Published in final edited form as:

J Asthma. 2019 April ; 56(4): 388–394. doi:10.1080/02770903.2018.1463377.

Pediatric metabolic outcome comparisons based on a spectrum of obesity and asthmatic symptoms

Ashley D. Perdue, BS^a, Lesley A. Cottrell, PhD^b, Christa L. Lilly, PhD^c, William A. Gower, MD^d, Brian A. Ely, MD^b, Brad Foringer^e, Melvin L. Wright, MD^b, and William A. Neal, MD^b

^aInstitute for Community and Rural Health, Morgantown, WV, USA

^bDepartment of Pediatrics, West Virginia University School of Medicine, Morgantown, WV, USA

^cDepartment of Biostatistics, West Virginia University School of Public Health, Morgantown, WV, USA

^dDepartment of Pediatrics, Wake Forest School of Medicine, Winston-Salem, NC, USA

^eDivision of Respiratory Therapy, West Virginia Hospital Association, Morgantown, WV, USA

Abstract

Rationale: Asthma and obesity are 2 of the most prevalent public health issues for children in the U.S. Trajectories of both have roughly paralleled one another over the past several decades causing many to explore their connection to one another and to other associated health issues such as diabetes and dyslipidemia. Earlier models have commonly designated obesity as the central hub of these associations; however, more recent models have argued connections between pediatric asthma and other obesity-related metabolic conditions regardless of children's obesity risk.

Objectives: To examine the relationships between asthma, obesity, and abnormal metabolic indices.

Methods: We conducted a cross-sectional study of 179 children ages 7 to 12 years recruited from a rural, Appalachian region. Our model controlled for children's smoke exposure, body mass index percentile, and gender to examine the association between children's asthma (based on pulmonary function tests, medical history, medications, and parent report of severity), lipids (fasting lipid profile), and measures of altered glucose metabolism (glycosylated hemoglobin and homeostatic model assessment 2-insulin resistance).

Results: Our findings revealed a statistically significant model for low density lipids, high density lipids, log triglyceride, and homeostatic model assessment 2-insulin resistance; however, Asthma had a significant effect for the mean triglycerides. We also found an asthma–obesity interaction effect on children's glycosylated hemoglobin with asthmatic obese children revealing significantly higher glycosylated hemoglobin values than non-asthmatic obese children.

CONTACT Ashley D. Perdue adperdue@mix.wvu.edu Institute for Community and Rural, Health PO Box 9009, Morgantown, WV 26506-9009, USA.

Declaration of interest

The authors report no conflicts of interest.

Conclusions: Our findings support a connection between asthma and children's glycosylated hemoglobin values; however, this association remains entwined with obesity factors.

Keywords

Childhood obesity; hemoglobin A1c (HbA1C); lipid; metabolic outcomes; pediatric asthma

Introduction

Asthma and obesity have increased in parallel as 2 of the most prevalent chronic health conditions of children in the United States [1]. The nature and magnitude of the association between them has been explored over the past decade with questions of causality and possible shared etiologies being of great interest [2–4]. More recently, researchers have explored how asthma and obesity may interact and subsequently impact other health concerns including diabetes or metabolic syndrome hypothesizing that asthma and obesity may have additive effects in the development of these outcomes [5–7].

In 2011, we published the results of a cross-sectional study of nearly 18,000 children (4–12 years of age). Our findings showed that children with parent-reported asthma had higher mean triglyceride levels and a greater proportion of the prediabetes marker, acanthosis nigricans (AN), than non-asthmatic children after controlling for socioeconomic status, gender, and smoke exposure. Furthermore, we found that the elevated triglyceride levels and AN prevalence among asthmatic children were present regardless of the body mass index (BMI) percentile [8].

Forno and colleagues showed an additive impact of metabolic syndrome (MS) and asthma, as well as obesity and insulin resistance, on lung function [9]. Other studies have shown that insulin resistance may account for much of the obesity-associated asthma risk [10–14]. The connection between asthma and MS may be, at least, in part due to alterations in epithelial cell function [15].

We designed the present study to further explore connections between asthma and obesity in childhood using a clinic-based, cross-sectional design to directly address the limitations from earlier studies. We sought to better define the presence of asthma using pulmonary function testing, and more accurately reflecting glucose metabolism using glycosylated hemoglobin (HgbA1c) and homeostatic model of insulin resistance (HOMA-IR). Bio specimen assessments were used to confirm environmental tobacco smoke exposure as well. Our hypothesis was that we would confirm, among a sample enriched for obesity and asthma, the association between select lipid and glucose metabolic indices and pediatric asthma.

Methods

Subject eligibility and recruitment

Children 7–12 years of age and at least one legal guardian were eligible for this study, regardless of the child's body composition, gender, or asthma history. Families were recruited using study flyers distributed throughout the rural, Appalachian setting of West

Virginia (WV), Pennsylvania, Ohio, and Virginia. Families were also recruited from general pediatric clinics and primary care providers throughout WV using the same flyers and study materials. One hundred ninety-four children and their families were approached to participate in the study; 183 were enrolled. Four children dropped out of the study due to not being able to schedule the clinic visit for the study.

Measures

Demographic and environmental factors known to be related to pediatric asthma, obesity, and other study variables were assessed immediately upon consent. Factors including children's age, gender, socioeconomic status, and adult respondent relationship to the child participant were collected by means of parental survey.

Children's asthma status was based on 3 points of information that were collected during the study clinic visit. Participants' current medications were recorded during the history and physical segment of the visit. Families were specifically prompted about use of asthma medications in the past 3 months. Participants were also asked if they had previously been diagnosed with asthma or had been diagnosed with other conditions such as allergic rhinitis, atopic dermatitis, allergic conjunctivitis. Finally, each participant received a pulmonary function test (PFT) implemented according to American Thoracic Society (ATS) guidelines [16] following withdrawal of any asthma medications for at least 24 hours prior to the assessment. Medications were reviewed and coded by a physician member of the study team William A. Gower (WAG) and spirometry results were reviewed by the principal investigator (PI) and respiratory therapist member of the study team (Lesley A. Cottrell (LAC) and Brad Foringer (BF)). Children were categorized in the "asthmatic" group if they had spirometry results showing reversible lower airways obstruction that was reversible with bronchodilation. Reversibility of an airway obstruction was determined based on the percent of change of FEV1, FEF 25–75, and FVC/FEV1 after the administration of a bronchodilator. ATS standards including: 1) inhalation maximally to total lung capacity with blast of air out; 2) no cough or hesitation during first second of maximal exhalation; 3) continued exhalation forcefully for at least 6 seconds and until a plateau is reached; 4) minimum of 3 acceptable maneuvers; 5) largest and second largest FVC and FEV1 values should be repeatable within 150 mL; and 6) largest FVC and FEV1 from acceptable efforts reported were followed.

Figure 1 illustrates how children enrolled in this study were classified into the asthmatic groups based on their histories, current medications, and spirometry results. Children who had a previous doctor's diagnosis of asthma and/or were on asthma medications (short-acting beta agonist, leukotriene receptor antagonist, inhaled corticosteroid with or without a long-acting beta-agonist), but had non-diagnostic or equivocal spirometry results were included in a "probable asthmatic" group. Children without a previous diagnosis of asthma, no use of asthma medications, and/or spirometry results that did not illustrate airways obstruction were classified in the "non-asthmatic" group.

Children's obesity risk was determined based on height (m) and weight (kg) assessments taken at the beginning of the study clinic visit using SECA Road Rod stadiometers and the SECA 840 digital scales (SECA Corp, Hanover, MD). Children were asked to remove their shoes, extra clothing (e.g., coats), and hats. These assessments were then used to calculate

children's BMI using the following equation: $BMI = \text{weight (kg)} / \text{height (m)}^2$. BMI percentile (BMI%) based on appropriate age- and gender-based references were then calculated [17]. Classifications were based on the BMI percentile and were determined to be defined as: underweight = BMI% from 0 to 4.9th%, normal weight = BMI% from 5.0 to 84.9th%, overweight = BMI% from 85.0 to 94.9th%, and obese = BMI% from 95.0 to 100th%. Underweight was combined with normal weight for most BMI category analyses.

Fasting lipid analyses were conducted by Lab Corp [18] based on 15 cc of blood collected at the beginning of the study clinic visit and transported to the local laboratory in purple top Ethylenediaminetetraacetic acid (EDTA) tubes. Lipid analysis panels included total cholesterol (TC; mg/dL), low density lipids (LDL; mg/dL), high-density lipoproteins (HDL; mg/dL), and triglycerides (TRIG; mg/dL).

Glucose metabolism indices analyzed by Lab Corp. from the same serum collection at the time of the study clinic visit included HbA_{1c} (%), insulin (INS; uIU/mL), and glucose (GLU; mg/dL). These values were then used by the research team to calculate HOMA2-IR using the computer model [19].

Smoke exposure was collected in 2 ways for this study. First, parents were asked whether their child had been exposed to smoke in the past 30 days. Parents were also asked if they smoked, if they smoked outside/inside the home, and the frequency of their smoking pattern, if applicable. These later measures were not used in this study given that we were attempting to control for any asthma, lipid, or metabolic outcome effects that might come from smoke exposure. For this purpose, we used the second form of data collection including a urine specimen from each child. Each urine sample was used to assess the cotinine level. These continuous levels depict any circulating nicotine and cotinine levels within the child around the time of the clinic visit and reflect exposure to environmental tobacco smoke within the past 30 days. Current cotinine levels at the time of the clinic visit were controlled for when examining the associations of study variables in this study.

Procedures

All eligible and interested children and their families were appropriately consented to participate in this study using materials and procedures approved by the West Virginia University Institutional Review Board (IRB). Sample size calculations with power set at .80, alpha at .05, and a medium effect size indicated that 40 individuals in each group would be needed to conduct the proposed analyses. Consent materials and surveys for parent and child report were mailed to participating families and returned during a clinic visit that was scheduled at a time most suitable for each family. Prior to the clinic visit, children had fasted for at least 12 hours from food and any medications, if deemed appropriate to do so by their primary care physician. Following consent/assent confirmation, child height and weight was measured. Subsequently, a project team member collected a peripheral venous blood sample. Up to 15 cc of whole blood was collected from each participant and processed for transport in lavender top tubes to Lab Corp for serum separation and analyses. In the remaining portion of the visit, we completed a history and physical test, conducted a complete skin allergy test, and spirometry with or without exercise challenge and post-bronchodilator

assessment. The clinic visit took an average of 2 hours to complete. Families received \$100 for their participation in the study.

Statistical analyses

All analyses were conducted using SPSS (IBM SPSS Statistics for Windows, Version 23.0. Armonk NY: IBM Corp.). We first characterized the sample using descriptive statistics. In initial analyses, we used a composite definition to categorize participants into 3 categories: no asthma, probable asthma, and definite asthma. A series of sensitivity analyses was conducted to assess the similarities or dissimilarities between the groups. In general, the probable asthma category was more similar to the no asthma than the definite asthma group, so final models reported here include the probable asthmatics into the no asthma group.

We used the asthma diagnosis variable including 3 levels: no, probable, and yes for the univariate analyses in this study. The probable asthmatics were later combined with the “no” group for multivariate analyses. Estimated marginal means and estimated marginal mean differences and their 95% confidence intervals are presented for significant models. Two-sided statistical tests were conducted where applicable.

Finally, a series of linear regression models were used to describe the relationship between asthma and the lipid and metabolic outcomes after controlling for smoke exposure (i.e., cotinine level), sex, and BMI percentile (as a continuous variable). For significant omnibus models, standardized and unstandardized beta weights with 95% confidence intervals are presented of the predictors. All tests used a significance level of $p < 0.05$ and assumptions were tested appropriate to the type of analysis. Sensitivity analysis was conducted on the differences and associations with grouping probably asthmatics on outcomes using a series of Mann–Whitney, *t*-Tests, and logistic regression analyses, as appropriate for the variable type.

Results

Sample characteristics

One hundred seventy-nine children were enrolled into this study. Table 1 shows general information about the total study sample and those of each asthma group. The average age for participants was 9.4 years (SD = 1.7). More than 85.1% ($n = 149$) were Caucasian; slightly more than half were male (56.3%, $n = 98$). These characteristics roughly reflect the composition of the state population for ethnicity/race and gender. Nearly 20% of the children’s mothers had completed a high school diploma, some high school coursework, or less ($n = 33$). Over 26% of the children’s history, current medication, and/or PFT result indicated asthma ($n = 46$). Within this sample of children, 17.1% ($n = 30$) of the children were overweight (85th–94.9th BMI%) and an additional 27.4% ($n = 48$) were obese (95th BMI%). The 95% confidence interval was $25.7\% \pm 6.4\%$.

Pediatric asthma, gender, and obesity

Children’s BMI% categorization and asthma status were not significantly related (Pearson chi-square $p = 0.65$). Of those who were under or normal weight, 24% ($N = 23$) were

asthmatic, as compared to 26.7% ($N = 8$) of overweight and 31.3% ($N = 15$) of obese children.

There was also no relationship between asthma and gender on BMI percentile ($p = 0.07$). Although there was a significant interaction effect for gender and asthma on BMI percentile ($p = 0.02$) with male asthmatics having slightly higher BMI percentiles, (mean = 74.63, SD = 25.63) than male non-asthmatics (mean = 61.44, SD = 32.90, $p = 0.04$), no differences were found among female participants (asthmatics: $N = 18$, mean = 62.55, SD = 36.14) and non-asthmatics ($N = 57$, mean = 73.46, SD = 25.61, $p = 0.25$).

Differences in pediatric obesity and asthma on lipids and measures of glucose metabolism

Children's fasting serum lipid levels, metabolic indices (HgbA1c, and HOMA2-IR) were examined based on their BMI category. We found no differences among weight groups or asthma on TC or HbA1c%. We did demonstrate support for main and interaction effects of asthma and BMI% categorization on HDL $p < 0.001$. Specifically, the main effect of BMI category was significant ($p < 0.001$); there was no main effect of asthma.

Relationship between asthma, lipid, and measures of glucose metabolism controlling for BMI, gender, and smoking

We subsequently conducted predictive models for lipid and glucose metabolism with asthma diagnosis as the predictor controlling for gender, children's smoke exposure, and BMI percentile. Our findings revealed a significant model for LDL, HDL, log TRIG, and HOMA2-IR (Table 2); a significant main effect for asthma was found for log-TRIG. We also found a significant interaction effect for asthma-obesity on children's HbA1c ($p = 0.032$) with asthmatic, obese children revealing significantly higher HbA1c values than non-asthmatic, obese children.

Discussion

The findings of this study show interesting relationships between asthma and lipid/metabolic indices that need to be further explored. In the present study, a gender difference related to asthma was not found. Obesity was found to be positively associated with log TRIG, LDL, HOMA2, while an inverse association was found with HDL. Furthermore, obese asthmatic children had significantly higher HbA1c values than non-asthmatic, obese children.

While this study did not find a clear association between obesity and asthma, recent research suggests that both are indeed related. A systematic review found that out of 35 studies 27 showed a positive association between overweight/obese children and asthmatic symptoms. Of the 4 longitudinal studies included, 3 reported that higher BMI during early childhood was a predictor for future increased incidence of asthma diagnosis or symptoms [20]. A meta-analysis by Chen and colleagues found that in overweight children asthma incidence increased by 20%. For obese children that risk was found to be twofold. This study also demonstrated a dose-response relationship between incident asthma and body weight which boys were found to follow more than girls [21]. The lack of an association between obesity and asthma in this study may have been due to a varied sample of asthmatics (e.g., allergic asthma). Combining the different types of asthma may dilute associations with obesity found

elsewhere in literature. Our study also used a stricter categorization for asthma status compared to many other epidemiologic studies. In this study, for a child to be placed into the asthma category they were required to use asthma medication and/or have a positive PFT test.

Different metabolic factors have been found to be associated with children who are both obese and asthmatic. Adolescent males who are obese and asthmatic have been found to have a significantly higher prevalence of metabolic syndrome compared to their obese non-asthmatic counterparts [22]. Another study reports that in children aged 10–15 years, LDL levels were significantly higher in male obese asthmatics compared to obese non-asthmatics [23]. These studies suggest that when asthma and obesity are coupled in patients their metabolic outcomes differ from those who are obese and not asthmatic. A study by Al-Shawwa and colleagues found that hypocholesteremia is associated with an increased frequency of asthma regardless of obesity [24]. A recent study also found that serum triglycerides and low HDL were associated with wheezing, a common asthma symptom, even after adjusting for adiposity [25].

In this study, children who were both obese and asthmatic were found to have significantly higher HbA1c values than children who were non-asthmatic and obese. A similar finding was observed by Black and colleagues who found that among youth with type 1 diabetes, those with asthma had significantly higher mean HbA1c levels than those without asthma after adjusting for age, gender, race/ethnicity, and BMI [26]. Both of the following findings suggest that asthma may play a role in glycemic control.

Limitations

Although the present study was designed to provide objective, clinical assessments rather than self-reported outcomes to examine a potential association between asthma and altered lipid and glucose metabolism, several limitations existed that could have impacted our findings. First, the sample for this study was relatively small leading to concerns about the generalizability of the findings and power needed to elicit associations if present. The size of the sample may have limited the extent to which differences based on gender and other factors were detected. Second, the focus on pre-pubertal children and lack of a pubertal measure replicated our initial approach but may have limited our ability to examine the contribution that puberty may have in the asthma and metabolic outcomes association, if present. Finally, the lack of a longitudinal design continues to limit our ability to assess the sequential relationship between asthma, obesity, and metabolic developments.

Conclusions

This study continues to highlight the importance of the association between asthma and obesity in regard to metabolic indices. This leads us to believe that when asthma is coupled with obesity it leads to other metabolic comorbidities. A possible mechanism could be that children with asthma have decreased physical activities leading to abnormal metabolic indices. Due to this association, we feel that future studies should further investigate asthma and associated metabolic outcomes independent of obesity. While some studies have found abnormal metabolic indices related to asthma regardless of whether a child is obese, the

research is limited at this time. It is also unclear if metabolic abnormalities increase the likelihood of asthma, or if asthma causes the metabolic abnormalities itself. Further investigation could provide additional guidance on appropriate measures to use when assessing developmentally changing youth who have asthma and potential metabolic risk, with or without obesity. For instance, in this study, a significant association was noted with HbA1c but not with other measures of metabolic function. Understanding differences in accuracy across measures will be important for future work in this area.

Acknowledgements

This study was supported through departmental funding. We wish to thank the families who participated in this study. We would also like to thank the following individuals for their time, expertise, and support in the process: Lennie Samsell, Samantha Shawley, Greg Hawkins, and Brian Ansell.

References

1. Cleave JV, Gortmaker SL, Perrin JM. Dynamics of obesity and chronic health conditions among children and youth. *JAMA* 2010;303(7):623–30. doi:10.1001/jama.2010.104. PMID:. [PubMed: 20159870]
2. Ali Z, Ulrik CS. Obesity and asthma: a coincidence or a causal relationship? A systematic review. *Respir Med.* 2013;107(9):1287–300. doi:10.1016/j.rmed.2013.03.019. PMID:. [PubMed: 23642708]
3. Egan KB, Ettinger AS, Bracken MB. Childhood body mass index and subsequent physician-diagnosed asthma: a systematic review and meta-analysis of prospective cohort studies. *BMC Pediatr.* 2012;13(1):1.
4. Papoutsakis C, Priftis KN, Drakouli M, Prifti S, Konstantaki E, Chondronikola M, Antonogeorgos G, Matziou V. Childhood overweight/obesity and asthma: is there a link? A systematic review of recent epidemiologic evidence. *J Acad Nutr Diet.* 2013;113(1):77–105. doi:10.1016/j.jand.2012.08.025. PMID:. [PubMed: 23260726]
5. Brumpton BM, Camargo CA, Romundstad PR, Langhammer A, Chen Y, Mai XM. Metabolic syndrome and incidence of asthma in adults. The HUNT study. *Eur Resp J.* 2013;42(6):1495–502. doi:10.1183/09031936.00046013.
6. Van Huisstede A, Cabezas MC, Birnie E, van de Geijn G-JM, Rudolphus A, Mannaerts G, Njo TL, Hiemstra PS, Braunstahl G-J. Systemic inflammation and lung function impairment in morbidly obese subjects with the metabolic syndrome. *J Obes.* 2013;2013:131349 PMID:. [PubMed: 23509614]
7. Lorente-Cebrian S, Costa AG, Navas-Carretero S, Zabala M, Martinez JA, Moreno-Aliaga MJ. Role of omega-3 fatty acids in obesity, metabolic syndrome, and cardiovascular diseases: a review of the evidence. *J Physiol Biochem.* 2013;69(3):633–51. doi:10.1007/s13105-013-0265-4. PMID:. [PubMed: 23794360]
8. Cottrell L, Neal WA, Ice C, Perez MK, Piedimonte G. Metabolic abnormalities in children with asthma. *Am J Respir Crit Care Med.* 2011;183:441–8. doi:10.1164/rccm.201004-0603OC. PMID:. [PubMed: 20851922]
9. Forno E, Han YY, Muzumdar RH, Celedon JC. Insulin resistance, metabolic syndrome, and lung function in US adolescents with and without asthma. *J Allergy Clin Immunol.* 2015;136(2):304–11. doi:10.1016/j.jaci.2015.01.010. PMID:. [PubMed: 25748066]
10. Agrawal A, Mabalirajan U, Ahmad T, Ghosh B. Emerging interface between metabolic syndrome and asthma. *Am J Respir Cell Mol Biol.* 2011;44:270–5. doi:10.1165/rcmb.2010-0141TR. PMID:. [PubMed: 20656947]
11. Thuesen BH, Husemoen LL, Hersoug LG, Pisinger C, Linneberg A. Insulin resistance as a predictor of incident asthma-like symptoms in adults. *Clin Exp Allergy.* 2009;39:700–7. doi:10.1111/j.1365-2222.2008.03197.x. PMID:. [PubMed: 19260867]

12. Appleton SL, Adams RJ, Wilson DH, Taylor AW, Ruffin RE. Central obesity is associated with nonatopic but not atopic asthma in a representative population sample. *J Allergy Clin Immunol*. 2006;118:1284–91. doi:10.1016/j.jaci.2006.08.011. PMID:. [PubMed: 17157658]
13. Terzano C, Morano S, Ceccarelli D, Conti V, Paone G, Petroianni A, Graziani E, Carnovale A, Fallarino M, Gatti A, et al. Effect of insulin on airway responsiveness in patients with type 2 diabetes mellitus: a cohort study. *J Asthma*. 2009;46:703–07. doi:10.1080/02770900903056203. PMID:. [PubMed: 19728209]
14. Cottrell L, Neal WA, Ice C, Perez MK, Pledimonte G. Metabolic abnormalities in children with asthma. *Am J respire Crit Care Med*. 2011;183:441–8. doi:10.1164/rccm.201004-06030C.
15. Baffi CW, Wood L, Winnica D, Strollo PJ, Gladwin MT, Que LG, Holguin F. Metabolic syndrome and the lung. *CHEST* 2016;149:1525–34. doi:10.1016/j.chest.2015.12.034. PMID:. [PubMed: 26836925]
16. American Thoracic Society. Statements, Guidelines, & Reports [Internet]. American Thoracic Society; [cited 2016 Aug 15] Available from: <https://www.thoracic.org/statements/>.
17. Centers for Disease Control. Child and teen BMI [Internet]. Centers for Disease Control, CDC; [cited 2016 Aug 15]. Available from: http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html.
18. LabCorp. Online Company Citation [Internet] [cited 2016 Aug 18] Available from: www.labcorp.com.
19. HOMA Calculator. Headington, Oxford: University of Oxford [Internet] [cited 2016 Aug 10] Available from: www.dtu.ox.ac.uk/homacalculator.
20. Liu P, Kieckhefer GM, Gau B. A systematic review of the association between obesity and asthma in children. *J Adv. Nurs* 2013;69(7):1446–65. doi:10.1111/jan.12129. PMID:. [PubMed: 23560878]
21. Chen YC, Dong GH, Lin KC, Lee YL. Gender difference of childhood overweight and obesity in predicting the risk of incident asthma: a systematic review and meta-analysis. *Obes Rev*. 2013;14(3):222–31. doi:10.1111/j.1467-789X.2012.01055.x. PMID:. [PubMed: 23145849]
22. Del-Rio-Navarro BE, Castro-Rodriguez JA, Garibay Nieto N, Berber A, Toussaint G, Sienra-Monge JJ, Romieu I. Higher metabolic syndrome in obese asthmatic compared to obese nonasthmatic adolescent males. *J Asthma* 2010;47(5):501–6. doi:10.3109/02770901003702808. PMID:. [PubMed: 20560825]
23. Yang CC, Kuan YT, Ching HT, Ming WS, Pei CW, Chien HC, Yungling LL. Lipid profiles in children with and without asthma: interaction of asthma and obesity on hyperlipidemia. *Diabetes Metab Syndr*. 2013;7(1):20–25. doi:10.1016/j.dsx.2013.02.026. PMID:. [PubMed: 23517791]
24. Al-Shawwa B, Al-Huniti N, Titus G, Abu-Hasan M. Hypercholesterolemia is a potential risk factor for asthma. *J Asthma* 2006;43(3):231–3. doi:10.1080/02770900600567056. PMID:. [PubMed: 16754527]
25. Fenger RV, Gonzalez-Quintela A, Linneberg A, Husemoen LLN, Thuesen BH, Aadahl M, Vidal C, Skaaby T, Sainz JC, Calvo E. The relationship of serum triglycerides, serum HDL, and obesity to the risk of wheezing in 85,555 adults. *Respir Med*. 2013;107:816–24. doi:10.1016/j.rmed.2013.02.001. PMID:. [PubMed: 23465506]
26. Black MH, Anderson A, Bell RA, Dabelea D, Pihoker C, Saydah S, Seid M, Standiford DA, Waitzfelder B, Macrovinia SM, et al. Prevalence of asthma and its association with glycemic control among youth with diabetes. *Pediatrics* 2011;128(4):e839–47. doi:10.1542/peds.2010-3636. PMID:. [PubMed: 21949144]

Figure 1.

Sample asthma group classification based on history, current medications, and spirometry results.

*The three participants were placed in the probable/no group due to ATS standard guidelines.

Table 1.

Demographic and descriptive statistics.

Variable	Unit	Total cohort (n = 174)	Non-asthmatic (n = 101)	Possible asthmatic (n = 27)	Asthmatic (n = 46)	p-value *
Male (n = 98)	n (%)	98 (56)	53(53)	17(63)	28 (60.9)	0.518
White	n (%)	148 (85.5)	82 (82)	24 (88.9)	42 (91.3)	0.454
Age		9.4 (1.7)	9.4 (1.8)	9.6 (1.6)	9.3 (1.8)	0.775
ETS exposed	n (%)	53 (31)	24 (23.8)	18 (39.1)	11 (40.7)	0.077
Maternal HS or less	n (%)	32 (19.0)	12 (12.5)	7 (25.9)	13 (28.9)	0.042
BMI category *						
Underweight	n (%)	5 (2.9)	3 (3)	1 (3.7)	1 (2.2)	(merged with normal weight)
Normal weight		91 (52.3)	55 (54.4)	14 (51.9)	22 (47.8)	0.751
Overweight		30 (17.2)	21 (20.8)	1 (3.7)	8 (17.4)	0.564
Obese		48 (27.6)	22 (21.8)	11 (40.7)	15 (32.6)	0.623
BMI%	%	67.7 (30.2)	66.9 (29.6)	67.4 (33.4)	69.9 (30.4)	0.854
Hgb A1c	%	5.4 (0.3)	5.4 (0.4)	5.4 (0.3)	5.3 (0.2)	0.159
HOMA2-IR		1.1 (0.9)	1.1 (0.6)	1.1 (0.7)	1.2 (0.6)	0.769
Glucose	mg/dL	82.7 (12.1)	85.2 (8.5)	71 (21.0)	83.5 (8.1)	<0.001
Insulin	uIU/mL	9.1 (7.9)	9.2 (9.0)	8.5 (6.2)	9.4 (6.0)	0.905
Total cholesterol	mg/dL	158.1 (25.1)	158.1 (26.5)	156.2 (26.7)	158.8 (21.3)	0.921
LDL	mg/dL	87.0 (21.2)	87.1 (22.1)	84.6 (22.1)	87.6 (18.8)	0.846
HDL	mg/dL	55.2 (13.4)	55.2 (13.4)	58.4 (12.7)	53.4 (13.7)	0.327
TRIG	mg/dL	82.4 (66.3)	78.9 (44.6)	65.9 (28.1)	100.7 (108.5)	0.079
Log-TRIG	—	1.8 (0.2)	1.8 (0.2)	1.8 (0.2)	1.9 (0.3)	0.104

* Note. column proportions by row do not significantly differ, $p > 0.05$.

Table 2.

Models findings predicting lipid and metabolic outcomes controlling for gender, smoke exposure, BMI, and steroid use.

Dependent variable	Predictors	B	SE B	Beta
LDL *	Gender	4.771	3.231	0.112
	Smoke	1.085	3.500	0.024
	BMI percentile *	0.210	0.053	0.300
	Asthma	0.156	3.672	0.003
TC	Gender	7.295	3.924	0.144
	Smoke	.238	4.259	0.023
	BMI percentile *	0.131	0.064	0.158
	Asthma	0.341	4.435	0.006
HDL **	Gender *	4.101	1.99	0.152
	Smoke	1.372	2.159	0.047
	BMI percentile *	-0.142	0.033	-0.321
	Asthma	-2.283	2.248	-0.075
Log-TRIG **	Gender	-0.058	0.033	-0.132
	Smoke	-0.047	0.036	-0.098
	BMI percentile *	0.002	0.001	0.263
	Asthma	0.069	0.037	0.139
HbA1C	Gender *	0.105	0.050	0.163
	Smoke	0.003	0.054	0.004
	BMI percentile	0.001	0.001	0.053
	Asthma *	-0.116	0.056	-0.160
HOMA2 **	Gender	-0.185	0.138	-0.098
	Smoke	0.083	0.150	0.040
	BMI percentile **	0.012	0.002	0.378
	Asthma	-0.003	0.157	-0.001

** Note. $p < 0.001$,

* $p < 0.05$.