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The Influence Of Maternal Obesity On Adolescent Obesity In Santiago, Chile

Yunnuo Zhu
Yale University, yunnuo@gmail.com

THE INFLUENCE OF MATERNAL OBESITY ON ADOLESCENT OBESITY IN SANTIAGO, CHILE

Yunnuo Zhu

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Abstract

***Objective*:** To examine the relationship between adolescent obesity and associated maternal obesity variables of interest, specifically maternal pre-pregnancy obesity and maternal obesity 10 years postpartum.

***Design*:** Secondary data analysis of a longitudinal study in Santiago, Chile (N=786 adolescents). Variables were extracted from multiple waves of data collection. The maternal obesity variables of interest and covariates were stratified by gender and associations of interest examined using univariate logistic regression analyses and multivariate logistic regression models with missing data imputation. Covariates included were birth weight percentile, breastfeeding for 6 months, father's presence, maternal age, and maternal education .

***Results*:** Adolescent obesity was associated with maternal obesity in both males and females. Female obese adolescents had a positive association with maternal obesity at 10 years postpartum (OR= 3.65, CI= 1.19, 11.14) and a negative association with father's presence in the household (OR= 0.236, CI= 0.07, 0.76). Among male adolescents obesity was positively associated with maternal pre-pregnancy obesity (OR= 9.59, CI= 1.25, 73.31).

***Conclusions*:** Maternal obesity status is an important predictor of adolescent obesity. Maternal obesity was a significant predictor of obesity among males and females but at chronologically different periods of the child's development. This may be attributed to gender related differences in genetic, behavioral, and/or environmental factors. Adolescent obesity intervention programs should provide varied approaches that cater to the different needs of males and females. Future research on maternal and other influences of adolescent obesity should focus on quantitative collection of parental and peer-related measures of anthropometrics, physical activity, dietary habits, as well as other sociobehavioral determinants of health.

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I. Introduction

Over the past couple decades, worldwide obesity rates have increased dramatically, more than doubling from 1980 to 2008.[1] The WHO estimates that as of 2010, there are approximately 43 million children under the age of five who are overweight and at risk of becoming overweight adults [2]. The relationship between childhood and adult obesity has been identified in multiple studies [3-6] and emphasizes the need to better understand and address potential pathways. While childhood obesity has its own immediate morbidity and mortality risks, its progression through adolescence into adulthood brings with it long-term medical and financial consequences. Overweight and obesity is currently ranked within the top five risk factors for death for middle- and high-income countries [1] and is associated with increasing mortality rates for type-2 diabetes, hypertension, and heart disease Therefore, identifying risk factors for infant and adolescent obesity and addressing early childhood predictors of obesity can mitigate widespread individual and social consequences of obesity.

Current research on early parental predictors of childhood and adolescent obesity focuses on maternal pre-pregnancy health in relation to fetal development, family socioeconomic status, and birth outcomes as predictors of childhood weight. Maternal pre-pregnancy weight and excessive weight gain during pregnancy have been the focus of previous studies investigating their association with childhood obesity due to its position as an early life, modifiable variable critical to early fetal development in the pathway to neonatal and long-term outcomes [7-10]. Obese mothers are more likely to give birth to large for gestational age (LGA) infants [10] although the pathway between the two have not been clearly identified [11, 12]. Studies have found infant birth weight to have a J-shaped curve association with later prevalence of overweight and obesity[13], thus showing large for gestational age children to have higher BMIs later on in childhood [12, 14, 15] and adulthood as well [5, 10]. A 1997 study by Whitaker et al. found the risk of obesity in adolescence to be almost three times greater if the mother was obese [5]. These studies show a strong long-term relationship between maternal pre-pregnancy weight and infant weight.

Breastfeeding has also been identified as a predictor of childhood obesity by some but not all studies. Breastfeeding exclusively for the first 6 months of life has been recommended both by the WHO and the American Academy of Pediatrics [16, 17]. Some studies and reviews have associated breastfeeding with a moderate decreased risk in obesity beyond the infancy period [18, 19] Harder et al.'s meta-analysis suggests a dose dependent relationship between breastfeeding duration and decreased risk of obesity in

early childhood and adolescence [20]. Other longitudinal studies such as Michels et al.'s Nurses' Health Study II did not identify any significant long term benefits of breastfeeding for at least 6 months for obesity risk in adolescence or adulthood [21]. Despite the inconclusive association with obesity, breastfeeding is still recommended due to other potential health benefits such as reducing respiratory tract infections or sudden infant death syndrome and health benefits for the mother including reduced risk of ovarian and breast cancer, hypertension and associated chronic diseases [22-27].

While the focus on parental predictors is heavily concentrated on maternal influence, paternal influence and overall family socioeconomic status may also play a significant role. Low socioeconomic status represented by parental income, education, or single parent households has been identified as a risk factor for obesity in the offspring[28, 29] although many other studies argue that socioeconomic status and race are a part of a complicated web of relationships that is not yet fully understood [30]. Hanson and Chen suggest that the association between low socioeconomic status and adolescent obesity is through poor diet and physical inactivity [31] while others such as Stematakis et al suggest that lower income families have less accessibility or awareness of health-related diet and physical activity recommendations[32]. Father's presence in the household has also been associated with obesity risk in adolescence. The Whitaker study identifying the risk of maternal obesity on infant weight gain in childhood and adulthood also found a lower yet still significant association of paternal obesity with future infant obesity [5]. Paternal absence, usually assessed as single-parent households, has been found in some studies to be significantly associated with childhood obesity [33, 34] whereas in others, to have no effect [35]. These socioeconomic and familial predictors reflect not only early parental influence on child obesity but also the development of personal risk factors in the children and adolescents.

This study examines early parental predictors of adolescent obesity in a unique context, at the end of the nutrition transition in Chile. Epidemiologically, the transition saw a decrease in undernutrition, maternal and infant mortality rates, and infectious diseases [36, 37]. Changes in dietary patterns, physical activity, and increased lifespan have led to an increased prevalence of obesity and chronic disease [38]. The transition presents a new parenting environment with new challenges and experiences potentially foreign to their upbringing. Changes in diet and food availability are risk factors for increased maternal and paternal BMI [5], excessive gestational weight gain [7], and present an unfamiliar landscape for parents to raise their children. In addition to the nutrition transition, this study captures infant growth data after the implementation of a successful nationwide breastfeeding promotion program [39]. This study is valuable in its ability to capture longitudinally the influence of parental risk factors on adolescent

obesity in this unique, transitioning environment. The objective of this analysis is to assess the association between maternal obesity indicators and adolescent obesity in a group of Chilean children. Specifically, this study attempts to answer the question: Does maternal pre-pregnancy obesity or maternal weight ten years postpartum independently influence the risk of obesity among adolescents?

II. Methods

a. Study Design

This study is a secondary data analysis examining the association between maternal weight indicators and adolescent obesity risk stratified by gender after adjusting for covariates. The variables used in these analyses are derived from a randomized controlled trial of iron-deficiency anemia prevention in Chilean infants with longitudinal follow-up waves of data collection [40].

Low- and middle-income working-class mothers from 4 contiguous working-class communities near Santiago, Chile were screened and enrolled in a double-blind, randomized, controlled trial on iron-deficiency anemia. Enrollment spanned from September 1991 to August 1996 until 1657 mother-infant pairs were recruited for the initial wave of the study.

Inclusion criteria for the infants included singleton, routine vaginal births with no major birth complications, congenital anomalies, no infant hospitalizations longer than 5 days, no chronic illness, and no phototherapy or iron therapy. There was a 3.0 kg birth weight cut-off regardless of gestational age due to preexisting iron supplementation programs in some Chilean clinics for infants weighing less than 3.0 kg. Exclusion criteria limited participants to those living within the 4 chosen communities; no other infant less than 12 months old in the household; no child in daycare; no illiterate or psychotic caregiver who is unable to consistently accompany child to appointments.

The original study protocol limited inclusion to infants being bottle-fed at 6 months of age in order to facilitate study-provided iron-fortified formula supplementation. However, in mid-1994, after preliminary study analyses showed significant difference in high and low iron formula supplementation for anemia prevention, the protocol was modified to allow for breast-fed infants with no iron supplementation. Due to the country's successful national breastfeeding program, all but 8 infants in the overall study were initially breastfed. The final study groups for the first wave of data collection totaled 1,657 infants. Follow-up data collection occurred at 5, 10, and 16 years of age. The follow-up cohort of 1127 returning children assessed at the 10 year mark represented a 69% retention rate. At the 10 year wave, parents provided written informed consent while children provided written assent. Adolescent data collection at the 16 year wave was successfully completed for 1062 participants. A more detailed explanation of the original study protocol is published with the primary study analyses [40]. The protocols and data collected from the study were approved and stored by the University of Michigan, University of California, San Diego, and the Institute of Nutrition and Food Technology, University of

Chile (INTA). Due to the secondary data analyses performed in this study using a data set with no personal identifiers, no IRB approval was required from Yale University.

While the original study focused on the affects of iron-deficiency anemia on behavioral and developmental outcome, the longitudinal data on maternal and child anthropometric variables as well as birth and household related variables allowed for the implementation of an analysis assessing the relationship between maternal body weight indicators and adolescent obesity. Using the key outcome, adolescent obesity, as our sample size limiting variable, 786 participants from the 16 year wave are eligible for this analysis.

b. Outcome Variable: Adolescent Obesity

Adolescent BMI was calculated using measured height and weight recorded during the 16 year data collection wave. Obesity in this study is defined as a z-score greater than 2. Study personnel used the Frankfurt position to assess subject height; subjects were weighed while wearing light clothing and no shoes. Due to the continuous collection of 16 year wave data over 5 years from 2007-2011, adolescents eligible for this secondary analysis were constrained to those who were 14.5-17.5 years of age during the time of data collection. Gender was extract from clinical birth records during the first wave of data collection that took place during infancy.

c. Key Independent Variable: Maternal Obesity

Mothers were asked to recall their pre-pregnancy weight during the 10-year data collection wave. Maternal weight and height were also measured during their 10 year visit and used to calculate their BMI at 10 years postpartum. The same methods described for measuring adolescent weight and height were used for maternal measures as well.

d. Covariates

Covariate measures included in this analysis are: birth weight percentile adjusted for gestational age, breastfeeding at 6 months, father's presence in the household, maternal age, and maternal years of education. Birth weight, length, and gestational age were extracted from infant clinical records. Birth weight percentile was calculated using birth weight and gestational age. To identify mothers who were breastfeeding at 6 months, clinicians asked if infants were still receiving breast milk at the 6 month check-up. This variable includes mothers who supplemented with formula or complementary feeding

and is not representative of exclusive breastfeeding. Father's presence, maternal age, and maternal years of education were self-reported by the caregiver present with the child during the initial evaluation. The caretaker was usually the mother and occasionally the father or grandmother. Maternal years of education was used as a proxy for socioeconomic status while maternal age was included to control for potential associations with maternal weight and differences in infant birth weight. Gestational age was not used as a covariate because exclusion criteria for the infancy wave excluded all infants born before 37 gestational weeks.

e. Statistical Analyses

Statistical analyses were conducted using SAS (version 9.2 and 9.3, SAS Institute, Cary, NC, US). Simple descriptive analyses for independent variables stratified by gender were obtained using PROC FREQ and PROC MEANS. The Mann-Whitney test and χ^2 test were used, respectively, for continuous and categorical variables to evaluate significant differences in obese and non-obese adolescents stratified by gender. Univariate logistic regression models were used to calculate odds ratios for predictor variables and identify independent associations with the outcome variable, adolescent obesity.

Pearson correlation coefficients were calculated to observe the simple bivariate correlations between all proposed study variables. The correlation matrix in Table 1 indicates a significant correlation between the two linear maternal weight variables, maternal pre-pregnancy weight and maternal weight 10 years postpartum. Multicollinearity between the two variables was assessed using a linear regression model. No multicollinearity was identified after assessing variance inflation factors, Eigenvalues, and condition indices which were all within acceptable ranges.

Due to a high percentage of missing data for some variables, multiple imputation via PROC MI was used for a more complete analysis of the relationship between adolescent obesity and identified predictor variables. The Markov chain Monte Carlo method (MCMC) using fully conditional specification (FCS) logistic regression method with 50 iterations was used for the imputation of the missing values in the dataset [41]. The bivariate Pearson correlation illustrates associations between the variables selected for this analysis.

Table 1: Pearson Correlation Table of Variables and Covariates for Regression

Correlation (Significance)	**Adolescent Obesity**	**Maternal Pre-pregnancy Obesity**	**Maternal Obesity at 10 Yr Postpartum**	**Birth weight Percentile**	**Breast Feeding at 6 Months**	**Father's Presence**	**Maternal Age at Birth**	**Maternal Education (Yrs)**
Adolescent Obesity	1.000							
Maternal Pre-pregnancy Obesity	**0.139*** <0.001	1.000						
Maternal Obesity at 10 Yr Postpartum	**0.079*** 0.035	**0.171*** <0.001	1.000					
Birth weight Percentile	0.023 0.522	**0.093*** 0.024	**0.129*** <0.001	1.000				
Breastfeeding at 6 months	-0.009 0.802	-0.003 0.937	-0.03980 0.2933	-0.005 0.890	1.000			
Father's Presence	**-0.100*** 0.011	-0.067 0.151	0.028 0.501	0.053 0.181	0.027 0.497	1.000		
Maternal Age at Birth	0.0120 0.7723	-0.052 0.208	**0.112*** 0.007	**0.154*** <0.001	-0.017 0.677	**0.163*** <0.001	1.000	
Maternal Education (Yrs)	0.004 0.932	0.027 0.509	-0.039 0.347	0.069 0.094	0.060 0.154	0.004 0.936	**-0.146*** <0.001	1.000

A complete case logistic regression was conducted prior to imputation in order to detect potentially significant predictor variables as well as to have a comparator for post-imputation regression analysis. By calculating the AIC value for the imputed regression models, it was clear that stratifying by sex could lead to a much better fit (smaller AIC value) compared to the initial model with no gender stratification. Using the imputed data, logistic regression models were created using PROC MIANALYZE stratified by gender for adolescent obesity with maternal pre-pregnancy weight, maternal weight at 10 years postpartum as the variables of interest and birth weight percentile, breastfeeding, father's presence, maternal age, and maternal education as potential covariates. For all tests of statistical significance, an α level of 0.05 was used as the cut-off value.

III. Results

a. Outcome Variable

Table 2 analyzes the descriptive characteristics of the study outcome of interest, obese and non-obese adolescents by maternal obesity indicators and covariates. The mean age of adolescents in this study was 16 years and 9 months with no significant difference by gender or weight status. Age fluctuated around 16 years due to the wide 5 year data collection interval. Prevalence of overall adolescent obesity in the study was 5.2% with no difference between female and male subjects (Table 2) despite a higher male birth weight percentile ($p<0.001$). While the average overall female BMI ($z=0.65$) was slightly higher than that of males ($z=0.43$), obese adolescent females had an insignificantly lower BMI ($z=2.20$) than their male counterparts ($z=2.29$).

Table 2: Description of the Sample by Adolescents Obesity Status Stratified by Gender

	Adolescent Obesity	No Adolescent Obesity	N (786)
Females	N=18 (4.57%)	N=376 (95.43%)	394 (50.13%)
Pre-pregnancy Obesity	8.3% (1/12)	1.9% (5/270)	282
*Maternal Obesity at 10 yr wave	68.8% (11/16)	37.7% (129/342)	358
Covariates			
Birth Weight Percentile^	61.9 ± 25.84	51.0 ± 24.32	394
Breastfed for 6 months or more	37.5% (6/16)	55.0% (199/362)	378
Family			
*Father's Presence	60.0% (9/15)	85.7% (264/308)	323
Maternal Age	29.2 ± 8.32	26.4 ± 5.70	279
Maternal Education (Years)	9.5 ± 2.97	9.4 ± 2.86	282
Males	N=23 (5.87%)	N=369 (94.13%)	392 (49.87%)
Pre-pregnancy Obesity[$]	11.1% (2/18)	1.0% (3/290)	308
Maternal Obesity at 10 yr wave	42.9% (9/21)	35.9% (123/343)	364
Covariates			
Birth Weight Percentile^	53.3 ± 21.07	58.1 ± 24.86	392
Breastfed for 6 months or more	65.2% (15/23)	56.8% (204/359)	382
Family			
Father's Presence	75.0% (12/16)	84.0% (251/299)	315
Maternal Age	25.1 ± 5.50	26.4 ± 6.34	308
Maternal Education (Years)	9.5 ± 2.94	9.5 ± 2.62	307

* $P < 0.05$ ^ adjusted for gestational age
Maternal Obesity at 10 yrs p=0.0174
Father's presence p=0.0169
[$] Fisher's Exact Test p=0.0291

b. Independent Variable

Reported overall maternal pre-pregnancy obesity was very low (1.9%) although 24.9% of the sample had missing data for pre-pregnancy BMI. Maternal obesity measured 10 years postpartum was more prevalent (34.6%) with only 8.1% of respondents having missing data for the variable. Overall maternal pre-pregnancy obesity and obesity at 10 years postpartum did not differ by adolescent gender. Among females, there was no association between adolescent obesity and maternal pre-pregnancy obesity (Table 3). The descriptive and univariate logistic regression analyses of obese male adolescents did show a positive association between obesity and maternal pre-pregnancy obesity (Tables 2, 3). However, with only two reports of pre-pregnancy obesity in the sample of obese males, these analyses required imputation of missing data for final interpretation.

Table 3: Univariate Logistic Regressions for Adolescent Obesity

Variable	Odds Ratio	OR 95% Confidence interval
Females		
Pre-pregnancy Obesity	4.818	(0.518, 44.814)
Maternal Obesity at 10 yr wave*	3.633	(1.234, 10.691)
Covariates		
Birth Weight Percentile	1.018	(0.998, 1.039)
Breastfeeding at 6 Months	0.491	(0.175, 1.381)
Father's Presence*	0.250	(0.085, 0.737)
Maternal Age	1.080	(0.984, 1.185)
Maternal Education (Years)	1.011	(0.824, 1.239)
Males		
Pre-pregnancy Obesity#	11.958	(1.864, 76.716)
Maternal Obesity at 10 yr wave	1.342	(0.550, 3.273)
Covariates		
Birth Weight Percentile	0.992	(0.975, 1.009)
Breastfeeding at 6 Months	1.424	(0.589, 3.445)
Father's Presence	0.574	(0.178, 1.854)
Maternal Age	0.965	(0.891, 1.045)
Maternal Education (Years)	1.001	(0.835, 1.199)

*$P < 0.05$ #Statistically significant but sample size too small for interpretable significance

Maternal obesity at 10 years postpartum was significantly more prevalent in the sample of obese female adolescents compared non-obese females (Table 2). Univariate logistic regression analyses identified a positive association between obese adolescent females and maternal obesity at 10 years postpartum (Table 3). Maternal obesity at 10 years postpartum showed no association with adolescent male obesity.

c. Covariates

The average birth weight percentile of the 786 infants in this study was 54.7%. In general, male infants had a higher birth weight percentile than female infants (57.8% vs. 51.5%; $p<0.001$). When comparing the birth weight of normal and obese adolescents, obese adolescents were found to have a slightly higher but insignificant birth weight percentile. There was also no significant difference in breastfeeding at 6 months by gender or adolescent obesity status. Univariate logistic regressions for adolescent males and females both showed no significant association between birth weight percentile or breastfeeding at 6 months and adolescent obesity (Table 3). While fathers were absent from the household for 13.0% of the sample while 18.8% had missing data for this variable. The univariate logistic regression model for female adolescents showed a negative association ($p=0.01$) between adolescent obesity and father's presence. By comparison, this association was not found among males.

The average maternal age at time of childbirth was 26.4 years and did not significantly differ by infant gender or by adolescent obesity. The majority of mothers in the study completed education up to the mandatory 8 years of primary school education with an average of 9.5 total years. Maternal age and maternal years of education did not vary by gender or weight status (Table 2). Neither was associated with adolescent obesity in the univariate logistic regression analyses.

d. Findings from Analyses with Imputed Data

The logistic regression model found that among adolescent females and males, there were significant associations between maternal and adolescent obesity. Maternal obesity at 10 years postpartum was significantly associated with female adolescent obesity after adjusting for all covariates (Table 4). An odds ratio of 3.65 (CI 1.194, 11.139) indicates a strong positive association. For adolescent males, the logistic regression model found a significant association with maternal obesity as well, maternal pre-pregnancy obesity in this instance (Table 4). The earlier issues with small sample were somewhat alleviate by the imputation process but may still be a problem; the large confidence interval reflects a sample size that is still quite small and calls for some uncertainty regarding the variable's significance.

Table 4: Multivariate Logistic Regression Models by Gender with Imputed Data

Multiple Imputation Logistic Regression among Females				
Predictor	**Coefficient**	**OR**	**95% Confidence Interval**	**P=**
Pre-pregnancy Obesity	0.549	1.731	(0.144, 20.761)	0.6650
Maternal Obesity at 10 yr Postpartum*	1.294	3.647	(1.194, 11.139)	0.0231
Covariates				
Birth Weight Percentile	0.010	1.010	(0.988, 1.032)	0.3745
Breastfeeding at 6 Months	-0.807	0.446	(0.147, 1.355)	0.1543
Father's Presence*	-1.443	0.236	(0.073, 0.764)	0.0160
Maternal Age	0.0607	1.062	(0.962, 1.174)	0.2314
Maternal Education (Years)	0.053	1.055	(0.867, 1.284)	0.5934

Multiple Imputation Logistic Regression among Males				
Predictor	**Coefficient**	**OR**	**95% Confidence Interval**	**P=**
Pre-pregnancy Obesity*	2.261	9.589	(1.254, 73.310)	0.0294
Maternal Obesity at 10 yr Postpartum	0.372	1.451	(0.552, 3.811)	0.4504
Covariates				
Birth Weight Percentile	-0.009	0.991	(0.973, 1.009)	0.3306
Breastfeeding at 6 Months	0.468	1.596	(0.639, 3.987)	0.3165
Father's Presence	-0.561	0.571	(0.181, 1.798)	0.3377
Maternal Age	-0.025	0.975	(0.896, 1.061)	0.5562
Maternal Education (Years)	-0.005	0.995	(0.830, 1.194)	0.9605

The only covariate associated with female adolescent obesity in the multivariate logistic regression model was father's presence (Table 4). Father's presence was negatively associated (OR 0.24; CI 0.073, 0.764) with adolescent obesity, suggesting that father's presence is potentially protective against adolescent obesity in females. None of the other predictor variables, birth weight percentile, breastfeeding at 6 months, maternal age, or maternal education, were even marginally associated with female obesity. For the male multivariate logistic regression model, no covariates were associated with adolescent obesity.

IV. Discussion

In this longitudinal Chilean study, findings suggest that maternal obesity is significantly associated with adolescent obesity and that gender differences exist in parental predictors of adolescent obesity. Maternal obesity had a significant positive association with adolescent obesity for both males and females but the influence of maternal obesity as a function of timing differed by gender. While maternal pre-pregnancy obesity was associated with obesity among adolescent males, maternal obesity at 10 years postpartum was associated with adolescent obesity amongst females. Both genders showed a slight adjusted negative correlation between father's presence and adolescent obesity but father's presence was found to have a protective association only for adolescent obese females. All other covariates, birth weight percentile, breastfeeding at 6 months, maternal age, and maternal education, were insignificantly associated with adolescent obesity for either gender.

Maternal obesity at 10 years postpartum may play a social and environmental role explaining its association with daughters' adolescent obesity. This analysis along with previous studies suggests that there is a stronger connection to family environment and maternal behaviors in adolescent girls than boys. Maternal obesogenic factors such as physical activity and dietary intake may affect and influence obesity in their daughters. Daughters from families with low levels of physical activity and high dietary intake were more likely to consume poor diets, perform less physical activity, and have increased weight gain [42]. Adolescent females may be more likely to inherit maternal habits than adolescent males. Park et al. found that not only are mothers more significantly influential on child obesity than fathers, but also that females are more likely to be affected by parental obesity [43, 44]. Home food environment studies have found harmful maternal dietary habits and the presence of unhealthy foods at home to be positively associated with the consumption of high energy drinks and snacks in adolescent girls [45]. As food purchasing responsibilities usually lie with the mother, maternal food choices are reflected in their own weight as well as that of their children [46]. In addition to diet and physical activity, genetics and potential lifestyle associated epigenetics may play a role in influencing the relationship between maternal and adolescent female obesity.

Male adolescent obesity was found to be significantly associated solely with maternal pre-pregnancy obesity, a variable commonly attributed as an early maternal predictor of obesity in their children. [5, 10]. Maternal weight at 10 years postpartum was not found to be a significant predictor of male adolescent obesity suggesting a potentially different behavioral, genetic, or behavior-related epigenetic

relationship between maternal and male adolescents compared to female adolescents. One culturally specific study in Korea noted that adolescent boys are more likely to be out of the house spending time with their peers than with family [43]. Cultural attitudes towards adolescent males and females may also differ in Chile. Previous studies have identified that male dietary choices are associated with maternal dietary choices or parenting styles but neither study controlled for potential peer influences [45, 47]. With regards to physical activity, maternal influence has been found to play a minimal role in predicting physical activity in their sons [48] while father's physical activity has been found to be predictive [49, 50]. It is possible that the unmeasured variable, paternal physical activity, is a more significant predictor of adolescent obesity than maternal dietary habits [51] or that peer relationships play a larger role in adolescent male behavior than parental influences [52, 53]. Future studies looking into predictors of male adolescent obesity may benefit from including variables pertaining to paternal physical activity involvement, perceived peer support, and self-reported physical activity levels.

Infant birth weight percentile was not a significant covariate of adolescent obesity for either gender. Due to exclusion criteria, low birth weight infants were not included in the study and therefore the lack of small for gestational age infants may bias the results towards the null. Low birth weight and preterm infants were excluded from the primary study design but have been identified as at risk populations for "catch-up" growth and later development of obesity and metabolic syndrome [10, 54].

Breastfeeding up to and beyond the recommended 6 months was not a significant protective predictor of adolescent obesity for either gender in this study. While breastfeeding's potential protective effects against obesity are affirmed as a benefit of breastfeeding by health organizations such as the WHO and American Academy of Pediatrics [16] and a recent meta-analysis by Arenz et al. [55], the scientific evidence remains inconclusive. The measure used in this study allowed for formula supplementation and did not account for complementary feedings. This may speak to the importance of breastfeeding exclusivity and the need to account for alternative feeding measures. Also, due to Chile's successful national breastfeeding campaign, all infants in this study were initially breastfed and therefore had some degree of exposure to the potential benefits of breastfeeding.

Father's presence was the sole covariate to be found associated with female adolescent obesity independent of maternal obesity. With an odds ratio of 0.24, father's presence may have a strong protective effect on adolescent female obesity. The measure of father's presence may represent both financial and social involvement of the father in their child's development and was protective of

adolescent obesity for females in this study. Conversely, father's absence has been associated with earlier onset of female puberty [56], lower household income [57], and decreased social support [58]. Earlier onset of puberty may be a result [59] and a risk factor for obesity in girls [60]. Changes in leptin and insulin resistance that occur during puberty can increase the risk of weight gain [61]. Physical activity decline is also associated with early puberty and increased BMI [62, 63].

Paternal absence from the household is also associated with lower household income [64] and increased maternal employment. The emphasis of financial responsibility on the mother can lead to individual and family stress [65] as well as less interaction with their children [58]. The lack of interaction, supervision, and increased stress are all associated with poor eating habits, more fast food, and less maternal supervision [66]. Single-mother and mother-stepfather headed households have been previously associated with skipping breakfast, which in turn has been associated with increased obesity risk [67, 68]. The significance of father's presence as a protective risk factor against adolescent obesity in females only suggests a gender differential in the sensitivity to family related stress and dietary habits. Paternal parenting styles have been found to play a protective role in vegetable and fruit intake in daughters but not sons [47]. Females may be more responsive to paternal social cues.

Maternal education and age were not associated with adolescent obesity for either gender. These variables may be insignificant again due to the strict exclusion criteria. Low birth weight and preterm infants are associated with the two extremes ends of maternal age and education [69, 70]. The absence of these infants from the study population leads to a bias towards the null of the significance of these variables [69, 71, 72]. The primary study was focused on a fairly uniform lower-middle class population so there may not be enough variation within the study group for any significant associations.

Several limitations of this secondary analysis should be noted. The use of recalled pre-pregnancy weight during the 10 year wave is potentially inaccurate and subject to recall bias and missing data. While all other weight and height measures were measured directly in the original study, this crucial independent variable was not. Due to the potential underreporting of pre-pregnancy weight, its significance in this study may be underreported and potentially bias its effects towards the null. Potential associations between pre-pregnancy obesity and adolescent obesity may be stronger if accurate weight measures were reported. The absence of data on exclusive breastfeeding and complementary feeding may also be biasing the relationship between these potential predictors and adolescent obesity towards the null. Early supplemental formula and complementary foods may decrease the protective effect of breastfeeding.

Although this analysis aimed to identify parental predictors of adolescent obesity, including other sources of potential predictors and related variables such as dietary, physical activity, and social support indicators could provide a much more comprehensive analysis. Finally, the inclusion of Tanner stages in this analysis would have also improved the accuracy of identification of the puberty transition in the study.

V. Conclusion

This study allowed for a better understanding of the significance of maternal weight and other covariates as a predictor of adolescent obesity in a longitudinal study setting. Chile's nutrition transition provided a novel setting for observing the effects of rapid industrialization and urbanization on a new generation of modern Chilean citizens. Parents raising their children in this new environment encounter new experiences in food choices, employment opportunities, and lifestyle changes that the generations before them had not confronted. Consistent with previous research, maternal obesity was a significant predictor of adolescent obesity. However, the temporal significance and influence of maternal obesity on adolescent obesity differed by gender. This study corroborates findings that adolescent girls are responsive to maternal dietary and physical activity behaviors as well as the family stress and lack of social support of a father's absence from the household. Obese male adolescents were found to be associated with maternal pre-pregnancy obesity but not any other covariates. This relationship supports prior research on biological long-term risks of maternal obesity and also suggests that other potential variables of influence are more significant in the association with male adolescent obesity.

These findings identified potential areas and opportunities for health promotion and obesity prevention. Adolescence is a pivotal period of growth and development that foreshadows subsequent adult health and potential health risks. By identifying modifiable predictors of adolescent obesity, prevention programs can be implemented at the most effective time-points to allow for the most efficient and cost-effective preventions and modifications. Prevention and support programs are especially crucial when dealing with obesity due to not only its high risk of other chronic diseases but also because of its persistence from generation to generation.

The findings from this analysis provide a unique contribution to the current body of literature due to its unique longitudinal analysis of adolescent obesity predictors in a middle-income country. The association of maternal obesity or other parental risk factors and adolescent obesity may differ by gender and may be attributed to genetic, behavioral, and environmental factors. Potential prevention and education programs should target adolescent and pre-pregnancy stages to promote healthier eating, physical activity, self-sufficiency, and alternative sources of social support. Obesity prevention programs should offer a variety of strategies in order to appeal to the varying needs of males and females. Future research should be conducted on larger more diverse populations. Longitudinal studies should encompass pre- and post-pregnancy data collection waves and focus on collecting valid

quantitative measures of anthropometrics, physical activity, diet, as well as sociobehavioral determinants of health. Data collection should encompass as many facets of potential adolescent influence as possible including maternal, paternal, and peer-related measures. Qualitative research may also provide supplemental information to improve understanding of adolescent gender differences in response to the influence of parental factors on their weight status.

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