Published in final edited form as:
J Asthma. 2011 September ; 48(7): 701–706. doi:10.3109/02770903.2011.604885.

Associations of Asthma with Body Mass Index and Adult Weight Change among Reproductive Age Women

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Abstract

Objective—To evaluate the cross-sectional relationship between asthma and pre-gravid body mass index (BMI); and to assess the risk of adult weight change among women with history asthma diagnosed in childhood or adulthood, respectively.

Study design—Study participants were 3,737 pregnant women enrolled in a cohort study. Information on history of asthma, pre-gravid BMI, adult weight change (difference between BMI at age 18 and pre-gravid BMI) and other socio demographic characteristics was collected using interviewer-administered questionnaires. Pre-gravid BMI was categorized into lean (BMI <18.5 kg/m^2), overweight (BMI 25–24.9 kg/m^2) and obese (BMI ≥30 kg/m^2). Logistic regression was used to estimate odds ratios (OR) and 95% confidence intervals (CI).

Results—Approximately 13.1% of study participants reported history of asthma. Compared with the reference group (BMI 18.5–24.9 kg/m^2), the odds of asthma was higher among overweight (OR=1.51; 95%CI 1.18–1.93) and obese (OR=1.47; 95% CI 1.06–2.03) women while it was lower among lean women (OR=0.42; 95% CI 0.21–0.84) (trend p-value <0.001). Women who gained ≥20 kg, compared with those who maintained their weight (±2.5 kg) had a 2.7-fold increased odds of asthma (95% CI 1.02–7.00).

Conclusion—Overweight and obese women were more likely to have history of asthma. Adult weight gain was positively associated with asthma diagnosis. Longitudinal studies designed to prospectively assess patterns of adult weight change in relation to asthma are warranted.

Keywords

Asthma; Pediatric Asthma; Body Mass Index; Obesity; Adult Weight Change

Introduction

The impact of asthma on health, quality of life, and the economy is substantial (1, 2,3). Reports from the Centers for Disease Control and Prevention (CDC) indicate that 34 million

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Declaration of Interest
This research was completed while Dr. Neway Fida was a Postdoctoral Research Fellow with the University of Washington Reproductive, Perinatal and Pediatric Research Training Program supported by the an award from the from the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, (T32 HD052462). This research was also supported by awards from the National Institutes of Health (R01 HD-032562 and R01 HD-055566).

Americans receive a diagnosis of asthma during their lifetime, and that asthma accounts for approximately 4,000 deaths per annum (1, 2,3). Being overweight (body mass index [BMI] 25–29.9 kg/m^2) and obese (BMI ≥30 kg/m^2) are now recognized national health threats and major public health challenges (1, 2, 4, 5, 6). According to the 2009 Behavioral Risk Factor Surveillance System survey (BRFSS), the overall prevalence of obesity in the United States in 2009 was 26.7 %. The prevalence was 27.4 % among men and 26.0% among women (7). Obese adults are at increased risk for many serious health conditions including coronary heart disease, hypertension, stroke, type 2 diabetes, certain types of cancer and, premature death (2, 4,8).

Accumulating evidence from basic science and population research suggests associations of overweight and obesity status with risk of asthma (2, 9, 10). Hypotheses based on mechanical, inflammatory and common genetic risk factors have been proposed to explain the potential relationships between obesity and asthma (2, 8, 11–15).

While some population studies have shown that overweight and obese individuals are at higher risk for developing asthma (30–90% higher risk) compared to individuals with normal weight (11, 13, 16–24) others have not observed similar associations (25). Further, few studies have investigated the relationship of asthma with overweight or obesity status among reproductive age women. Given inconsistencies and gaps in the literature and the potential public health significance of both overweight and obesity status and asthma, we conducted the present study. Using data from a large prospective cohort, we examined the crosssectional relationship between asthma and pre-gravid obesity among reproductive aged women. We first sought to assess the cross sectional relationship of adult weight change and adult-diagnosed asthma. We hypothesized that reproductive age women who gained more weight were likely to have a higher odds of asthma. In order to address the potential temporal relationship between asthma and weight change, we examined associations of pediatric-diagnosed asthma with adult weight change.

Methods

Study population

This cross-sectional study was conducted using information collected from participants in the Omega study, a prospective study designed to examine metabolic/dietary predictors and other risk factors for pregnancy complications. Participants were recruited from women who initiated prenatal care prior to 20 weeks of gestation at affiliated clinics of Swedish Medical Center, Seattle, WA or Tacoma General Hospital, Tacoma, WA. Ineligibility criteria included < 18 years of age, not speaking or reading English, not planning to carry the pregnancy to term and/or not planning to deliver at the two hospitals.

During the time period between 1996 and 2008, 5,063 women were approached and 4,000 (79%) consented to participate in the study and were enrolled. More than 95% of enrolled participants were followed through pregnancy completion. Women with incomplete information on history of asthma(N=263)were excluded from the current analysis. The analysis for this report is based on information collected from 3,737 pregnant women enrolled in the study. The study protocol was approved by the Institutional Review Boards of Swedish Medical Center and Tacoma General Hospital; and accepted under cooperative agreement, by the University of Washington. All participants provided written informed consent.

Data collection

Trained research personnel interviewed participants (16 weeks of gestation, on average) using a structured questionnaire to gather information on socio-demographic and

anthropomorphic characteristics (including height and weight characteristics, described below), lifestyle habits, and medical and reproductive histories. Interviewers asked each woman to report her height without shoes, her recalled weight at age 18, and her weight immediately before the study pregnancy. Participants' history of asthma diagnosis was determined by self-report using the response to the question "Has a doctor ever told you that you have asthma?" Pediatric asthma was defined as asthma diagnosed before the age of 18 while adult-diagnosed asthma was defined as asthma diagnosed on or after participants' 18th birthday.

BMI was computed by dividing weight (in kg) by height (in m) squared. Based on their pre-pregnancy BMI, participants were categorized in to the following groups; <18.5 kg/m^2 (lean), 18.5–24.9 kg/m^2 (normal weight), 25.0–29.9 kg/m^2 (overweight), ≥30 kg/m^2 (obese). For some additional exploratory analyses, participants were further categorized for pre-pregnancy BMI in the following expanded categories: <18.5 (lean), 18.5–24.9 (normal), 25.0–29.9 (overweight), 30.0–34.9 (obese), 35.0 to 39.9 (severely obese) and ≥40.0 kg/m^2 (morbidly obese). Based on reported weights at age 18 years and pre-gravid weight, adult weight change was calculated as the difference between the last self-reported weight before the current pregnancy and weight at age 18. Adult weight change was categorized as follows: loss of 2.5 kg or more, net change of fewer than 2.5 kg (reference), 2.5–4.9 kg gain, 5.0–9.9 kg gain, 10.0–19.9 kg gain and gain of 20 kg or more.

Statistical analysis

Selected characteristics of the study population were compared using mean and standard deviation (SD) for continuous variables and number (%) for categorical variables, across pre-pregnancy BMI categories. Statistically significant differences among the groups were assessed using one-way ANOVA (for continuous variables) and Chi Square (for categorical variables) tests. We used unadjusted and multivariable-adjusted logistic regression model to compute odds ratios (OR) and 95% confidence intervals (CIs) relating pre-pregnancy BMI and prevalent asthma. We evaluated associations of adult weight change with adult-diagnosed asthma after excluding participants who had pediatric asthma. We also evaluated associations of pediatric asthma with adult weight change after excluding participants who developed adult-diagnosed asthma. In multivariable models, we adjusted for *a priori* selected potential confounders and covariates that altered unadjusted ORs by at least 10%. These included participants' age at interview, parity, race/ethnicity, smoking, income, education, history of hypertension or diabetes mellitus, smoking, exercise and BMI at age 18. Parity, employment status and exercise were not found to be confounders. All reported p values are two-tailed, and CIs were calculated at the 95% level. Statistical significance was determined using the cut-off p-value (or α <0.05). Statistical analysis was performed using SPSS version 16 software (SPSS, Inc., Chicago, IL).

Results

Selected socio-demographic, medical and behavioral characteristics of the study participants (N= 3,737) are shown in Table 1. The mean age of participants was 32.6 years old (SD: 4.5 years). About 16.4% and 9.0% of participants had pre-pregnancy overweight and obese status, respectively. As expected, participants with higher pre-pregnancy BMI were older, tended to be more physically inactive and had either personal or family history of hypertension or diabetes (all p-values <0.001).

Overall, 13.1% of the cohort included in the current study reported having history of physician diagnosed asthma (488 of 3,737), of which 7.3% were pediatric diagnosed asthma (n=272) and 5.6% adult-diagnosed asthma (n=211). Age at asthma diagnosis was unknown for 5 study participants with an asthma diagnosis. Prevalence estimates of asthma among the

lean, normal, overweight and obese categories of participants were 5.6%, 12.0%, 17.1% and 17.2%, respectively (Table 2).

As shown in Table 2, in both unadjusted or multivariable adjusted models, pre-pregnancy BMI was strongly associated with the prevalence of asthma (trend p-value < 0.001) (Table 2). Compared with the referent normal BMI group, the odds of asthma were 57% lower among lean participants adjusted OR(aOR)=0.43; 95% CI 0.21–0.84). On the other hand, compared with the referent group, the odds of asthma were increased 51% (aOR=1.51; 95% CI 1.18–1.93) and 47% (aOR=1.47; 95% CI 1.06–2.03) for overweight and obese participants respectively for overweight women and for obese women. Results from analyses that further explored the odds of asthma according to extended categories of BMI indicated that the odds of asthma was approximately 2-fold higher (aOR=1.96; 95% CI 1.04–3.69) for morbidly obese (≥40 kg/m^2) women.

We next evaluated the odds of pediatric and adult-diagnosed asthma for women in the different categories of adult weight change.

The prevalence of adult-diagnosed asthma was associated with adult weight change (trend p-value=0.045) (Table 3). Adult weight gain of 10–19.9 kg was associated with 1.4 fold higher odds of adult-diagnosed asthma (95% CI 0.57–3.66), though the association was not statistically significant. The odds of adult-diagnosed asthma among participants who gained ≥20.0 kg in adulthood was 2.7 (95% CI 1.02–7.00). We observed no evidence of an association of pediatric diagnosed asthma and adult weight change (trend p-value=0.63).

Discussion

In our current study of reproductive age women we found significant associations of odds of asthma with pre-pregnancy BMI. The odds of asthma were 1.5-fold among women who are overweight or obese, compared with normal weight women. We also noted that adult-diagnosed asthma was positively associated adult weight change of ≥20 kgs.

However, pediatric-diagnosed asthma was not associated with adult weight change. Our findings of higher odds of asthma among overweight and obese women are consistent with some previous reports (11–13, 16, 18, 21, 22, 26), but not others (25, 27).

Mc Hugh and colleagues reported positive association between measured BMI and self-reported asthma among women participants of (2001–2002 and 2003–2004) National Health and Nutrition Examination Survey (NHANES) (16). The authors noted that, after adjusting for race/ethnicity, BMI, smoking history and poverty income ratio, obese (BMI >30 kg/m^2) women (OR=1.6, 95% CI, 1.2–1.9) and extremely obese (BMI >40 kg/m^2) women (OR=1.7, 95% CI, 1.3–2.2) had increased odds of asthma compared to women with normal BMI (18.5–24.9 kg/m^2) (16). Additionally, Behren and colleagues, in their California Teachers Study cohort of 88,304 women, reported that compared with those of normal weight, the adjusted OR for adult-diagnosed asthma increased from 1.40 (95% CI 1.31–1.49) for overweight women to (3.30;95% CI 2.85–3.82) for extremely obese women (26). However, these findings were not replicated in several other studies others (25, 27). For instance, Barranco and colleagues found no association between overweight or obesity status with asthma diagnosis in their cross-sectional multicenter study of Spanish adults, (OR=0.89; 95% CI 0.60–1.38) for overweight and (OR= 0.93; 95% CI 0.57–1.48) for obese participants, respectively. In fact, the risk of asthma was lowest among underweight women in our population. These contradictory findings could have resulted from study population differences. In our population, being underweight may reflect the higher health conscious segment of the population that potentially has healthy habits that are associated with lower

risk of asthma. On the other hand, being underweight may be a reflection of poor health status and characteristics that reflect a higher risk of asthma (e.g., smoking).

Inconsistencies across findings of previous studies could also attributable to differences in study population characteristics (e.g. differences in age, sample sizes or differences in asthma diagnosis methods). In our study population, which is relatively affluent, being underweight may reflect the higher health conscious segment of the population that potentially has healthy habits that are associated with lower risk of asthma. On the other hand, being underweight, in other settings, may be a reflection of poor health status and characteristics that reflect a higher risk of asthma (e.g., smoking). Further, the differences in age of study populations may be directly related to differences in prevalence of asthma or other risk factors (e.g. obesity).

Our findings of positive associations of weight gain in adulthood with prevalence of adult-diagnosed asthma are generally consistent with previous reports from the Nurses' Health Study, the California Teachers Study and the Black Women's Health Study (19, 26, 28, 29). For instance, using the Nurse's Health Study, Camargo and colleagues, reported that, participants who gained 10–20 kg since age 18 had a 1.4 fold increased risk (RR=1.4; 95% CI 1.2–1.7), those who gained 21–25 kg and those who gained more than 25 kg had more than doubled risk (95% CI 1.6–2.5; and RR= .5; 95% CI 2.0–3.1 respectively) of developing adult-diagnosed asthma compared with those participants whose weight had remained stable.

Several plausible hypotheses concerning biological mechanisms (based on physiological, immunological, mechanistic, dietetic and environmental mechanisms) have been put forth to explain previously reported relationships between obesity and asthma (2, 10, 15). Among overweight or obese adults, respiratory compliance is reduced by excess soft tissue weight compressing the thoracic cage, fatty infiltration of the chest wall, and an increase in pulmonary blood volume (2, 11). Reduction in lung volume is associated with reduction in the diameter of peripheral airways that can lead to changes in the function of bronchial smooth muscle. This in turn leads to a change in the actin-myosin cross-bridge cycle, which can potentially increase both obstruction and bronchial hyper reactivity (8, 12, 14, 15). Further, obesity is believed to be a state of chronic low grade systemic inflammation (15). The increase in the normal functioning of adipose tissue in obese subjects leads to a systemic pro-inflammatory state which produces a rise in serum concentrations of several cytokines, the soluble fractions of their receptors and chemokines (2, 8, 15, 30), some of which (e.g., leptin, adiponectin, interleukin (IL)-4, IL-6, C-reactive protein) may contribute to development of asthma. Others have suggested that systemic oxidative stress due to obesity might cause airway oxidative stress and inflammation and lead to asthma assessing oxidative stress using plasma F2-isoprostane levels among levels among asthmatics and non-asthmatics (14). Others have hypothesized that the enzyme aromatase which is present in adipose tissue, results in increased production or peripheral sensitivity of female hormone - estrogen. Estrogen is assumed to result in disruption of lung development and regulation of airway tone in pubertal girls and reached to a conclusion that obesity was related to development of asthma in women than men (15). On the other hand, the use of steroids as pharmacological treatment of asthma and the decreased mobility related to the symptoms of asthma might also contribute to the excess weight gain among asthmatics (29,31).

Our study has some limitations that deserve mention. Majority of participants were Caucasians, which limits the generalizability. However, as described earlier, concordance of our findings with reports of other studies conducted among diverse racial, ethnic and socio-economic background is reassuring (28). We used self reported weight (at age 18 years and pre-gravid) and height to determine BMI, which may have resulted in some degree of

misclassification. However, findings from earlier studies have shown high correlations between self reported weight and measured weight (r=0.96) (32, 33). In a previous study, investigators reported that young women's self-reported height and weight correlated well with measured values, women slightly underreported their weight at age 18 (34). Such misclassification, if present in the current study, is unlikely to be related to asthma diagnosis and would thus likely bias OR estimates toward the null hypothesis of no association. We also used self report of physician diagnosed asthma which may result in inclusion of more severe asthma. We note, however, that the prevalence of asthma in our population is similar to the adult self reported asthma prevalence reports from other national studies (33). Our large sample size may have led to highly statistically significant p-values despite small difference across BMI groups, particularly for analyses reported in Table 1. Finally, use of steroids as treatment of asthma was not recorded in our study. Consequently, we could not investigate the role of asthma medication in the development of overweight and obesity status.

Conclusion

In summary, we have found significant associations between prevalence of asthma and overweight and obese status among women of reproductive age. Moreover, found significant associations of adult-diagnosed asthma with adult weight change of ≥20 kg. Our findings add to the body of knowledge of the relationship between asthma and BMI among reproductive age women. Future prospective studies that address the temporal relationship between BMI, weight gain and prevalent asthma are warranted.

Acknowledgments

The authors wish to thank the staff of the Center for Perinatal Studies, Swedish Medical Center, Seattle, WA, USA for their technical assistance with this research.

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Table 1

Selected characteristics of the study population according to pre-gravid body mass index, Seattle and Tacoma, Washington, USA 1996–2008

		Pregravid Body mass index (kg/m^2)				
	Total N=3,737	**<18.5 N=160**	**18.5–24.9 N=2,626**	**25.0–29.9 N=613**	**(≥30.0) N=338**	**P value Trend test****
Characteristics	**n (%)**	**n (%)**	**n (%)**	**n (%)**	**n (%)**	
Age (y) mean*	32.6±4.5	32.0±0.3	32.7±4.3	33.0±4.8	32.1±5.3	0.001
Race, Non-Hispanic White	3228(86.4)	135(84.4)	2304(87.7)	518(84.5)	271(80.2)	0.001
Weight, kg, at age 18*	57.4± 9.0	50.3±5.4	55.6± 6.5	60.5± 9.2	69.5±14.3	<0.003
BMI, kg/m^2 at age 18*	20.7± 2.9	17.9± 1.5	20.0± 1.9	21± 2.8	25.3± 4.7	<0.001
Weight change, kg, since age 18*	8± 10.9	−0.05± 4.5	4.4± 5.6	14.3± 8.3	28.9± 17.3	<0.001
Education ≤ high school	153(4.1)	3(1.9)	86(3.3)	28(4.6)	36(10.7)	<0.001
Currently not employed	693(18.5)	38(23.8)	485(18.5)	101(16.5)	69(20.4)	0.145
Married	3401(91.0)	147(91.9)	2411(91.8)	554(90.4)	289(85.5)	<0.002
Multiparous	1386 (37.1)	66 (41.2)	916(34.9)	247 (40.3)	157 (46.4)	<0.001
Physically active	3442(92.1)	148(92.5)	2457(93.6)	559(91.2)	278(82.2)	<0.001
History of hypertension	178(4.8)	3(1.9)	59(2.2)	53(8.6)	63(18.6)	<0.001
History of diabetes mellitus	48(1.3)	1(0.6)	25(1.0)	6(1.0)	16(4.7)	<0.001
Family history of diabetes mellitus	552(14.8)	14(8.8)	346(13.2)	108(17.6)	84(24.9)	<0.001
Family history of hypertension	1870(50.0)	74(46.2)	1272(48.4)	314(51.2)	210(62.1)	<0.001
Ever smoker	1041(27.9)	35(21.9)	693(26.4)	209(34.1)	104(30.8)	<0.001
Asthma	488(13.1)	9(5.6)	316(12.0)	105(17.1)	58(17.2)	<0.001

* Mean ± standard deviation otherwise Number (%)

** continuous variable: one-way ANOVA test, categorical variables: use the Chi-Square test or Fisher's Exact test (if the cell number <5)

Table 2

Unadjusted and adjusted odds ratios (OR) and 95% confidence intervals (CI) of asthma according to categories of participants' pre-gravid body mass index Seattle and Tacoma, Washington, USA 1996–2008

	Body mass index (kg/m^2)				P-value for trend
	Lean <18.5 (N=160)	**Normal 18.5–24.9 (N=2626)**	**Overweight 25.0–29.9 (N=613)**	**Obese ≥30.0 (N=338)**	
	n (%)	**n (%)**	**n (%)**	**n (%)**	
Asthma					
No, n (%)	151 (94.4)	2310 (88.0)	508 (82.9)	280 (82.8)	
Yes, n (%)	9 (5.6)	316 (12.0)	105 (17.1)	58 (17.2)	
Unadjusted OR (95%CI)	0.44(0.22–0.86)	1.00 (Reference)	1.51(1.18 – 1.92)	1.51(1.11–2.05)	<0.001
* Adjusted OR (95%CI)	0.43(0.21–0.84)	1.00 (Reference)	1.51(1.18 – 1.93)	1.47(1.06 – 2.03)	<0.001

* Adjusted for age, race/ethnicity, educational attainment, marital status, smoking, exercise status, history of chronic hypertension or diabetes mellitus, family history of diabetes and family history of hypertension

Table 3

Adjusted odds ratios (OR) and 95% confidence intervals (CI) of adult diagnosed asthma and history of pediatric-diagnosed asthma according to adult weight change among participants, Seattle and Tacoma, Washington, USA 1996–2008

Variables of interest	No Asthma N=3249	Adult-Diagnosed Asthma* N=211		History of Pediatric –Diagnosed Asthma** N=272	
	n (%)	n (%)	Adjusted OR (95% CI)	n (%)	Adjusted OR (95% CI)
Adult weight change (kg)					
≤ -2.5	263 (8.1)	10 (4.7)	0.57 (0.12–2.76)	17 (6.2)	0.63 (0.22–1.75)
−2.5 to 2.5	737 (22.7)	36 (17.1)	1.00 (Reference)	62 (22.8)	1.00 (Reference)
2.6 to 4.9	482 (14.8)	33 (15.6)	1.31 (0.46–3.76)	38 (14.0)	0.94 (0.43–2.07)
5.0 to 9.9	723 (22.3)	37 (17.5)	0.82 (0.29–2.36)	62 (22.8)	0.94 (0.47–1.84)
10.0–19.9	665 (20.5)	52 (24.7)	1.44 (0.57–3.66)	64 (23.5)	0.96 (0.49–1.90)
≥20.0	329 (10.1)	41 (19.4)	2.67 (1.02–7.00)	26 (9.6)	0.57 (0.21–1.53)
missing	50 (1.5)	2 (1.0)		3(1.1)	
P-value for trend			*0.045*		*0.63*

Adjusted for age, race, physical activity, smoking, educational attainment, history of diabetes mellitus or hypertension and family history of diabetes mellitus or hypertension

NOTE: Five asthma patients did not have age at diagnosis information

* Pediatric-diagnosed asthma cases excluded

** Adult – diagnosed asthma cases excluded