Asthma and Maternal Body Mass Index Are Related to Pediatric Body Mass Index and Obesity: Results from the Third National Health and Nutrition Examination Survey

Leonard H. Epstein, *§ Yow-Wu B. Wu, † Rocco A. Paluch, * Frank J. Cerny, ‡ and Joan P. Dorn§

Abstract

EPSTEIN, LEONARD H., YOW-WU B. WU, ROCCO A. PALUCH, FRANK J. CERNY, AND JOAN P. DORN. Asthma and maternal body mass index are related to pediatric body mass index and obesity: Results from the Third National Health and Nutrition Examination Survey. *Obes Res.* 2000;8:575–581.

Objective: Clinical research has shown an increased prevalence of obesity in children with asthma. This study was designed to assess the relationship between asthma and pediatric body mass index (BMI) in a national database and to examine factors that may modify this relationship.

Design: The cross-sectional relationship between asthma and pediatric BMI and obesity (BMI \ge 85th percentile) was studied. Variables that may influence the relationship between asthma and pediatric BMI, such as race/ethnicity and television watching were included in the model for the total sample. A smaller sample of 3009 white and African American youth were studied in regression models including maternal BMI.

Study Population: A nationally representative crosssectional sample of 5154 children and adolescents of 6 to 16 years of age from the Third National Health And Nutrition Examination Survey.

Results: In the full sample, asthma and television watching were related to BMI, accounting for 3% of the variance in BMI. When maternal BMI was included in the non-Hispanic sample, television watching, maternal BMI, and

the interaction of maternal BMI and asthma were related to youth BMI, accounting for 15% of the variance. The standardized BMI z-score for those youth without asthma and no maternal obesity was 0.06, which increased to 0.33 if the youth had asthma, to 0.70 if the youth did not have asthma but the mother was obese, and to 1.71 if the youth had asthma and the mother was obese. Asthma, television watching, and maternal BMI were independent predictors of youth obesity.

Conclusions: BMI and prevalence of obesity is higher in youth with asthma. Pediatric BMI, but not obesity, is also related to the interaction of asthma and maternal BMI in white and African American youth. Comorbidity of asthma and obesity may complicate treatment of either condition, and prevention of obesity should be encouraged for asthmatic children.

Key Words: obesity, asthma, youth, maternal obesity, race

Introduction

Obesity and asthma are two major causes of morbidity in the United States (1,2), with asthma the principle cause of chronic illness and school absenteeism in children (1). These are also two of the most prevalent pediatric disorders in the United States. Approximately one-fourth of children and adolescents are obese (2), and 10% to 15% of boys and 7% to 10% of girls are diagnosed with asthma during childhood (3). The prevalence of obesity (2) and asthma (1,4) is increasing.

Recently, we examined the relationship between asthma and obesity in a predominantly Hispanic inner-city pediatric sample of 171 children and adolescents and found that youth with asthma had significantly greater body mass index (BMI) values than youth without asthma, and that >2.5 times as many children with asthma were obese com-

Submitted for publication November 2, 1999.

Accepted for publication in final form May 16, 2000.

^{*}Departments of †Pediatrics, Nursing, ‡Physical Therapy, Exercise, and Nutritional Sciences, and §Social and Preventive Medicine, State University of New York, Buffalo, New York.

Address correspondence to Leonard H. Epstein, Ph.D., Division of Behavioral Medicine, Department of Pediatrics, State University of Buffalo, Farber Hall, Room G56, 3435 Main Street, Building No. 26, Buffalo, NY 14214-3000. E-mail: lhenet@acsu.buffalo.edu. Copyright © 2000 NAASO

pared with children without asthma (5). These results are consistent with the relationships that have been observed between obesity and asthma for other groups of minority children (6,7), but it is important to confirm these clinical observations on a more representative sample.

Identification of comorbidity between these two prevalent pediatric disorders has important implications for the prevention and treatment of these problems. If asthma is a risk factor for the development of obesity, it may be important for pediatricians to monitor BMI in youth with asthma and associated risk factors to prevent comorbidity of asthma and obesity. If obesity does develop in children with asthma, implementation of dietary and activity programs is needed. However, there may be limitations in attempts to increase physical activity in children with asthma. Children with asthma frequently have exercise-induced bronchospasm, which can decrease their participation in sports and vigorous free play (1,8), and the aversiveness of subjective symptoms associated with exercise-induced bronchospasm (9) may make it more difficult to increase physical activity in an obese child with asthma.

The purpose of this study was to determine the relationship between asthma and obesity in a large pediatric sample. We hypothesize that asthma, along with the traditional risk factors for obesity of television watching (10) and maternal obesity (11), will be related to pediatric BMI. We also assess whether asthma interacts with other risk factors to increase youth BMI.

Research Methods and Procedures

Data and Sample

The Third National Health And Nutrition Examination Survey (NHANES III) assesses a representative sample of the population in the United States. Data were collected in two phases by the National Center for Health Statistics of the Center for Disease Control and Prevention, between 1988 and 1991 and between 1991 and 1994. The total sample interviewed across all ages was 33,994. Our sample was composed of the 5154 youths with obesity and asthma data between the ages of 6 and 16, which was 76.3% of the available sample.

Measurements

The major dependent variable was pediatric BMI (BMI = kg/m^2 , height measured with a stadiometer and weight, with a balance beam scale). To control for age and gender influences, BMI values for children, mothers, and fathers were converted to z-scores using gender- and age-appropriate means and SDs reported in NHANES II (12). Obesity status was based on being equal to or greater than the 85th percentile BMI in the NHANES II reference data.

Race and ethnicity codes were developed from the NHANES questions on race and ethnicity. Participants were

classified as white, African American, or Hispanic based on responses to the race/ethnicity question. Additional subjects were classified as Hispanics based on affirmative responses to Mexican American of unknown race on the race question and other Hispanic ethnicity on the ethnicity question. Asthma status (yes/no) was determined by the parent-completed item, "Did a doctor ever say that (*child's name*) had asthma?" Hours of watching television were estimated with a question from the youth examination that asked, "How many hours of television did (*child's name*) watch yesterday?," ranging from 0 to \geq 5 hours. Family income was estimated using the poverty income ratio, which is based on family income and family size. A ratio of 1 is at the poverty level.

Analytical Plan

Analyses and descriptives statistics used NHANES weighting variables that take the complex sampling design into account to estimate population values. Analyses were completed with replicate weights for estimating variance and SEs for testing parameter coefficients, using the Wes-Var PC software package (Westat, Rockville, MD) (13). The full sample weight variable was used, with Fay's method of balanced repeated replication, using a perturbation factor of 70% as the replication method. Multiple linear regression models were constructed to examine variables that predicted youth BMI standardized for age and gender. Categorical predictor variables were dummy-coded before entry into the model. Interactions between asthma and gender, age, race/ethnicity, television watching, and maternal BMI were explored. In addition, logistic regression models were used to predict youth obesity. Odds ratios (ORs) for obesity were determined controlling for gender, age, and race/ethnicity.

The sample size for the relationship between obesity, asthma, and television watching was the entire cohort of children (N = 5154) with obesity and asthma data. In addition to these relationships, we also were interested in examining other variables that may relate to pediatric obesity, including parental BMI (11) and income (14). However, there was missing data for parental BMI and income. A series of χ -square analyses was conducted to evaluate whether inclusion of maternal BMI, paternal BMI, or income was associated with significant differences in the proportion of subjects who were eliminated for each racial/ethnic group. Inclusion of any of these variables produced a significant reduction in Hispanic subjects. When the sample was limited to non-Hispanic youth, maternal BMI could be studied without a significant loss of subjects between white and African American samples. However, when paternal BMI or income was included in addition to maternal BMI, missing data caused a significant reduction in the percentage of African American youth included in the sample. The second

set of analyses added maternal BMI as an additional predictor of pediatric BMI and obesity in the non-Hispanic population.

Results

Table 1 shows the descriptive characteristics of the entire sample. Age was equally distributed across race/ethnic groups. Slightly more than 25% (26.4%) of the sample was obese, and 11.6% had diagnoses of asthma. Results from the multiple regression model (Table 2) showed that asthma and television watching were related to obesity. Controlling for gender, age, and race/ethnicity, children with asthma were 1.53 times more likely (confidence interval [CI] = 1.25 to 2.46) to be obese than children without asthma, and children who watched 4 hours or more of television per day were 1.63 times more likely (CI = 1.42 to 2.48) to be obese than those who watched for 1 hour or less per day. No significant interactions between asthma and other predictors were observed. The model had an *R* of 0.18 and accounted for 3.4% of the variation in children's BMI. The logistic regression

predicting pediatric obesity was significant F(6,47) = 9.27, p < 0.001, accounting for 1.9% of the variability in pediatric obesity. Significant independent predictors of obesity included Hispanic race/ethnicity (p < 0.025), television watching (p < 0.001), and pediatric asthma (p < 0.005). Hispanic youth were 1.35 times more likely to be obese than white youth.

The descriptive characteristics of the restricted sample that did not include Hispanic subjects are shown in Table 3. This restricted sample included 25.3% of the children as obese, and 11.1% with asthma. Over 28% (28.5%) of the mothers of these children were obese. The results from the regression model for this sample (Table 4) showed that maternal standardized BMI was an additional significant predictor of pediatric BMI, with an OR of 2.2 (CI = 2.18 to 3.59) controlling for gender, age, and race/ethnicity. Asthma was not a significant independent predictor, but maternal standardized BMI interacted with pediatric asthma to predict pediatric BMI. Television remained a significant predictor, with an OR of 1.98 (CI = 1.69 to 3.30) for those

Table 1. Characteristics of the total sample (N = 5154), including the percentage who were obese (>85th BMI percentile) and average and standardized BMI (\pm SD)*

Characteristics	N	%	BMI	z-BMI	% Obese	OR	95% CI
Child BMI							
Non-obese	3683	73.6	17.7 ± 2.5	-0.28 ± 0.54			
Obese	1471	26.4	24.8 ± 5.1	2.06 ± 1.39			
Gender							
Female	2539	51.6	19.4 ± 4.6	0.36 ± 1.40	26.5	1.0	
Male	2615	48.4	19.7 ± 4.7	0.32 ± 1.27	26.2	1.02	0.85-1.22
Age							
6–7	1046	18.3	16.6 ± 2.8	0.32 ± 1.41	24.6	1.0	
8–10	1583	28.1	18.0 ± 3.6	0.28 ± 1.24	26.0	1.07	0.85-1.41
11–13	1372	27.2	20.3 ± 4.2	0.29 ± 1.13	26.5	1.08	0.87-1.41
14–16	1153	26.4	22.6 ± 5.1	0.47 ± 1.57	27.9	1.15	0.90-1.59
Race/ethnicity							
White	1369	69.6	19.4 ± 4.6	0.29 ± 1.32	24.7	1.0	
African American	1825	16.2	19.8 ± 5.0	0.42 ± 1.46	28.2	1.15	1.00-1.43
Hispanic	1960	14.2	20.0 ± 4.4	0.49 ± 1.26	32.6	1.35	1.12-1.85
Child asthma							
No asthma	4670	88.4	19.4 ± 4.3	0.28 ± 1.23	25.0	1.0	
Asthma	484	11.6	20.9 ± 6.4	0.78 ± 1.90	37.0	1.53	1.25-2.46
Hours of TV watched/day							
0–1	1716	39.0	19.1 ± 3.9	0.17 ± 1.11	21.8	1.0	
2–3	2212	42.4	19.5 ± 4.7	0.35 ± 1.40	26.6	1.23	1.08-1.56
≥ 4	1226	18.6	20.9 ± 5.5	0.67 ± 1.56	35.3	1.63	1.42-2.48

* All values are based on weighted estimates, except N; the OR is adjusted for gender, age, and race/ethnicity.

watching for 4 or more hours per day, versus those watching for 1 hour or less per day. The model had an *R* of 0.39 and accounted for 15.0% of the variation in children's BMI. As shown in Figure 1, pediatric asthma was associated with an increment of 1 z-score unit if the mother was obese (1.71 to 0.70), whereas pediatric asthma only showed an increment of 0.27 z-score unit if the mother was not obese (0.33 to 0.06). If maternal standardized BMI was not included, then television (p < 0.001) and asthma (p = 0.02) were significant predictors of pediatric BMI, accounting for 3.7% of the variance, similar to the amount of variance accounted for in the regression model for the full sample with the same predictors.

The logistic model was significant F(6,47) = 17.12, p < 0.001, accounting for 6.9% of the variability in pediatric obesity. Youth asthma (p < 0.025), television watching (p < 0.001), and maternal BMI (p < 0.001) were significant independent predictors of pediatric obesity. Youth with asthma were 1.65 times (CI = 1.2 to 3.02) more likely to become obese than youth without asthma, controlling for gender, age, and race/ethnicity.

Discussion

This study confirms clinical observations (5) that pediatric BMI is related to asthma during development, and provides the new observation that pediatric BMI is related to the interaction of maternal BMI and pediatric asthma. Asthma may influence higher BMI by increasing caloric intake or decreasing expenditure. Asthma and maternal BMI were significant independent predictors of pediatric obesity, but maternal BMI did not interact with youth asthma to predict pediatric obesity. Research suggests that caloric intake is similar in children with or without asthma, but children with asthma are less active than those without asthma (15,16). Resting energy expenditure, which makes the largest contribution to total energy expenditure, is greater, rather than suppressed, in children with asthma in comparison with children without asthma (16). Taken together, these data support the hypothesis that increases in obesity in children with asthma are related to decreases in physical activity. The relationship between obesity and television watching observed in this and other studies (10) is consistent with inactivity being a major influence for the development of obesity in children. Asthma did not interact with television watching to influence obesity, perhaps because the influence of television watching on pediatric obesity is also very strong for children and adolescents without asthma.

Maternal obesity is a traditional risk factor for pediatric obesity (17). Forty percent of children with obese mothers were obese, compared with 19.3% of children whose mothers were not obese, a doubling of the risk. Maternal BMI may predispose the child to a higher body weight based on a number of factors. Genetic influences (18) may predispose youth to develop obesity by having a lower threshold for

Table	2.	Regr	essior	n mode	l pred	icting	standard	ized
youth	BM	II by	age,	gender,	race,	asthm	a status,	and
televis	ion	watel	hing (N = 51	154)			

		SE	
Variable	$oldsymbol{eta}^*$	$oldsymbol{eta}^\dagger$	р
Intercept	-0.140	0.136	0.309
Child's age	0.016	0.012	0.179
Child's gender	-0.013	0.057	0.821
Race/ethnicity dummy codes			
R1 (African American $= 1$)	0.058	0.063	0.369
R2 (Hispanic $= 1$)	0.142	0.079	0.079
Asthma status	0.486	0.177	0.008
Television watching	0.107	0.017	0.0001
$R = 0.18, R^2 = 0.03; F(6, 47)$	= 15.94; p	p < 0.00	001 for
the complete model.			

* β = estimated population regression coefficient.

† SE β = SEs of the estimated regression coefficients.

positive energy balance, such that children with maternal obesity have to consume fewer calories or be more active than children with lean parents to maintain normal body weight (19). Gestational diabetes and metabolic dysregulation during prenatal development (20) may predispose children with obese mothers to develop obesity. Environmental factors related to having a mother with high BMI, such as modeling inactivity (21), development of food preferences for high-calorie foods (22), and use of food as a reward (23), may influence the development of obesity. Maternal BMI also interacted with pediatric asthma to predict the continuous variable of pediatric BMI. The combination of a genetic or environmental predisposition to develop obesity in combination with increased sedentary behavior and lowered energy expenditure in children with asthma could increase the susceptibility of these children to higher BMI values. However, asthma and maternal BMI had significant and independent effects when the dichotomous outcome, pediatric obesity, was considered. Thus, there are differences in predictors based on whether the continuous variable of pediatric BMI or the dichotomous variable of non-obese/ obese is the dependent variable. Pediatric obesity is independently related to asthma and maternal BMI, whereas pediatric BMI is related to maternal BMI and the interaction of maternal BMI and asthma. These differences may be due to maternal BMI and asthma interacting in relationship to pediatric BMI at lower levels than the 85th BMI percentile. Likewise, the failure to observe the interaction of maternal BMI and pediatric asthma may be due to reductions in power to detect the interaction effect when dichotomous rather than continuous variables are used as the dependent variables.

Characteristics	N	%	BMI	z-BMI	% Obese	OR	95% CI
Child BMI							
Non-obese	2203	74.7	17.6 ± 2.5	-0.29 ± 1.45			
Obese	806	25.3	24.8 ± 5.3	2.08 ± 1.45			
Gender							
Female	1472	51.7	19.3 ± 4.6	0.32 ± 1.41	24.7	1.0	
Male	1537	48.3	19.6 ± 4.7	0.29 ± 1.28	25.8	1.04	0.86-1.29
Age							
6–7	604	18.3	16.5 ± 2.7	0.29 ± 1.40	23.1	1.0	
8–10	948	28.9	17.9 ± 3.6	0.25 ± 1.24	24.5	1.06	0.80-1.47
11–13	780	26.9	20.1 ± 4.0	0.23 ± 1.08	26.9	1.08	0.83-1.53
14–16	677	26.0	22.6 ± 5.3	0.46 ± 1.64	27.6	1.12	0.92-1.76
Race/ethnicity							
White	1304	81.4	19.3 ± 4.6	0.28 ± 1.32	24.5	1.0	
African American	1705	18.6	19.9 ± 5.0	0.44 ± 1.45	28.6	1.13	1.03-1.48
Child asthma							
No asthma	2700	88.9	19.2 ± 4.3	0.24 ± 1.22	23.8	1.0	
Asthma	309	11.1	21.0 ± 6.8	0.84 ± 1.74	37.2	1.65	1.21-3.02
Maternal BMI							
Non-obese	1920	71.5	18.7 ± 3.8	0.09 ± 1.09	19.3	1.0	
Obese	1089	28.5	21.3 ± 5.9	0.85 ± 1.74	40.3	2.20	2.18-3.59
Hours of TV watched/day							
0-1	1017	40.4	18.8 ± 3.8	0.10 ± 1.07	19.1	1.0	
2–3	1274	42.4	19.4 ± 4.7	0.34 ± 1.44	26.7	1.44	1.23-1.96
≥4	781	17.2	21.0 ± 5.7	0.70 ± 1.59	36.2	1.98	1.69–3.30

Table 3. Characteristics of the sample for white and African American families (N = 3009) including the percentage who were obese (>85th BMI percentile) and average and standardized BMI (\pm SD)*

* All values are based on weighted estimates, except N; the OR is adjusted for gender, age, and race/ethnicity.

We have demonstrated previously that pediatric asthma and obesity were related in a predominately Hispanic sample (5), but methodological problems in the current study limited studying maternal BMI as a predictor of obesity in Hispanic youth. Future research is needed to examine the joint influence of maternal BMI and asthma on weight in Hispanic youth. Methodological issues also limited the study of physical activity and caloric intake as predictors of pediatric obesity. The only measurement of physical activity available was the single question that asked children, "How many times in the last week did you play or exercise enough to make you sweat or breathe hard?" Because asthma may alter the threshold for breathing hard, this definition may not be measuring similar degrees of physical activity for asthmatic and non-asthmatic youth. The common method for collection of nutritional information for children and adolescents in NHANES III was a 24-hour dietary recall, with the recall completed by the parents for children younger than 12. It is well known that self-reported dietary information underestimates caloric intake in comparison with objective measures of energy balance (24,25). Consistent with these data, previous analyses of the NHANES III database showed no differences in the caloric intake of obese and non-obese children (26). Dietary data were not included to reduce the possibility of reporting a relationship based on inaccurate self-reports.

If asthma is under proper control, children with asthma experience few physical limitations (1). Children with asthma who experience respiratory symptoms during exercise have poor control of their asthma, and even mild asthma can be very restrictive if it is not properly controlled with medications and environmental alterations. Children with asthma and active lifestyles can have similar exercise tolerance to control children without asthma. Only the children with mild asthma and a sedentary lifestyle had poor physical fitness (27). Likewise, when children with and without asthma were matched on body

Table 4. Regression model predicting pediatric BMI, in	icluding the predictors of maternal standardized BMI and
the interaction of maternal standardized BMI*/youth as	sthma status ($N = 3009$)

Variable	$oldsymbol{eta}^*$	SE β †	р
Intercept	-0.212	0.147	0.155
Age	0.009	0.012	0.489
Gender	0.030	0.052	0.565
Race/ethnicity dummy codes			
African American $= 1$, Caucasian $= 0$	-0.093	0.069	0.181
Asthma status	0.178	0.101	0.083
Television watching	0.117	0.018	< 0.001
Maternal standardized BMI	0.283	0.029	< 0.001
Maternal BMI*/asthma status	0.421	0.195	0.036
$R = 0.39, R^2 = 0.15; F(7, 46) = 21.21; p < 0.000$	1 for the complete model.		

* β = estimated population regression coefficients.

† SE β = SEs of the estimated regression coefficients.

size and habitual physical activity, they did not differ in fitness or pulmonary function (28).

Obesity may influence bronchial hyperactivity in children without asthma (29). For example, wheezing was related to increases in BMI or triceps skinfolds in NHANES II (30). A significantly greater frequency and degree of bronchospasm of the smaller airways occur in obese children without asthma compared with their nonobese counterparts, which is related to the amount of subcutaneous fat (29). Changes in lung volume, as with obesity, can increase airway responsiveness in subjects without asthma, and chest wall loading associated with obesity may mechanically alter airways, increasing airway reactivity (31,32). Research suggests that adult BMI is prospectively related to onset of asthma (33).



Figure 1. Children's mean standardized BMI values for asthmatic and non-asthmatic children with obese or non-obese mothers.

Research on the causal relationship between obesity and asthma in youth is needed, including prospective research to evaluate whether the onset of obesity precedes asthma in pediatric samples, and whether weight loss improves pulmonary functioning in pediatric obese asthma patients.

There are several clinical implications to the current results. Pediatricians should be aware of the relationship between asthma and obesity, and initiate preventive dietary and activity measures if the child or adolescent with asthma is in a family with an obese mother or if the child with asthma begins to show large increases in BMI for his/her age and gender. Children and adolescents with comorbid asthma and obesity should be provided treatments for their obesity, which include decreasing inactivity and increasing vigorous physical activity to decrease body fat and improve exercise tolerance. Comorbidity of these prevalent pediatric conditions suggests that attention to reducing sedentary behaviors and increasing physical activity may be important in the prevention of obesity and treatment of asthma.

Acknowledgments

This research was supported in part by Grants HD 34284 and HD 25997 (to L.H.E.).

References

- Cypcar D, Lemanski RF. Asthma and exercise. *Clin Chest* Med. 1994;15:351–68.
- Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults. *JAMA*. 1994;272:205–11.
- Schaubel D, Johansen H, Mao Y, Dutta M, Manfreda J. Risk of preschool asthma: incidence, hospitalization, recurrence, and readmission probability. *J Asthma*. 1998;33: 97–103.

- 4. Woolcock AJ, Peat JK. Evidence for the increase in asthma worldwide. *Ciba Found Symp.* 1997;206:122–34.
- Gennuso J, Epstein LH, Paluch RA, Cerny F. The relationship between asthma and obesity in urban minority children and adolescents. *Arch Pediatr Adolesc Med.* 1998; 152:1197–200.
- Luder E, Melnick TA, DiMaio M. Association of being overweight with greater asthma symptoms in inner city black and Hispanic children. *J Pediatr*. 1998;132:699–703.
- Unger R, Kreeger L, Christofell KK. Childhood obesity: medical and familial correlates and age of onset. *Clin Pediatr*. 1990;29:368–73.
- Kawabori I, Pierson WE, Conquest LL, Bierman CW. Incidence of exercise-induced asthma in children. J Allergy Clin Immunol. 1998;58:447–55.
- Kyle JM, Walker RB, Hanshaw SL, Leaman JR, Frobase JK. Exercise-induced bronchospasm in the young athlete: guidelines for routine screening and initial management. *Med Sci Sports Exerc.* 1992;24:856–9.
- Andersen RE, Crespo CJ, Bartlett SJ, Cheskin LJ, Pratt M. Relationship of physical activity and television watching with body weight and level of fatness among children: results from the Third National Health and Nutrition Examination Survey. JAMA. 1998;279:938–42.
- Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med.* 1997;337:869–73.
- Najjar MF, Rowland M. Anthropometric Reference Data and Prevalence of Overweight, United States, 1976–1980. Washington, DC: US Government Printing Office; 1987.
- 13. Westat. A User's Guide to WesVar PC. Rockville, MD: Westat, Inc.; 1996.
- 14. **Sobal J, Stunkard AJ.** Socioeconomic status and obesity: a review of the literature. *Psychol Bull.* 1989;105:260–75.
- Goldey DH, Mansmann HC, Rasmussen AI. Zinc status of asthmatic, prednisone-treated asthmatic, and non-asthmatic children. J Am Diet Assoc. 1984;84:157–63.
- Zeitlin SR, Bond S, Wootton S, Gregson RK, Radford M. Increased resting energy expenditure in childhood asthma: does this contribute toward growth failure? *Arch Dis Child*. 1992;67:1366–9.
- Garn SM, Clark DC. Trends in fatness and the origins of obesity. *Pediatrics*. 1976;57:443–56.
- Grilo CM, Pogue-Geile MF. The nature of environmental influences on weight and obesity: a behavior genetic analysis. *Psychol Bull.* 1991;110:520–37.
- Griffiths M, Payne PR. Energy expenditure in small children of obese and non-obese parents. *Nature*. 1976;260:698–700.

- Whitaker RC, Dietz WH. Role of the prenatal environment in the development of obesity. *J Pediatr.* 1998;32:768–76.
- Moore LL, Lombardi DA, White MJ, Campbell JL, Oliveria SA, Ellison RC. Influence of parents' physical activity levels on activity levels of young children. *J Pediatr.* 1991; 118:215–9.
- Fisher JO, Birch LL. Fat preferences and fat consumption of 3- to 5-year-old children are related to parental adiposity. *J Am Diet Assoc.* 1995;95:759–64.
- Birch LL, Marlin DW, Rotter J. Eating as the "means" activity in a contingency: effects on young children's food preference. *Child Dev.* 1984;55:431–9.
- Bandini LG, Schoeller DA, Cyr HN, Dietz WH. Validity of reported energy intake in obese and non-obese adolescents. *Am J Clin Nutr.* 1990;52:421–5.
- Lichtman SW, Pisarska K, Berman ER, et al. Discrepancy between self-reported and actual caloric intake in obese subjects. *N Engl J Med.* 1992;327:1893–8.
- Strauss RS. Comparison of serum concentrations of αtocopherol and β-carotene in a cross-sectional sample of obese and non-obese children (NHANES III). *J Pediatr.* 1999;134: 160–5.
- Fink G, Kaye C, Blau H, Spitzer SA. Assessment of exercise capacity in asthmatic children with various degrees of activity. *Pediatr Pulmonol.* 1993;15:41–3.
- Santuz P, Baraldi E, Filippone M, Zacchello F. Exercise performance in children with asthma: is it different from that of healthy controls? *Eur Respir J.* 1997;10:1254–60.
- 29. Kaplan TA, Montana E. Exercise-induced bronchospasm in nonasthmatic obese children. *Clin Pediatr.* 1993;32:220–5.
- Schwartz J, Gold D, Dockery DW, Weiss ST, Speiezer FE. Predictors of asthma and persistent wheeze in a national sample of children in the United States: association with social class, perinatal events, and race. *Am Rev Respir Dis.* 1990; 142:555–62.
- Ding DJ, Martin JG, Macklem PT. Effects of lung volume on maximal methacholine-induced bronchoconstriction in normal humans. *J Appl Physiol*. 1987;62:1324–30.
- Regnard J, Baudrillard P, Salah B, Xuan ATD, Cabanes L, Lockhart A. Inflation of antishock trousers increases bronchial response to methacholine in healthy subjects. *J Appl Physiol.* 1990;68:1528–33.
- Camargo CA Jr, Weiss ST, Zhang S, Willett WC, Speizer FE. Prospective study of body mass index, weight change, and risk of adult-onset asthma in women. *Arch Intern Med.* 1999; 159:2582–8.