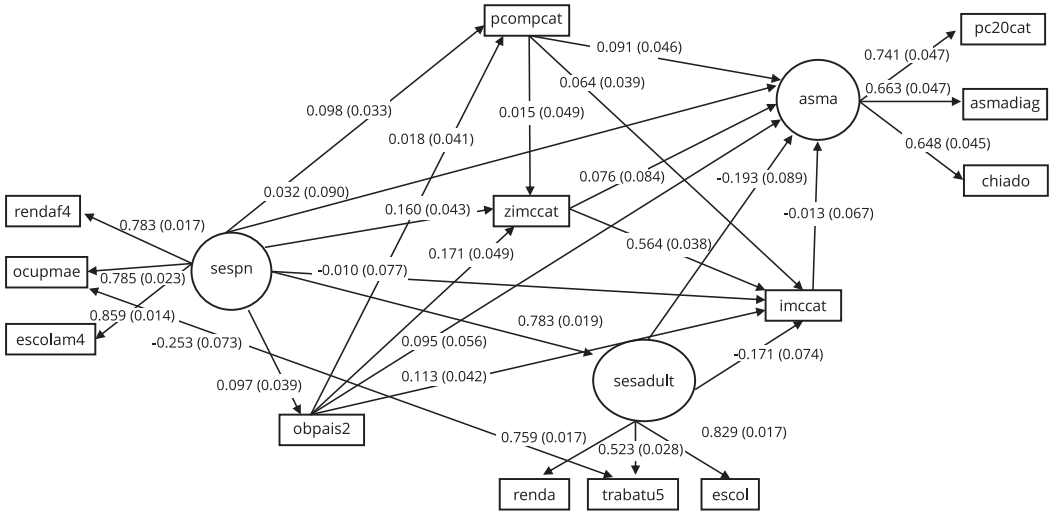
Results

The characteristics of the adults from the cohort and of their parents are listed in Tables 1 and 2. Among the adults, the prevalence of bronchial hyperresponsiveness was 20.7% (n = 427), the prevalence of medical diagnosis of asthma was 15.2% (n = 313) and the prevalence of wheezing over the last 12 months was 19.4% (n = 401) (Table 2).

The initial latent variable "Asthma" in EFA showed convergent loads higher than 0.50 for the following indicators: medical diagnosis of asthma (standardized coefficient – SC = 0.745), wheezing (SC = 0.553) and measurement of bronchial hyperresponsiveness (SC = 0.702), with these three

Figure 2

Structural equation model exploring the effects of socioeconomic situation and lifetime overweight on adult asthma. 1978/1979 Ribeirão Preto Birth Cohort, São Paulo, Brazil, 1978-2002/2004.



asma: latent variable of asthma; asmadiag: diagnosis of asthma of the 4th cohort follow-up. chiado: presence of wheezing in the last 12 months of the 4th cohort follow-up; escol: years of study for the adult schooling of the 4th cohort follow-up; escolam4: maternal schooling at birth; imccat: adult BMI of the 4th cohort follow-up; obpais2: history of parental obesity; ocupmae: maternal occupation at birth; pc20cat: measurement of bronchial hyperresponsiveness in mg/dL of the 4th cohort follow-up; pcompcat: weight per length z-score at birth; renda: monthly family income of the 4th cohort follow-up; rendaf4: family income at birth; sesadult: latent variable of the socioeconomic situation of the adult of the 4th cohort follow-up; sespn: latent variable of the socioeconomic situation of the family at birth; trabatu5: adult occupation of the 4th cohort follow-up; zimccat: BMI for schoolchild age of 9-11 years.

indicators being used in the formation of the latent variable "Asthma" in the SEM analysis (data not shown in tables).

In the SEM model, each indicator of the latent variables "prenatal SES" and "current adult SES" had factorial loadings above 0.60 and all the indicators of these latent variables had significant p-values ($p < 0.0001$) (Table 3; Figure 2).

Higher "prenatal SES" values were associated with parental obesity ($SC_{direct} = 0.101$, $p = 0.009$) and with overweight at birth ($SC_{total} = 0.131$, $p < 0.0001$, and $SC_{direct} = 0.126$, $p < 0.0001$) (Table 4).

Higher "prenatal SES" values ($SC_{total} = 0.183$, $p < 0.0001$, and $SC_{direct} = 0.155$, $p < 0.0001$) and parental obesity ($SC_{total} = 0.170$, $P = 0.001$, and $SC_{direct} = 0.166$, $p = 0.001$) were associated with overweight at school age. "Prenatal SES" also had a positive indirect effect on overweight at school age, with this effect being mediated by parental obesity ($SC_{indirect} = 0.017$, $p = 0.039$) (Table 4).

Parental obesity ($SC_{total} = 0.211$, $p < 0.0001$, and $SC_{direct} = 0.115$, $p = 0.007$) and overweight at school age ($SC_{total} = 0.565$, $p < 0.0001$, and $SC_{direct} = 0.565$, $p < 0.0001$) were associated with adult overweight, whereas lower "current adult SES" values protected against adult overweight ($SC_{direct} = -0.171$, $p = 0.016$). Parental obesity also had a positive indirect effect on adult overweight, with this effect being mediated by overweight at school age ($SC_{indirect} = 0.094$, $p = 0.001$) (Table 4).

An association was also observed between "prenatal SES" and "current adult SES" ($SC_{direct} = 0.779$, $p < 0.0001$) (data not shown in the tables).

Among the lifetime overweight indicators studied here, parental obesity was associated with asthma continuous variables during adult life ($SC_{total} = 0.105$, $p = 0.047$). And, at the other extreme,

Table 1

Sociodemographic, economic and nutritional characteristics during the prenatal period and at school age. Ribeirão Preto, São Paulo, Brazil, 1978-2002/2004.

Variable	n	%
1st cohort follow-up – baseline		
Maternal schooling (years of study)		
0-4	920	44.6
5-8	557	27.0
9-11	331	16.0
12 or more	215	10.4
No information *	40	1.9
Maternal occupation		
Unskilled manual worker	1,748	84.7
Semi-skilled and skilled manual worker	231	11.2
Non-manual worker	46	2.2
No information *	38	1.8
Family income at birth (minimum wages) **		
0.0-1.9	309	15.0
2.0-2.9	329	15.9
3.0-4.9	443	21.5
5.0 or more	611	29.6
No information *	371	18.0
Nutritional status at birth		
Underweight	72	3.5
Normal weight	1,473	71.4
Overweight	367	17.8
No information *	151	7.3
2nd cohort follow-up – school age		
Nutritional status of the schoolchildren		
Malnutrition	51	2.5
Normal weight	892	43.2
Overweight	204	9.9
No information *	916	44.4
Total	2,063	100.00

* Values ignored or not provided;

** Values of the national minimum wage effective in 1978/1979.

Table 2

Sociodemographic, economic, nutritional and respiratory characteristics of adults. Ribeirão Preto, São Paulo, Brazil, 1978-2002/2004.

Variable	n	%
Adults' sex		
Male	995	48.2
Female	1,068	51.8
Adults' schooling (years of study)		
0-4	58	2.8
5-8	262	12.7
9-11	1,039	50.4
12 or more	704	34.1
Current occupation of the adults		
Uskilled and semi-skilled manual worker	424	20.6
Skilled manual worker	342	16.6
Non-manual worker	434	21.0
No information *	863	41.8
Family income of the adults (minimum wages) **		
< 1.0	220	10.7
1.0-2.9	461	22.3
3.0-4.9	631	30.6
5.0-9.9	404	19.6
≥ 10.0	196	9.5
No information *	151	7.3
History of parental obesity		
No	1,349	65.4
Yes, mother or father	288	14.0
Yes, mother and father	58	2.8
No information *	368	17.8
Nutritional status of the adults		
Malnutrition	118	5.7
Normal weight	1,201	58.2
Overweight	741	35.9
No information *	3	0.2
Bronchial hyperresponsiveness measurements		
> 4mg/mL (without bronchial hyperresponsiveness)	1,495	72.5
≤ 4mg/mL (with bronchial hyperresponsiveness)	427	20.7
No information*	141	6.8
Medical diagnosis of asthma		
Yes	313	15.2
No	1,739	84.3
No information *	11	0.5
Wheezing		
Yes	401	19.4
No	1,660	80.5
No information *	2	0.1
Total	2,063	100.00

* Values ignored or not provided;

** Values of the national minimum wage effective in 2002/2004.

Table 3

Factorial loadings, standard error and p-value for the final latent variables of prenatal socioeconomic situation, current socioeconomic situation of adults and asthma in adults, by structural equation modeling (SEM). Ribeirão Preto, São Paulo, Brazil, 1978-2002/2004.

Latent variable	Factorial loadings	Standard error	p-value
Prenatal SES *			
Family income	0.783	0.017	< 0.0001
Maternal schooling	0.857	0.015	< 0.0001
Maternal occupation	0.786	0.023	< 0.0001
Current adult SES **			
Family income	0.752	0.017	< 0.0001
Adult's schooling	0.842	0.016	< 0.0001
Current adult's occupation	0.601	0.022	< 0.0001
Asthma			
Bronchial hyperresponsiveness measurement	0.732	0.046	< 0.0001
Medical diagnosis of asthma	0.671	0.047	< 0.0001
Wheezing	0.649	0.045	< 0.0001

SES: socioeconomic situation.

* Prenatal SES: latent variable of the socioeconomic situation of the family;

** Current adult SES: latent variable of the current socioeconomic situation of the adults.

underweight at birth was positively associated with adult's asthma continuous variables (SC_{total} = -0.124, p = 0.009 e SC_{direct} = -0.131; p = 0.007) (Table 5).

Higher "prenatal SES values" (SC_{total} = -0.105, P = 0.009) and higher "current adult SES" (SC_{total} = -0.179, p = 0.041 e SC_{direct} = -0.182, p = 0.039) had a protective effect in adult's asthma continuous variables (Table 5).

Discussion

In the present study, parental obesity and overweight at school age were associated with overweight in adults, whereas a higher "current adult SES" protected them against this outcome. Parental obesity and underweight at birth were variables related to asthma. While the larger "SES of prenatal" and larger "SES of the adult" were protective of asthma in adult life.

Parental obesity was associated with adult obesity, having a direct effect as well as an indirect effect via overweight at school age. These findings are consistent with the literature, which shows that a higher maternal [39,40] or paternal [41,42] BMI is associated with a higher childhood BMI. Maternal obesity has already been associated with obesity of the child during adult life [43,44]. Meta-analysis findings have also revealed an association between a high childhood BMI and adult obesity [45]. Parental obesity appears as a marker for the complex interaction between genetic, epigenetic and environmental factors, thus illustrating the joint contribution of them and/or family shared trajectories [42].

Higher "prenatal SES" values were a risk factor for parental obesity, for birth overweight and for overweight at school age, while higher "current adult SES" values were a protective factor against adult overweight. These distinct effects of latent SES variables on overweight outcomes at different time points in cohort follow-ups reflect the nutritional transition that has been occurring over the last decades [46]. In Brazil obesity was shifting towards groups with greater social vulnerability [47].

The effect of parental obesity asthma in adult life may be explained by the development of synergic interactions between genes and environment, with the children of obese individuals being genetically predisposed to excess weight gain within obesogenic domestic environments [48]. Taken together, these factors may result in other inflammatory and/or chronic diseases, asthma being one of them [49].

Table 4

Effect of socioeconomic situation and of lifetime overweight outcomes on adult asthma. Ribeirão Preto, São Paulo, Brazil, 1978-2002/2004.

Explanatory variable	Intermediate outcomes of lifetime overweight	Effect	Factor loading	Standard error	p-value
Prenatal SES *	Parental obesity	Direct	0.101	0.039	0.009
	Nutritional status at birth	Total	0.131	0.033	< 0.0001
		Direct	0.126	0.034	
	Nutritional status of the schoolchildren	Total	0.183	0.042	< 0.0001
		Direct	0.155	0.044	< 0.0001
		Indirect via parental obesity	0.017	0.008	0.039
Parental obesity	Nutritional status of the schoolchildren	Total	0.170	0.049	0.001
		Direct	0.166	0.057	0.001
	Nutritional status of the adult	Total	0.211	0.039	< 0.0001
		Direct	0.115	0.042	0.007
		Indirect via nutritional status of the schoolchildren	0.094	0.029	0.001
Nutritional status of the schoolchildren	Nutritional status of the adult	Total	0.565	0.038	< 0.0001
		Direct	0.565	0.038	< 0.0001
Current SES of the adult **	Nutritional status of the adult	Direct	-0.171	0.071	0.016

SES: socioeconomic situation.

* Latent variable of the prenatal socioeconomic situation;

** Latent variable of the socioeconomic situation of the adult.

Even if parental obesity was the only variable related to lifetime overweight with an effect on adult's continuous asthma variable, the association of parental obesity with the intermediate outcomes of schoolchild nutritional status and of adult nutritional status may help explain why previous studies have detected an association of these last variables with asthma [6,9,16,50].

At the other extreme regarding weight, underweight at birth was associated to asthma in adult life. Meta-analysis studies have shown an association of both low birthweight [50] and high birthweight8 with the future risk of asthma using weight for gestational age. The present study added knowledge to this theme, since it classified the nutritional status at birth according to an anthropometric indicator, permitting the verification of underweight and overweight, as well as the pathways triggered from them over time.

Intrauterine mechanisms that affect immunity or pulmonary development have been suggested as possible explanations for this effect of fetal underweight on asthma [51]. However, the epigenetic mechanism of the sparing phenotype, by which the intrauterine environment of nutritional restriction increases the chance of adult obesity [52,53], may not be an explanation for our asthma findings since the effect of underweight at birth on asthma continuous variables was direct, without a pathway that would go through overweight.

The effect of "prenatal SES" and "current adult SES" on the risk of asthma corroborates findings from other studies which showed that the indicators related to a lower socioeconomic level are associated with a higher risk of asthma, using observed variables such as education, occupation, income, and material goods [54,55,56]. The present study contributes to this knowledge by showing that inequality situations represented by lower values of the latent variable SES, with a lower determination error and observed at two time points in life were associated with adult's asthma continuous variables.

These results showed that "current adult SES" exerted a proximal and direct protective effect on adult's asthma continuous variables, a fact that may be explained by the access to healthier dietary choices due to higher income and schooling [57,58], to labor activity or to living in urban environments with lower risks for asthma [54].

A limitation of this study was that the prenatal obesity variable was reported by the participants during the 4th cohort follow-up, without a precise indication of the beginning of this condition. How-

Table 5

Effect of socioeconomic situation and of lifetime overweight outcomes on adult asthma continuous variable. Ribeirão Preto, São Paulo, Brazil, 1978-2002/2004.

Variables explaining adult asthma/Effect	Factor loading	Standard error	p-value
Prenatal SES *			
Total	-0.105	0.040	0.009
Direct	0.027	0.091	0.764
Indirect via current adult SES **	-0.142	0.069	0.040
Current adult SES **			
Total	-0.179	0.088	0.041
Direct	-0.182	0.089	0.039
Parental obesity			
Total	0.105	0.053	0.047
Direct	0.100	0.056	0.072
Nutritional status at birth			
Total	-0.124	0.047	0.009
Direct	-0.131	0.049	0.007
Nutritional status of the schoolchildren			
Total	0.080	0.058	0.170
Direct	0.090	0.084	0.281
Nutritional status of the adult			
Direct	-0.018	0.067	0.785

Note: factorial loading of the effect evaluated.

* Latent variable of the prenatal socioeconomic situation;

** Latent variable of the socioeconomic situation of the adult.

ever, parental obesity was used as a proxy for the interaction of genotype-phenotype components in the history of the family environment [59]. In this respect, in thisstudy parental overweight was found to be the starting point for other indicators of lifetime overweight in the offspring.

It was not possible here to confirm the association between parental obesity and schoolchild overweight with childhood asthma [6] since these data were not available for the cohort. However, more distal variables linked to weight such as parental obesity and underweight at birth were associated with adult asthma.

Strong points of this study were the pioneering approach to the exploration of this set of variables as indicators of overweight in a manner interrelated with asthma during adult life, the use of a birth cohort of Brazilian children involving a large sample analyzed at three distinct times, i.e., birth, school age and adulthood, and the use of weighting by the inverse of the probability of participating in the follow-ups, minimizing the possibility of selection biases due to sample losses and increasing the external validity of the study.

We also emphasize the SEM analysis with the formation of latent variables for "prenatal SES" and "current adult SES", permitting the formation of these variables of complex definition and reducing the measurement errors. In addition, SEM permitted to estimate the total, direct and indirect effects, for a better interpretation of the results obtained, signaling these effects in the temporal relationships between lifetime overweight and asthma in adulthood.

Another strong point of this study was the continuous variable "Asthma", which included the variable observed with the bronchial hyperresponsiveness test by methacholine bronchoprovocation associated with other symptoms of the disease [60]. Other symptoms of asthma were included in the latent variable reducing the error of determination in the diagnosis of the disease. So far, only one study has used a latent variable for the construction of this asthma variable and was conducted on a target public differing from the present study, i.e., in children [61].

Parental obesity was the starting point for the overweight pathways in childhood and adulthood, with an evident transgenerational effect. A greater weight at childhood continued to be a risk for overweight during adulthood. The effect of "prenatal SES" was a marked risk for parental obesity and for overweight at birth and during childhood. On the other hand, SES at the fourth follow-up of the cohort was a protective factor against adult overweight, characterizing the nutritional transition that has been occurring in Brazil over the last few decades.

Regarding weight pathways in asthma, parental obesity and underweight at birth were risk factors for asthma continuous variables during adulthood. SES at the two follow-up times of the cohort was a protective factor against adult asthma. Taken together, these data suggest that public policies should focus on appropriate weight within the family environment and at birth as a form of preventing lifetime obesity and asthma.

Contributors

L. L. Padilha performed the data analysis and wrote the article. C. C. C. Ribeiro formulated the research question, wrote and revised the article. J. X. P. T. Nascimento, V. M. F. Simões, F. P. Vitti and A. A. M. Silva and E. O. Vianna has critically reviewed the article. V. C. Cardoso, M. A. Barbieri and H. Bettiol participated in the research and review of the article. All authors approved the final version of the article for publication.

Additional informations

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Resumo

Os estudos sobre obesidade e asma frequentemente analisam o peso em um determinado momento; portanto, a modelagem de trajetórias de sobrepeso ao longo da vida pode ajudar a explicar os aspectos temporais dessa relação. O estudo atual modelou as trajetórias na associação entre história de sobrepeso e asma na vida adulta, utilizando dados da Coorte de Nascimento de 1978/1979, Ribeirão Preto, São Paulo, Brasil (n = 2.063), coletados ao nascer (linha de base), na idade escolar (9-11 anos) e na idade adulta (23-25 anos). Foi proposto um modelo teórico para explorar a associação entre o sobrepeso ao longo da vida e asma na vida adulta, analisada pela modelagem de equações estruturais. Obesidade dos pais (CP – coeficiente padronizado$_{global}$ = 0,211, p < 0,001; CP$_{direto}$ = 0,115; p = 0,007) e sobrepeso na idade escolar (CP$_{global}$ = 0,565; p < 0,0001; CP$_{direto}$ = 0,565; p < 0,0001) mostraram associação com sobrepeso na idade adulta. Obesidade dos pais (CP$_{direto}$ = 0,105; p = 0,047) e estado nutricional ao nascer (CP$_{global}$ = - 0,124; p = 0,009; CP$_{direto}$ = -0,131; p = 0,007) mostraram associação com asma na idade adulta. "Condição socioeconômica" mais alta na vida adulta mostrou associação inversa com sobrepeso (CP$_{direto}$ = -0,171, p = 0,020) e com asma na vida adulta (CP$_{global}$ = -0,179; p = 0,041; CP$_{direto}$ = -0,182; p = 0,039). Obesidade dos pais mostrou um efeito transgeracional sobre o peso, como gatilho na infância e no sobrepeso na vida adulta. Em paralelo ao baixo peso ao nascer, a obesidade dos pais também esteve associada com asma na vida adulta. A condição socioeconômica na vida adulta mostrou efeito protetor contra sobrepeso e asma.

Sobrepeso; Estágios do Ciclo da Vida; Modelo de Equação Estrutural; Asma

Resumen

Los estudios que se centran en la obesidad y asma frecuentemente consideran el peso en un determinado momento; por este motivo, la creación de modelos de patrones de sobrepeso a lo largo de la vida quizás puede contribuir a elucidar aspectos temporales en esta relación. Este estudio modeló los patrones en la asociación de sobrepeso a lo largo de la vida con el asma en etapa adulta, usando datos de una cohorte nacimientos de 1978/1979, en Ribeirão Preto, São Paulo, Brasil (n = 2.063), considerando: nacimiento (base de referencia), edad escolar (9-11 años) y edad adulta (23-25 años). Se propuso un modelo teórico para analizar los efectos del sobrepeso a lo largo de la vida en el asma, durante la etapa adulta, analizado mediante modelos de ecuaciones estructurales. La obesidad de los padres (CE – coeficiente estandarizado$_{total}$ = 0,211, p < 0,001; CE$_{directo}$ = 0,115; p = 0,007) y sobrepeso en edad escolar (CE$_{total}$ = 0,565; p < 0,0001; CE$_{directo}$ = 0,565; p < 0,0001) estuvieron asociados con sobrepeso en la vida adulta. La obesidad de los padres (CE$_{directo}$ = 0,105; p = 0,047) y el estatus nutricional al nacer (CE$_{total}$ = - 0,124; p = 0,009; CE$_{directo}$ = -0,131; p = 0,007) estuvieron asociados con el asma en la vida adulta. Un "condición socioeconómica actual en la etapa adulta" más alto estuvo inversamente asociado con el sobrepeso (CE$_{directo}$ = -0,171; p = 0,020) y al asma en la vida adulta (CE$_{total}$ = -0,179; p = 0,041; CE$_{direct}$ = -0,182; p = 0,039). La obesidad de los padres mostró un efecto transgeneracional en el peso, desencadenando sobrepeso en la infancia y etapa adulta. Junto al bajo peso al nacer, la obesidad de los padres fue también un riesgo para el asma en la etapa adulta. Mientras que el estatus socioeconómico en la etapa adulta protegía tanto ante el sobrepeso como el asma.

Sobrepeso; Estadios del Ciclo de Vida; Modelos de Ecuaciones Estructurales; Asma

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