

Asthma control in pregnancy is associated with pre-conception dietary patterns

Jessica A Grieger¹, Luke E Grzeskowiak¹, Lisa G Wood² and Vicki L Clifton^{1,*}

¹Robinson Research Institute, School of Paediatrics and Reproductive Health, University of Adelaide and Lyell McEwin Hospital, Haydown Road, Elizabeth Vale, SA 5112, Australia; ²Hunter Medical Research Institute, School of Biomedical Science and Pharmacy, University of Newcastle, Kookaburra Circuit, New South Wales, Australia

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Abstract

Objective: To examine pre-conception dietary patterns in pregnant asthmatic women and to identify associations between maternal diet and asthma control during pregnancy.

Design: Cross-sectional study. Pre-conception food frequency data were collected retrospectively. Asthma control was assessed using the Global Initiative for Asthma guidelines. Dietary patterns were derived using factor analysis. Binary logistic regression analyses were used to test the association between uncontrolled asthma and each dietary pattern (Z-score), with values presented as odds ratio and 95% confidence interval.

Setting: Antenatal clinic in a tertiary hospital, Adelaide, Australia, May 2009–July 2013.

Subjects: One hundred and fifty-eight asthmatic pregnant women.

Results: Three dietary patterns were identified: (i) 'high protein/fruit' (strong food group loadings for fish, meat, chicken, fruit); (ii) 'high fat/sugar/takeaway' (takeaway foods, crisps, refined grains); and (iii) 'vegetarian-type' (vegetables, fruit, soya milk, whole grains). A 1 SD increase in score on the high fat/sugar/takeaway pattern was associated with increased likelihood of uncontrolled asthma (adjusted OR = 1.54; 95% CI 1.07, 2.23; $P = 0.022$). Women with uncontrolled asthma ($n = 115$) had higher energy-adjusted intakes of saturated fat, monounsaturated fat, carbohydrate, sugar and fibre compared with women with controlled asthma ($n = 43$, all $P \leq 0.05$).

Conclusions: Pre-pregnancy dietary patterns may influence maternal asthma control. Our work highlights the importance of achieving a healthy diet before pregnancy that is low in saturated fat, sugar and takeaway foods, and therefore higher in lean meats, poultry and fish, as well as fruits, vegetables and whole grains. A healthy dietary pattern should be encouraged in all asthmatic women who are of childbearing age, and should additionally be promoted before pregnancy and beyond.

Keywords
Pre-conception diet
Asthma
Factor analysis
Dietary patterns
Pregnancy
Australia

Asthma is a heterogeneous and complex disease, involving alterations in airway structure and airway inflammation. Worldwide, asthma represents one of the most common chronic diseases to affect pregnancies, affecting between 8 and 13% of the population^(1–3). A meta-analysis from 2011 demonstrated that asthma in pregnancy was associated with up to 54% increased risk for low birth weight, small for gestational age, preterm delivery and pre-eclampsia⁽⁴⁾. A subsequent meta-analysis revealed that asthma exacerbations in pregnancy increased the risk for low birth weight (relative risk (RR) = 3.02; 95% CI 1.87, 4.89) and compared with women with mild asthma, women with moderate to severe asthma were at increased risk for

babies born with low birth weight (RR = 1.15; 95% CI 1.05, 1.26) or small for gestational age (RR = 1.24; 95% CI 1.15, 1.35)⁽⁵⁾. These studies suggest that worsening asthma during pregnancy may contribute to poor outcomes for the fetus and that approaches to improve asthma control may be of benefit⁽⁶⁾.

In non-pregnant asthmatics, unhealthy diets have been shown to be an underlying contributor to poorly controlled asthma and subsequent risk of asthma exacerbations. In healthy individuals, a single high-fat meal has been linked to increased exhaled nitric oxide⁽⁷⁾ and in non-obese asthmatics, a high-fat meal increased neutrophilic inflammation and attenuation of airway

*Corresponding author: Email vicki.clifton@adelaide.edu.au

bronchodilator response⁽⁸⁾. A low-fibre, high-fat diet has been linked to worse airway inflammation and lung function in severe asthmatics⁽⁹⁾. Furthermore, a diet low in fruits and vegetables has been shown to increase the risk of asthma exacerbation by twofold⁽¹⁰⁾. In pregnancy, altered food patterns have previously been associated with excess gestational weight gain⁽¹¹⁾, risk for small for gestational age⁽¹²⁾, risk of preterm delivery⁽¹³⁾ and development of childhood allergies⁽¹⁴⁾. However, the connection between dietary intake and asthma control in pregnancy is not clear.

Only one study to date has assessed dietary intakes in pregnant women with asthma in which nutrient intake was generally higher in mildly asthmatic women, yet lower in moderate to severe asthmatics, compared with pregnant women without asthma⁽¹⁵⁾. Moreover, little attention has been paid to maternal dietary intakes pre-conception and the potential impact this might have on maternal asthma control. If pre-conception diet is linked to asthma control, this would present an ideal time and opportunity to consume a healthy diet to control asthma during pregnancy, thus optimising subsequent perinatal outcomes.

The aims of the present study were to: (i) identify associations between pre-conception dietary patterns and maternal asthma control; and (ii) determine whether there are any differences in dietary intakes between women with controlled *v.* uncontrolled asthma. We hypothesise that pre-conception diet quality influences maternal asthma control during pregnancy.

Methods

Study setting and population

The current retrospective, cross-sectional study was part of a larger prospective cohort study from the Lyell McEwin Hospital, Adelaide, Australia, assessing the effects of asthma during pregnancy on the mother, placenta and baby. Pregnant women aged >18 years and attending their first antenatal visit were invited to participate. Recruitment and data collection for all women and their babies took place between May 2009 and July 2013. Of the 400 pregnant women who consented, ninety-one women withdrew from further participation due to the following: unable to be contacted (*n* 10), could not attend appointments/not interested (*n* 56), relocated (*n* 9), miscarriage (*n* 12) or voluntary termination of pregnancy (*n* 4), leaving 309 women to be analysed. The project was approved by The Queen Elizabeth Hospital and Lyell McEwin Hospital Human Research Ethics Committee and The University of Adelaide Human Research Ethics Committee. All women gave written informed consent. We have recently examined the relationship between pre-conception dietary patterns in a sample of 309 pregnant women⁽¹³⁾. We now present data from a subgroup of asthmatic women (*n* 159), in whom we have used the same dietary pattern analysis.

Maternal data collection

At the first antenatal clinic visit, maternal body weight was measured to the nearest 0.1 kg using calibrated electronic scales (Professional Medical Scale, ScalesPlus, Australia) and height was measured by a wall-mounted stadiometer. BMI was calculated as [weight (kg)]/[height (m)]². Previous obstetric history, medical, mental and surgical health information, and socio-economic status (Socio-Economic Indexes for Areas) were obtained from medical records. Gestational age was determined by date of the last menstrual period and confirmed at 18-week ultrasound. Smoking history was recorded: women who did not smoke at any stage during pregnancy or who were former smokers (i.e. quit a median of 3 years ago) were classified as non-smokers, while current smokers and those who quit smoking in pregnancy (median 5.5 weeks' gestation) were classified as smokers.

Asthma assessment

Women attending their first booking visit at the Lyell McEwin Hospital antenatal clinic were identified as controls (non-asthmatic) or asthmatics, by the attending midwife. To determine asthma, the midwife asked 'Have you been told by a doctor that you have asthma?' and 'Have you used any asthma medications in the last year like salbutamol or a preventer?' Women identified as having asthma received a baseline assessment of their asthma during their first study visit (median 13 weeks' gestation). Given the importance of evaluating asthma control, rather than asthma severity, to guide asthma management decisions⁽¹⁶⁾, asthma control was assessed using the Global Initiative for Asthma (GINA) guidelines⁽¹⁷⁾. These guidelines define asthma control as: minimal or no daytime symptoms; no limitations of daily activities; no nocturnal symptoms; minimal or no need for rescue therapy and normal lung function⁽¹⁷⁾. Therefore, based on current symptoms women were identified as having either uncontrolled or partly controlled/controlled asthma. Lung function was assessed using an EasyOne diagnostic spirometer (Niche Medical, Zurich, Switzerland). Forced expiratory volume at one second (FEV₁) and forced vital capacity (FVC) were measured before and after administration of salbutamol. Predicted values for lung function were calculated based on women's height and age according to the equations of Gore *et al.*⁽¹⁸⁾.

Dietary intakes and food group consumption

At the first antenatal visit (median 13 weeks' gestation), the validated Cancer Council of Victoria's Dietary Questionnaire for Epidemiological Studies FFQ was used to obtain dietary intake data covering the 12 months prior to pregnancy. One hundred different foods (g/d) were obtained from the FFQ and were assigned into thirty-three food groups (g/d), based on a previous Australian study⁽¹⁹⁾ and used for analysis.

Factor analysis

Dietary patterns were derived using factor analysis with factor loadings extracted using the principal component method and varimax/orthogonal rotation. The number of dietary patterns identified was based on eigenvalues >1.5 , on identification of a break point in the scree plot and on interpretability⁽²⁰⁾. Using these criteria, a three-factor solution was chosen and re-run with the resulting factor scores saved and converted to Z-scores for analysis. Items with factor loadings >0.25 were considered as the items of relevance for the identified factor. These items represent the foods most highly related to the identified factor⁽²¹⁾. Foods that cross-loaded on several factors were retained. Inter-item reliability for each factor was assessed using Cronbach's α coefficients. Three hundred and nine women had complete FFQ data and the three major dietary patterns that were identified in the factor analysis have been published⁽¹³⁾.

Energy adjustment

Due to the potential for confounding due to differences in total energy intake, we analysed the relationship between both absolute and energy-adjusted nutrient intakes and the presence of uncontrolled asthma. Energy adjustment of nutrient intake was performed using the residuals method, in accordance with previously published descriptions⁽²²⁾. The residuals method involves performing a linear regression analysis with nutrient intake as the dependent variable and total energy intake as the independent variable for each nutrient. This regression equation calculates the expected mean nutrient intake of the study population according to the mean total energy intake of the study population. Subsequently, the energy-adjusted intake for each individual was calculated by adding the expected mean macronutrient intake of the study population to the residual derived from the regression analysis.

Statistical analysis

Frequencies and descriptive statistics are expressed as number and percentage and as mean and standard deviation, respectively. Prior to hypothesis testing, data were examined for normality. Distribution was normal except for α -carotene, β -carotene, lutein, lycopene and long-chain *n*-3 PUFA, which were normalised using natural logarithmic transformation. Calculation of BMI categories was carried out in accordance with the WHO criteria⁽²³⁾. Pearson correlation coefficients were used to test the associations between dietary pattern scores and nutrient intakes. Binary logistic regression analyses were used to test the association between uncontrolled asthma and each dietary pattern (Z-score), with values presented as odds ratio and 95% confidence interval. All analyses were undertaken adjusting for potential confounders, including maternal age, maternal BMI, smoking status (never/former, quit in pregnancy/current), socio-economic

status (level 1 or ≥ 2), parity (0 or ≥ 1) and ethnicity (Caucasian or other). Multicollinearity was tested using the variance inflation factor, in which no multicollinearity was observed between any of the independent variables. Differences in mean nutrient intakes between women with controlled and uncontrolled asthma were compared using an unpaired *t* test. Binary logistic regression analyses were used to test the association between uncontrolled asthma and each nutrient, while adjusting for potential confounders including maternal age, smoking status, socio-economic status, BMI and parity. Statistical significance was considered at $P < 0.05$. All statistical procedures were carried out using the statistical software package IBM SPSS Statistics version 19.

Results

Characteristics of the 158 asthmatic women in the current sample are reported in Table 1. There was no difference in mean weight gain during pregnancy between those with

Table 1 Characteristics of the study population at the first visit*: asthmatic pregnant women (*n* 158), Adelaide, Australia, May 2009–July 2013

	Mean or <i>n</i>	SD or %
Maternal age (years)†	27	6
Gravidity‡		
≥ 2	106	65
Parity‡		
≥ 1	57	35
BMI (kg/m ²)†	28.34	7.11
Smoking status‡		
Never/former	112	69
Quit in pregnancy	18	11
Current smoker	33	20
Ethnicity‡		
Caucasian	149	91
Other	14	9
Socio-economic status‡		
5 (highest)	9	6
4	10	6
3	3	2
2	51	33
1 (lowest)	81	53
Asthma medication‡		
SABA	112	69
SABA + ICS	11	7
SABA + ICS/LABA	40	24
Baseline ICS dose ($\mu\text{g}/\text{d}$)†	544	362
Spirometry†		
FEV ₁ (litres)	2.93	0.48
% predicted FEV ₁	0.92	0.11
FVC (litres)	3.66	0.62
% predicted FVC	0.98	0.12
FEV ₁ /FVC	0.80	0.07
FEV ₁ /FVC%	0.95	0.07

SABA, slow-acting β agonist; ICS, inhaled corticosteroid; LABA, long-acting β agonist; FEV₁, forced expiratory volume at one second, FVC, forced vital capacity.

*Median 13 weeks' gestation.

†Values presented are mean and standard deviation.

‡Values presented are number and percentage.

Table 2 Pearson correlation coefficients (*r*) between dietary pattern scores and nutrient intakes among asthmatic pregnant women (*n* 158), Adelaide, Australia, May 2009–July 2013

Nutrient	High protein/fruit	High fat/sugar/takeaway	Vegetarian-type
Energy (kJ)	0.58	0.69	
Total fat (g)	0.58	0.71	
Saturated fat (g)	0.52	0.72	
Polyunsaturated fat (g)	0.48	0.52	
Monounsaturated fat (g)	0.62	0.69	
Protein (g)	0.77	0.52	
Carbohydrate (g)		0.66	
Sugar (g)	0.43	0.42	
Fibre (g)	0.44		0.61
Ca (mg)	0.51		
Cholesterol (mg)	0.74	0.51	
Dietary folate equivalents (µg)	0.50		0.57
Fe (mg)	0.65	0.50	
Mg (mg)	0.65	0.43	0.41
Niacin equivalents (mg)	0.75	0.51	
Riboflavin (mg)	0.51	0.42	0.41
Na (mg)	0.68	0.62	
Thiamin (mg)	0.49	0.47	
Zn (mg)	0.70	0.55	
Vitamin C (mg)	0.40		0.49
Vitamin E (mg)	0.42	0.58	0.47
Vitamin A retinol equivalents (µg)		0.51	0.56
Linoleic acid (mg)		0.53	
α-Linolenic acid (mg)		0.75	
Long-chain <i>n</i> -3 PUFA (mg)	0.88		

Only correlations of $r > 0.40$ are included.
All correlations are significant ($P \leq 0.01$).

Table 3 Odds ratios for likelihood of uncontrolled asthma* at the first visit during pregnancy according to pre-conception dietary pattern among asthmatic pregnant women (*n* 158), Adelaide, Australia, May 2009–July 2013

	Unadjusted OR	95 % CI	<i>P</i>	Adjusted OR†,‡	95 % CI	<i>P</i>
Uncontrolled asthma at first visit (ACQ > 1.5)						
High protein/fruit	1.09	0.85, 1.41	0.485	1.04	0.80, 1.37	0.757
High fat/sugar/takeaway	1.59	1.13, 2.25	0.008	1.54	1.07, 2.23	0.022
Vegetarian type	1.10	0.78, 1.56	0.579	1.26	0.86, 1.84	0.231

ACQ, Asthma Control Questionnaire.

*According to Global Initiative for Asthma (GINA) classification measured in the first trimester⁽¹⁷⁾ at median 13 weeks' gestation.

†Adjusted for maternal age, maternal BMI, smoking status (never/former, quit in pregnancy/current), socio-economic status (level 1 or ≥ 2), parity (0 or ≥ 1) and ethnicity (Caucasian or other).

‡Indicates change in risk per 1 sd increase in factor score.

controlled *v.* uncontrolled asthma (11.13 (SE 0.55) kg *v.* 9.03 (SE 1.01) kg, $P = 0.084$).

Three dietary patterns were identified (see online supplementary material, Supplemental Table 1). Factor 1 was labelled 'high protein/fruit' as there were several protein-rich food groups loading on the pattern (i.e. fish, meat, chicken) as well as fresh fruit and canned fruit. Factor 2 was labelled 'high fat/sugar/takeaway' as takeaway foods, crisps, fried potato, refined grains and cakes/pastries loaded on this pattern. Factor 3 was labelled 'vegetarian-type' as many of the food groups loading on this pattern were different types of vegetables, soya milk and whole grains. There were strong correlations between the high protein/fruit pattern and long-chain *n*-3 PUFA, protein, cholesterol and Zn; there were strong correlations between the high fat/sugar/takeaway pattern and total fat,

α-linolenic acid, SFA and Na; and there were strong to moderate correlations between the vegetarian-type pattern and fibre, dietary folate and vitamin A retinol equivalents (Table 2).

Asthma control was measured at a median of 13 weeks' gestation. In multivariate regression analysis, a 1 sd increase in the score on the high fat/sugar/takeaway pattern was associated with 50 % increased likelihood for uncontrolled asthma (adjusted OR = 1.54; 95 % CI 1.07, 2.23; $P = 0.022$; Table 3). After adjusting for total energy intake, women with uncontrolled asthma had higher intakes of SFA, MUFA, total carbohydrate, sugar and fibre (Table 4). For each additional 40 g of carbohydrate intake, the odds of uncontrolled asthma increased by 40 %, while for each additional 10 g increase in sugar, the odds of uncontrolled asthma increased by 10 % (Table 4).

Table 4 Mean dietary intakes and nutrient density of the diet according to controlled and uncontrolled asthma status among asthmatic pregnant women (*n* 158), Adelaide, Australia, May 2009–July 2013

	Controlled asthma (<i>n</i> 115)		Uncontrolled asthma (<i>n</i> 43)		<i>P</i> value*	Adjusted OR†,‡	95 % CI	<i>P</i> value
	Mean	SE	Mean	SE				
Absolute intake								
Energy (kJ/d)	7700	331	9352	619	0.026	1.00	1.00, 1.00	0.201
Protein (g/d)	90.6	5.15	105	8.29	0.216	1.01	0.99, 1.01	0.111
Total fat (g/d)	80.9	3.95	99.2	7.57	0.040	1.01	0.99, 1.02	0.071
SFA (g/d)	35.0	1.80	43.7	3.45	0.031	1.02	0.99, 1.04	0.086
PUFA (g/d)	10.6	0.46	12.1	0.96	0.143	1.05	0.97, 1.13	0.206
MUFA (g/d)	28.4	1.48	35.1	2.84	0.045	1.03	1.00, 1.05	0.048
Carbohydrate (g/d)	190	7.07	234	14.4	0.006	1.01	1.00, 1.01	0.020
Sugar (g/d)	85.4	3.65	103	6.11	0.028	1.01	1.00, 1.02	0.015
Fibre (g/d)	18.2	0.70	23.0	1.28	0.003	1.08	1.03, 1.14	0.002
Cholesterol (mg/d)	294	17.3	327	28.4	0.379	1.00	0.99, 1.00	0.330
Energy-adjusted intake								
Protein (g/d)	88.3	5.06	102	8.12	0.225	1.01	0.99, 1.01	0.113
Total fat (g/d)	69.1	2.72	80.7	6.80	0.092	1.01	0.99, 1.02	0.572
SFA (g/d)	33.8	1.75	42.3	3.37	0.031	1.02	0.99, 1.04	0.088
PUFA (g/d)	8.87	0.39	10.0	0.85	0.202	1.05	0.96, 1.14	0.283
MUFA (g/d)	25.9	1.37	32.1	2.64	0.047	1.03	1.00, 1.06	0.049
Carbohydrate (g/d)	193	7.22	239	14.6	0.006	1.01	1.00, 1.02	0.014
Sugar (g/d)	87.9	3.73	106	6.27	0.027	1.01	1.00, 1.01	0.020
Fibre (g/d)	20.0	0.77	25.1	1.37	0.003	1.07	1.03, 1.13	0.003
Cholesterol (mg/d)	296	17.4	330	28.6	0.374	1.00	0.99, 1.00	0.325

**P* value represents differences between controlled asthma and uncontrolled asthma groups.

†Logistic regression analysis for each dietary nutrient that included the following covariates in the model: maternal age, smoking status, socio-economic status, BMI and parity.

‡Indicates change in risk per unit of dietary intake.

Discussion

The current study represents the first piece of evidence linking pre-conception dietary patterns in pregnant women to asthma control. We identified that a dietary pattern containing higher consumption of takeaway foods, fried potato, crisps and refined grains was associated with uncontrolled asthma. Women with uncontrolled asthma had higher intakes of SFA, MUFA, total carbohydrate, sugar and fibre, such that for each additional 40 g of carbohydrate intake, equivalent to 2.5 slices of white bread per day, the odds of uncontrolled asthma increased by 40%, while for each additional 10 g increase in sugar intake, equivalent to two teaspoons of added sugar per day, the odds of uncontrolled asthma increased by 10%.

Our findings are important as human epidemiological studies have shown in asthmatic adults that higher fat and lower fibre intakes were associated with worse airway inflammation and lung function⁽⁹⁾ and fast-food consumption ≥ 3 times per week was associated with increased risk of severe asthma in children and adolescents⁽²⁴⁾. Most recently, in a meta-analysis of observational studies, total intake of fruit and vegetables was inversely associated with risk of asthma in adults (six studies: RR = 0.64; 95% CI 0.41, 1.01) and children (two studies: RR = 0.57; 95% CI 0.42, 0.77), but higher consumption of fruit and vegetables during pregnancy was not associated with risk of asthma in offspring⁽²⁵⁾. Acute studies in human subjects have demonstrated that a single high-fat meal

increased exhaled nitric oxide⁽⁷⁾, increased circulating fatty acid levels and percentage neutrophils in sputum, and attenuated airway bronchodilator responses⁽⁸⁾. Comparatively, a randomised controlled trial showed that two servings of fruit and five servings of vegetables daily reduced the risk of asthma exacerbation in adults with stable asthma⁽¹⁰⁾. Animal studies have also shown that a high-fat diet can induce pro-inflammatory responses both systemically⁽²⁶⁾ and in the lungs⁽²⁷⁾. SFA can induce an inflammatory response via activation of pattern recognition receptors⁽²⁸⁾ and by activation of downstream pro-inflammatory signalling pathways⁽²⁹⁾. Worsening asthma in pregnancy could therefore be attributable in part to dietary intake.

Previous studies have shown that pre-conception diet does not differ significantly from dietary habits during pregnancy^(30,31). Although the mechanisms linking pre-conception diet with maternal and neonatal outcomes have not been investigated in human subjects, the Dutch famine studies that occurred during 1944–1945^(32,33) are probably the closest data that show how the timing of nutrients from the mother to the fetus impacts perinatal outcomes in both the short and long term. It was identified that babies exposed to famine in late gestation had reduced growth compared with babies not exposed to famine, whereas babies who were exposed to famine in early gestation were heavier at birth⁽³⁴⁾. Interestingly, in adulthood, those who were exposed to famine in early gestation (and were heavier at birth) had a more atherogenic lipid profile⁽³⁵⁾, a higher BMI^(35,36) and typically had

higher risk of CHD^(37,38). The prevalence of obstructive airways disease was increased in people exposed to famine in mid-gestation (OR=1.7; 95% CI 1.1, 2.6) and non-significantly higher in those exposed in early gestation (OR=1.5; 95% CI 0.9, 2.6)⁽³⁹⁾. Thus, alterations in fetal nutritional supply can drive developmental adaptations influencing growth and metabolism. These results, and ours, demonstrate how nutritional challenges periconceptionally and in early gestation can influence maternal and perinatal outcomes.

A limitation of the present study includes the relative small sample size of asthmatic women, which is reflected in the relatively large confidence intervals generated in the multivariate analysis. As such, it is not possible to rule out a smaller, yet still clinically significant difference between the dietary patterns and asthma control, beyond those observed in the study. Therefore, the full effect of maternal diet on asthma control is still worthy of investigation in subsequent larger studies. The FFQ is a reliable tool covering long-term food intake and the FFQ used in the current study has been validated in women of childbearing age⁽⁴⁰⁾, as well as in young⁽⁴¹⁾ and older⁽⁴²⁾ adults. However, use of the questionnaire also has limitations including memory recall, and several details of dietary intake are not measured; thus quantification of intake is not as accurate as with repeated 24 h recalls or food records. Inaccuracies may also result from an incomplete listing of all possible foods and from errors in frequency and usual serving size estimations. In addition, although women were informed of the dietary intake period to cover, it is possible that current (i.e. first trimester) dietary intakes might have influenced the records. However, as this was a retrospective report about dietary intakes prior to pregnancy, a food frequency approach was necessary. Unpublished data from our 24 h recalls, which were collected throughout gestation, indicated that similar foods (i.e. fruits, takeaways, butter, cereal-based dishes, vegetables) were consumed by a high percentage of women. In the literature, there is limited information on diet before pregnancy; however, the few studies that have been published reported minimal differences in dietary patterns just prior to and during pregnancy^(30,31), in which one of these studies identified the dietary patterns persisted 6 months postpartum⁽³¹⁾. Strengths of the present study include the detailed assessment of asthma control and the use of lung function tests to confirm asthma status. Further studies have the potential to expand on these findings by assessing dietary patterns and the relationship to asthma exacerbations, and conducting randomised controlled trials in asthmatic pregnant women to assess the positive effects of dietary manipulation on asthma outcomes.

Dietary intake is an important modifiable risk factor for asthma and our data suggest that nutrition pre-pregnancy influences maternal asthma control during pregnancy. This work highlights the importance of promoting a healthy diet that is low in saturated fat, sugar and

takeaway foods, and therefore higher in lean meats, poultry and fish, as well as fruits, vegetables and whole grains. This dietary pattern needs to be encouraged to all asthmatic women who are of childbearing age, and should additionally be promoted during pregnancy and beyond.

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Supplementary material

To view supplementary material for this article, please visit <http://dx.doi.org/10.1017/S1368980015001226>

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