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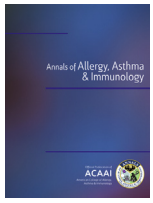

Review article

Association between Western diet pattern and adult asthma: a focused review

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ABSTRACT

Objective: Radical changes in diet have paralleled the increase in asthma with shifts toward a "Western" diet pattern, characterized by the high intake of processed meats and refined grains, high-fat dairy products, and sugary desserts and drinks. Because diet represents a modifiable risk factor in numerous chronic diseases, the authors examined the association between consumption of a Western diet pattern and asthma incidence, prevalence, and morbidity in adults.

Data Sources: PubMed, Cochrane, Web of Science, and Scopus were searched for peer-reviewed publications published from January 1980 to April 2014.

Study Selection: Studies retrieved for inclusion assessed dietary patterns representative of a Western diet and asthma incidence, prevalence, respiratory symptoms, and lung function.

Results: Ten observational studies conducted in North American, European, and Asian countries, ranging from 153 to more than 70,000 individuals, did not provide evidence to support an association between a Western dietary pattern and asthma incidence and prevalence. Five of these studies also investigated asthma morbidity, with variable findings.

Conclusion: Current evidence does not support an association between a Western diet and incident or prevalent adult asthma but does suggest a possible link between a Western diet pattern and adult asthma morbidity.

Introduction

Asthma is a global concern affecting an estimated 300 million people worldwide.[1] Asthma accounts for approximately 15 million disability-adjusted life years lost each year and approximately 1 in every 250 deaths worldwide.[1] More affluent countries and those transitioning to a Westernized lifestyle bear a disproportionate burden of disease and the causes for these patterns in asthma prevalence and morbidity remain unknown.[1,2] As the urban population grows with continued adoption of a Western lifestyle, an additional 100 million asthma diagnoses are anticipated by 2025.[1] Identification of modifiable risk factors for incident asthma and asthma morbidity are needed to support interventions aimed at decreasing the burden of this disease.

The change in diet associated with Westernization and economic growth is a potential contributing factor to asthma burden.[3] Increased access to prepackaged, highly processed, "convenience" foods has been linked to multiple chronic diseases, including cancer[4,5] and cardiovascular disease.[6,7] These population-level dietary changes have occurred in parallel with the increasing burden of asthma.[8–10] This coincidence of epidemics has led to interest in the potential role of diet in asthma incidence, prevalence, and morbidity.

A common approach to assessing diet has been to focus on single foods and nutrients. Individual "anti-inflammatory" and "antioxidant" vitamins have demonstrated some positive association with improved asthma or decreased wheeze in observational studies.[11] However, other studies have shown inconsistent or negative results with single-micronutrient supplementation for prevention or improvement of asthma.[11] Instead of focusing on individual dietary components, assessment of dietary patterns represents an alternative approach for the evaluation of combinations of foods. This

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approach lowers the likelihood that associations observed with single foods and nutrients are confounded by other foods or nutrients that are commonly consumed with the nutrients of interest. Furthermore, the effect of single nutrients or food items might be too small to detect, whereas the effect of a dietary pattern could be large enough to create an impact at the population level.[12] The evidence from dietary pattern studies also might be more easily translated to the general public because people eat a combination of foods rather than single foods or nutrients. Given associations between dietary patterns and other chronic diseases, it is important to consider dietary pattern analysis in the study of asthma.[13–16]

Research has focused on favorable dietary patterns, such as a "prudent" diet pattern, consisting of higher levels of fruits, vegetables, whole grains, poultry, and fish, and a "Mediterranean" diet pattern. The Mediterranean diet pattern can be defined as a variation of the prudent diet pattern that contains foods common to the Mediterranean, such as olive oil, legumes, fish, and wine in moderation. A countering question is whether an unfavorable diet pattern exists that increases the likelihood of developing asthma or worsens existing asthma. The authors reviewed the evidence as to whether a "Western" diet pattern, typified by a higher intake of processed foods (eg, meats and refined grains), high-fat dairy products, and sugary desserts and drinks, is associated with asthma incidence, prevalence, and symptoms or morbidity in adult populations.

Methods

Data Sources

The PubMed, Cochrane, Web of Science, and Scopus databases were searched for studies published in English from January 1980 to April 2014 that contained data on the effects of a Western diet pattern, or fast foods or processed foods considered exemplary of a Western diet, on adult asthma. Searches used the following medical subject heading terms and/or text words: *asthma* or *lung allergy* and *dietary fats*, or *fast foods*, or *Western diet*, or *refined grain*, or *processed food*, or *standard American diet*, or *Mediterranean diet*, or *dietary patterns*, or *high fat*, and combined with *diet*.

Results

After screening retrieved citations and reviewing additional references from select bibliographies, 10 full-text studies were identified that met the search criteria (Table 1).[17–26] All studies were observational and included cross-sectional,[19,21,22] cross-sectional with a subset followed longitudinally,[20] prospective cohort,[18,23–26] and matched case–control[17] studies. Sample sizes ranged from 153 to more than 70,000 participants and studies were conducted in Asia,[22,18] North America,[24] and Europe.[17,19–21,23,26]

Exposure and Outcome Assessment of Included Studies

Exposure (diet) and outcome (asthma incidence, prevalence, and symptoms or morbidity) assessment varied considerably among studies (Table 1). Most studies (8 of 10) used food frequency questionnaires (FFQs) or questions abstracted from an FFQ and principal components analysis with or without factor analysis dietary pattern analyses.[17–20,22,24–26] Two studies evaluated prepackaged and fast food intake rather than dietary patterns using simple, short questionnaires regarding frequency of food consumption and reported results based on these frequencies.[21,23] One study combined approaches, completing an evaluation of fast food individually and dietary pattern.[22]

Asthma diagnosis and/or symptoms were almost exclusively defined by self-report[17–19,21–26] and were confirmed with medication usage in the majority.[17–19,21–25,26] Although all studies adjusted for age, sex, and smoking, other confounders of interest (body mass index, socioeconomic status, physical activity level) were variably examined. The heterogeneity in the study methods, particularly related to dietary assessment, precluded a quantitative analysis integrating study findings but allowed for a qualitative assessment. All 10 of the included studies evaluated the outcome of incident or prevalent asthma, whereas only 5 of the studies[17,19,20,22,26] examined the link between a Western diet pattern and asthma symptoms or morbidity.

Western Diet and Incident or Prevalent Asthma

Nine of the 10 studies showed no association between a Western diet and asthma incidence or prevalence.[17–19,20,22–26] Despite the presence of large cohorts (representing >70,000 persons) in the retrieved results, only 1 study (N = 717) in Albania reported a significant association between the intake of processed foods at least once a week, consistent with a Western diet, and asthma symptoms thought to be indicative of the presence of asthma (odds ratio [OR] 1.6–1.8, $P < .05$).[21] The definition of asthma was based on symptoms or reported prior diagnosis and prescribed medication with or without atopy, determined by a positive skin test result. A limitation of this study was that certain known potential confounders, including body mass index and socioeconomic status, were not addressed in the analysis.[21] Notably, adjustment for confounders varied considerably among the included studies (Table 1).

Two of the largest studies were prospective US cohorts that included adult-onset asthma as a secondary outcome (primary outcome was the incidence of chronic obstructive pulmonary disease).[24,25] The 2 studies used FFQs and principal components analysis for the creation of Western and prudent diet patterns, although the study populations differed by age and sex, with women 30 to 55 years old included in one cohort and[24] and men 40 to 75 years old in the other.[25] Neither study reported an association between a Western dietary pattern and adult-onset asthma.

The findings in the US studies that did not suggest an association between a Western dietary pattern and incident asthma are corroborated by the results of 2 studies in East Asia.[18,22] The largest, a study of 52,325 Chinese Singaporeans, investigated exposure to a "meat-dim sum" dietary pattern that, although not entirely typical of a Western diet given the different ethnic ingredients, contained numerous commonalities with a Western dietary pattern. Findings demonstrated no significant association between adherence to a meat-dim sum diet and the incidence of asthma.[18] The second study consisted of 153 female university students in Japan (15.7% with asthma) and found no association between fast food intake, assessed as part of questions abstracted from a FFQ, and asthma prevalence.[22]

Five European studies reported consistently negative results for the association of a Western diet with asthma.[17,19,20,23,26] A French cohort of 56,672 women (the E3N Study) were followed prospectively for 10 years and investigation showed 3 diet patterns: "Western," "prudent," and "nuts and wine." However, no association was found between a Western diet pattern and history of asthma, asthma prevalence, or asthma incidence.[26] A Swedish study of 8,150 adults also showed no association between fast food intake and asthma prevalence.[23] Similarly, a study of 12,648 adults in the Netherlands showed no association between a "refined-foods" diet and asthma prevalence (OR 1.1, 95% confidence interval [CI] 0.8–1.6).[20] In the United Kingdom, 5,999 asthma cases and 854 controls were assessed,[17] and a Western dietary pattern was not found to be associated with asthma. A European cohort of 1,174 adults showed no association between a "meat-and-potato" pattern (which the researchers noted is similar to a Western diet) and asthma prevalence.[19] Two additional large studies were identified that did not meet our a priori inclusion criteria (examination of Western diet pattern or fast foods/processed foods), but assessed dietary components commonly included in the Western dietary pattern.[27,28] A study in Australia (n=156,035) linked a meat/cheese

Table 1
Summary of studies examining associations between a Western diet pattern (via comprehensive dietary assessment or as represented by fast food or processed food intake) and adult asthma

Study	Location	Study population	Dietary assessment	Dietary analyses	Asthma assessment	Adjustments	Prevalence/incidence	Symptoms/morbidity
Priftanji et al,[21] 2002 Cross-sectional study of factors associated with asthma	Albania	717 adults (20–44 y old)	question regarding frequency of prepackaged food intake	binary scale (consumption <1 vs >1×/wk)	self-report: asthma attack, nighttime awakening for shortness of breath, or current treatment	age, sex, smoking	prepackaged food intake was associated with possible allergic asthma (OR 1.61, 95% CI 1.01–2.55) and possible nonallergic asthma (OR 1.82, 95% CI 1.17–2.83)	
Butler et al,[18] 2006 Prospective study of dietary patterns and persistent cough with phlegm	Singapore	52,325 adults (45–74 y old)	165-item FFQ	PCA	self-report: incident asthma confirmed by symptom and medication report	age, energy intake, dialect group, sex, smoking status, age at smoking initiation, cigarettes/d, secondhand smoke, education; BMI explored and not correlated with dietary pattern	adherence to a diet rich in meat, sodium, and refined carbohydrates was not significantly associated with incident asthma	
Varraso et al,[24] 2007 Prospective study of dietary patterns and chronic obstructive pulmonary disease	United States	72,043 women (30–55 y old)	116-item FFQ recording intake during previous 12 mo	PCA-FA	self-report: physician diagnosis and use of medication for asthma within previous 12 mo	age, race/ethnicity, energy intake, spouse's educational attainment, supplement use, physical activity, smoking, secondhand smoke, menopausal status, physician visits, US region	no association was found between Western diet pattern and adult-onset asthma	
Varraso et al,[25] 2007 Prospective study of dietary patterns and chronic obstructive pulmonary disease	United States	42,917 men (40–75 y old)	131-item FFQ	PCA-FA	self-report: physician diagnosis and use of medication for asthma within previous 12 mo	age, race/ethnicity, energy intake, smoking, supplement use, BMI, physical activity, physician visits, US region	no association was found between Western diet pattern and adult-onset asthma	
Uddenfeldt et al,[23] 2010 Long-term follow-up study of diet and asthma in 3 age groups	Sweden	8,150 adults (16, 30–39, 60–69 y old)	7 questions about consumption of foods	consumption score by frequency, summary score for fruit and fish	self-report: asthma or use of asthma medication	age, sex, smoking, socioeconomic group, BMI heredity, hay fever, GERD, snoring, physical activity, building dampness	fast food intake was not associated with incident asthma	

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Table 1 (*continued*)

Study	Location	Study population	Dietary assessment	Dietary analyses	Asthma assessment	Adjustments	Prevalence/incidence	Symptoms/morbidity
Takaoka and Norback,[22] 2008 Cross-sectional study of diet and asthmatic symptoms, infections, pollen and furry pet allergy	Japan	153 female university students (mean age 21 y)	11 questions abstracted from a FFQ and a question on type of oil used for cooking	FFQ (intake) scales and PCA-FA	self-report: "current asthma"—current asthma medication use or attack in previous 12 mo	age, smoking, parental asthma/allergy	no association was found between any dietary factors (including fast food) and asthma prevalence	fast food intake was associated with wheeze (OR 1.68, 95% CI 1.02–2.78) and nocturnal breathlessness (OR 2.26, 95% CI 1.06–4.79); a diet pattern score, including fast food, juice, and soft drinks, was related to wheeze (OR 1.19, 95% CI 1.04–1.37)
Varraso et al,[26] 2009 Prospective study of dietary patterns and asthma in E3N Study	France	54,672 women (40–65 y old)	French FFQ	PCA-FA	self-report: history of asthma attacks and, when available, self-reported physician diagnosis	age, energy intake, smoking status, years of education, hay fever, supplement use, BMI, physical activity, menopausal status	no association of dietary patterns was found with asthma incidence, ever asthma, or current asthma	Western diet pattern was associated with increased odds of frequent ($\geq$1/wk) asthma attacks only in those with asthma not taking vitamin supplements (OR 1.56–1.79, $P < .05$)
Hooper et al,[19] 2010 Cross-sectional study of dietary patterns and asthma in residents of 3 countries in European Community Respiratory Health Survey-–II	Europe	1,174 adults (29–55 y old)	German and UK FFQs recording intake during previous 12 mo and supplemental questions using local food tables	PCA	self-report: asthma or nocturnal shortness of breath, attack in previous 12 mo, or asthma medication use	age, sex, social class, smoking status, exercise, BMI, energy intake	diet pattern was not associated with asthma prevalence	meat-and-potato diet (similar to Western diet) was associated with increased per-quintile asthma symptoms in the United Kingdom (ratio 1.34, 95% CI 1.09–1.67) and Norway (ratio 1.24, 95% CI 1.00–1.55), but decreased per-quintile asthma symptoms in Germany (ratio 0.81, 95% CI 0.68–0.97)
McKeever et al,[20] 2010 Combined cross-sectional and longitudinal study of diet and relation to respiratory disease and lung function	Netherlands	cross-sectional, 12,648 adults (20–59 y old); longitudinal, 2,911 adults	178-item FFQ	PCA-FA	physician-diagnosed asthma	age, sex, educational level, smoking status, pack-years height, BMI	a refined diet pattern (similar to a Western diet pattern) was not associated with asthma prevalence	a refined diet pattern (similar to a Western diet pattern) showed a trend for increased wheeze prevalence across increasing quintiles of intake ($P = .07$)

Bakolis et al,[17] 2010 Population-based case–control study of dietary patterns and asthma	United Kingdom	599 cases, 854 controls (16–50 y old)	>200-item FFQ	PCA	self-report: asthma attack, waking, shortness of breath in previous 12 mo, asthma medication use	age, sex, ethnicity, BMI, social class, housing tenure, employment status, single parent, smoking, secondhand smoke, energy intake, number of siblings, paracetamol use, supplement use	no association was found between a Western dietary pattern and asthma prevalence after adjustment	no association was found between a Western dietary pattern and nocturnal asthma symptoms or asthma quality-of-life score

Abbreviations: BMI, body mass index; CI, confidence interval; FFQ, food frequency questionnaire; GERD, gastroesophageal reflux disease; OR, odds ratio; PCA, principal components analysis; PCA-FA, principal components analysis and factor analysis.

factor in men and a meat factor in women with increased odds of prevalent asthma/hay fever.[28] A study in China (n=1486) investigating intake of monosodium glutamate also assessed dietary patterns, though features of the Western diet were divided among two patterns, neither of which was associated with prevalent asthma.[28]

Western Diet and Asthma Morbidity

Five of the studies examined the association between a Western diet pattern and asthma morbidity, typically assessed with symptom outcomes.[17,19,20,22,26] Studies ranged from 153 to more than 50,000 participants and were conducted in Asia and Europe. Three of the 5 studies suggested an association between a Western diet pattern and asthma symptoms,[20,22,26] although 1 study found no association[17] and 1 study showed conflicting results.[19]

Two of 4 European studies reported a positive association between a Western diet and asthma morbidity.[20,26] The E3N Study in France demonstrated an association between adherence to a Western diet pattern and increased risk of frequent asthma attacks (OR 1.79, 95% CI 1.11 to 3.73) in those not taking multivitamin supplements, but not in the entire cohort.[26] In a study of more than 12,000 adults in the Netherlands, higher quintiles of refined-food intake (similar to a Western diet pattern) demonstrated a nonsignificant trend toward increased prevalence of wheeze (P for trend = .07).[20] In a subset (n = 2,911), participants were followed longitudinally to evaluate the effect of diet on lung function over 5 years of follow-up. Interestingly, those in the highest quintile of refined-food intake had increased lung function decline (forced expiratory volume in 1 second −48.5 mL, 95% CI −80.7 to −16.3) compared with the lowest quintile during the 5-year study period, approximating an additional loss of 10 mL in forced expiratory volume in 1 second annually.[20] Notably, those with a higher intake of the refined-food pattern in the full cohort tended to be current smokers, and the prevalence of current smokers was lower in the longitudinal subset than in the cross-sectional cohort. Although multivariable models adjusted for smoking, there remains a possibility of residual confounding.

Two smaller European studies (n = 1,000–2,000) were representative of multiple countries and reported negative or conflicting results.[17,19] Hooper et al[19] found no association between the meat-and-potatoes diet pattern (similar to a Western diet according to the investigators) and asthma symptoms examined in the entire cohort (OR 1.07, 95% CI 0.84 to 1.36) or between diet pattern and forced expiratory volume in 1 second (coefficient −0.01, 95% CI −0.04 to 0.03) or bronchial hyperreactivity (coefficient −0.08, 95% CI −0.27 to 0.10). The study included participants in 3 European countries, and when the investigators stratified by site, increasing quintiles of meat-and-potato dietary intake emerged as a significant predictor of increased asthma symptoms in the United Kingdom (OR 1.34, 95% CI 1.09–1.67, P = .007) and Norway (OR 1.24, 95% CI 1.00–1.55, P = .051), but was protective in Germany (OR 0.81, 95% CI 0.68–0.97, P = .025). The investigators concluded that regional variation could represent uncontrolled confounding among centers. Indeed, the exact composition of each dietary pattern varied among centers due in part to the use of country-specific FFQs. However, an additional study with more than 6,000 participants performed in the United Kingdom showed no relation between the Western diet pattern and asthma quality-of-life scores or nighttime awakening with asthma symptoms.[17]

A Japanese study (n = 153) by Takaoka and Norback[22] found that fast food intake was associated with increased odds of wheeze (OR 1.68, 95% CI 1.02 to 2.78) and nocturnal breathlessness (OR 2.26, 95% CI 1.06 to 4.79) and a dietary pattern score, including fast food, juice, and soft drinks, was related to the prevalence of wheeze (OR 1.19, 95% CI 1.04 to 1.37). The analysis was performed in all

university students who responded to a distributed questionnaire, which introduces the possibility of selection bias. Of those who responded, 15.7% reported a history of physician-diagnosed asthma and only 3.3% reported current asthma medication use.[22] The authors further investigated the association between diet and an asthma symptom score[29] based on the number of positive answers to 8 questions. Fast food intake was associated with worse asthma symptom scores ($P < .05$), and the fast food, juice, and soft drink diet pattern score showed a trend toward worse asthma symptom scores ($P = .06$).[22]

Discussion

Results of the present literature review do not support an association asthma prevalence or incidence in adult populations but suggest a possible link between a Western diet and asthma morbidity in adults. There was substantial heterogeneity among studies in methodology, study size, and geographic region. Furthermore, as diet varies by study region, the exact composition of the Western diet pattern and its comparison also varies between studies. Although these issues create challenges in directly comparing studies and informing overall effect, the breadth of populations represented in these dietary pattern analyses is perhaps a strength of the literature. Furthermore, the use of dietary patterns could increase applicability of results within each geographic region of interest.

Mechanistic Rationale

There are several pathways by which a Western diet pattern could influence adult asthma. Because these pathways are not mutually exclusive, it is plausible that they could combine to create a multiplicative proinflammatory response in patients with asthma as proposed in those with other chronic diseases.[14]

The Western diet pattern is rich in proinflammatory foods and poor in antioxidant foods, which could influence responses to environmental exposures. Omega-6 fatty acids, in excess in processed and fast food products found in the Western diet pattern, are converted into arachidonic acid, which is converted further into leukotrienes and prostaglandins (mediators of inflammation).[30] For example, prostaglandin E2 suppresses T-helper cell type 1 and promotes the T-helper cell type 2 phenotype, which is prominent in allergic asthma.[30] Furthermore, low levels of antioxidants can fail to prevent activation of nuclear factor-κB through elevated concentrations of reactive oxygen species,[31,32] thereby activating the innate immune response through cytokine release and destruction of cellular components such as DNA, proteins, and lipids.[33] These pathways plausibly could prime the lung for increased reactivity to environmental stimuli, characteristic of the asthmatic response.

Furthermore, processed foods, prevalent in the Western diet, are high in preservatives and additives. Although there is inconclusive evidence as to whether salt contributes to all types of asthma, previous studies of allergic and exercise-induced asthma have suggested a trend toward improvement in lung function with low salt intake.[34,35] Other processing chemicals can trigger asthma through proinflammatory reactions, such as non-azo dyes, sodium benzoate:parahydroxybenzoic acid,[36] and sulfur dioxide.[37] These compounds also can act as food allergens,[38] which may increase the respiratory morbidity of individuals with asthma.[39]

An alternative pathway by which a Western diet might affect asthma is through the gut microbiome. Food affects the composition of bacteria in the gut, which in turn affects the processing of nutrients.[40,41] Protection against allergic disease is dependent on the maintenance of immune tolerance, which is at least in part regulated by interactions between the gut microbiome and gut epithelial cells, antigen presenting cells, and regulatory T cells.[42–46] For example, a recent study demonstrated a compelling link between soluble fiber intake, changes in the gut microbiome, and improvements in lung inflammation and airway hyperreactivity in a murine model.[47]

The obesity epidemic has closely paralleled an increasing asthma prevalence and transition to a Western diet pattern and could represent a mediator on the pathway from diet to asthma. Adipose tissue is metabolically active and capable of releasing proinflammatory cytokines and adipokines, such as interleukin-6, tumor necrosis factor-α, and C-reactive protein.[48] Studies have demonstrated links between increased fat content and cytokine and adipokine expression (eg, tumor necrosis factor-α, IL-6, leptin) and increased asthma, although conflicting results have led to hypotheses that serum levels might not accurately represent biologic activity in the lungs.[49,50] In addition, there is evidence that increases in body mass could be linked to increased airway hyperresponsiveness.[51] It is possible that increases in body mass mediate the effects of diet on asthma and therefore it is necessary to account for body composition in studies of diet. Notably, 3 of the 10 total studies[21,22,24] (although only 1 of 5 studies examining symptoms or morbidity[22]) failed to control for an anthropometric measurement (Table 1).

Alternative Approach: A "Healthy" Diet

Although this review has focused on the effects of a Western dietary pattern on asthma, in many of the studies discussed, a favorable dietary pattern served as a comparison group.[17–19,20,22,24–26] Evidence relating a healthier diet to improved asthma symptoms or morbidity is more robust and is supported by at least 2 completed prospective trials. A recent randomized controlled trial of the Mediterranean diet in 38 adults with symptomatic asthma showed promising improvements in quality of life and spirometry.[52] The investigators concluded that a larger study powered to detect clinical end points is necessary. Another trial found a decreased risk of asthma exacerbation with a high antioxidant diet, changes effected primarily through whole food intervention (fruit and vegetables intake).[53] An additional pilot study of the Dietary Approaches to Stop Hypertension diet (high in fresh fruit, vegetables, nuts, and antioxidants) is being launched in an adult population to determine the effects on poorly controlled asthma.[54] Although these studies will provide information on the effects of healthy diets on asthma, they also will indirectly estimate effects of the absence of these foods in a Western diet.

Challenges of Dietary (Exposure) and Asthma (Outcome) Assessment

Measuring the complex exposure of diet is challenging. The assessment of diet in a small or large population requires proper tools. Diet information can be assessed retrospectively by recall of varying periods (during the past 3 days, week, or year) or prospectively by food diaries. Each has inherent flaws. Retrospective collection depends on the recollection of the participant, the training and experience of the interviewer, and the applicability of the questionnaire to the population in question. Prospective food diaries depend on the reliability, comprehension, and accuracy of the participant. Investigators must appropriately gauge their population to identify the most suitable method of data collection. After data are collected, the choice of an a priori (less specific to a population, more comparable across studies) versus an a posteriori (more specific to a population, less comparable across studies) approach significantly affects data presentation.

In addition, comparison of diet across countries is complicated by several issues. Potentially different compositions of food can retain the same label. For example, bread in Germany might be composed primarily of unprocessed rye, whereas bread in the United Kingdom might be composed primarily of refined wheat or white flour. Furthermore, unmeasured foods can be co-ingested

with measured foods differentially by location. For example, sauerkraut is commonly consumed with meat dishes in Germany but not in the United Kingdom. Because the intake of fermented foods such as sauerkraut might be associated with decreased atopy,[55,56] meat intake theoretically could appear falsely protective if sauerkraut intake is not recorded or accurately determined. This concern also highlights the importance of variation in food preparation techniques, because boiled cabbage could have different implications for asthma and atopy than fermented cabbage. These complexities could partly explain the differential effects of the meat-and-potato diet in Germany vs the United Kingdom on asthma morbidity noted by Hooper et al,[19] illustrating how regional differences in food composition and preparation can modify effect estimates. In addition, there are inherent difficulties in assessing aspects of the diet that are not food, such as supplements and nutraceuticals, which can contribute substantially to nutrient intake. Moreover, it is critical to use standardized and validated approaches to measuring asthma morbidity that will allow comparison of results between study populations and allow detection of meaningful differences.

Challenge of Temporality

The timing of dietary assessment in relation to asthma incidence and prevalence complicates the assessment of an association. It is unclear whether there is a lag between exposure and development of asthma, what that lag could be, and the best time to assess diet in relation to diagnosis. Although prior longitudinal studies performed in adult cohorts have demonstrated reasonable tracking of diet,[57] given the number of possible confounding factors in the diet–asthma relationship, it is possible that signal dilution occurs easily. This might be less of an issue with asthma symptoms and morbidity in those who already have the diagnosis, given the proposed proinflammatory consequences outlined earlier.

Conclusions

Although current evidence does not support a link between a Western diet and incident or prevalent asthma, some studies have suggested that a Western diet pattern could contribute to asthma symptoms in adults. The current evidence base assessing the association between a Western dietary pattern and asthma morbidity is limited. Future studies assessing relations between dietary patterns and asthma morbidity outcomes, such as symptoms, lung function, and health care use, are needed. Further, inclusion of respiratory and systemic biomarkers in such studies is critical to determine the mechanistic underpinnings of the link between Western dietary patterns and respiratory health effects. Clinical trials have the potential to provide the strongest evidence of the effects of dietary modification on asthma morbidity and studies in the US population are needed given the radical dietary shifts that have occurred in recent decades and the increasing asthma prevalence. The benefits of avoidance of foods consistent with a Western diet pattern are well known for cardiovascular health; however, further research is needed to define the effects of a Western diet pattern on pulmonary, and specifically asthma, morbidity.

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References

[1] Masoli M, Fabian D, Holt S, Beasley R. Global Initiative for Asthma (GINA) Program. The global burden of asthma: executive summary of the GINA Dissemination Committee report. *Allergy*. 2004;59:469–478.
[2] Anandan C, Nurmatov U, van Schayck O, Sheikh A. Is the prevalence of asthma declining? Systematic review of epidemiological studies. *Allergy*. 2010;65:152–167.
[3] Kearney J. Food consumption trends and drivers. *Phil Trans R Soc*. 2010;365:2793–2807.
[4] Tseng M, Breslow RA, DeVellis RF, Ziegler RG. Dietary patterns and prostate cancer risk in the National Health and Nutrition Examination Survey Epidemiological Follow-up Study cohort. *Cancer Epidemiol Biomarkers Prev*. 2004;13:71–77.
[5] Kolahdooz F, Ibiebele TI, van der Pols JC, Webb PM. Dietary patterns and ovarian cancer risk. *Am J Clin Nutr*. 2009;89:297–304.
[6] Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med*. 1997;336:1117–1124.
[7] Fung TT, Willett WC, Stampfer MJ, Manson JE, Hu FB. Dietary patterns and the risk of coronary heart disease in women. *Arch Intern Med*. 2001;161:1857–1862.
[8] Kim JH, Ellwood PE, Asher MI. PMC2703624; Diet and asthma: looking back, moving forward. *Respir Res*. 2009;10:49.
[9] von Mutius E. The rising trends in asthma and allergic disease. *Clin Exp Allergy*. 1998;28(suppl 5):45–51.
[10] Eder W, Ege MJ, von Mutius E. The asthma epidemic. *N Engl J Med*. 2006;355:2226–2235.
[11] Varraso R. Nutrition and asthma. *Curr Allergy Asthma Rep*. 2012;12:201–210.
[12] Jacques PF, Tucker KL. Are dietary patterns useful for understanding the role of diet in chronic disease? *Am J Clin Nutr*. 2001;73:1–2.
[13] Hu FB, Rimm EB, Stampfer MJ, Ascherio A, Spiegelman D, Willett WC. Prospective study of major dietary patterns and risk of coronary heart disease in men. *Am J Clin Nutr*. 2000;72:912–921.
[14] Manzel A, Muller DN, Hafler DA, Erdman SE, Linker RA, Kleinewietfeld M. Role of "Western diet" in inflammatory autoimmune diseases. *Curr Allergy Asthma Rep*. 2014;14:404.
[15] Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities Study. *Circulation*. 2008;117:754–761.
[16] Gao X, Chen H, Fung TT, et al. Prospective study of dietary pattern and risk of Parkinson disease. *Am J Clin Nutr*. 2007;86:1486–1494.
[17] Bakolis I, Hooper R, Thompson RL, Shaheen SO. Dietary patterns and adult asthma: population-based case–control study. *Allergy*. 2010;65:606–615.
[18] Butler L, Koh W, Lee H, Tseng M, Yu M, London S. Prospective study of dietary patterns and persistent cough with phlegm among Chinese Singaporeans. *Am J Respir Crit Care Med*. 2006;173:264–270.
[19] Hooper R, Heinrich J, Omenaas E, et al. Dietary patterns and risk of asthma: results from three countries in European Community Respiratory Health Survey–II. *Br J Nutr*. 2010;103:1354–1365.
[20] McKeever TM, Lewis SA, Cassano PA, et al. Patterns of dietary intake and relation to respiratory disease, forced expiratory volume in 1 s, and decline in 5-y forced expiratory volume. *Am J Clin Nutr*. 2010;92:408–415.
[21] Priftanji AV, Qirko E, Burr ML, Layzell JCM, Williams KL. Factors associated with asthma in Albania. *Eur J Allergy Clin Immunol*. 2002;57:123–128.
[22] Takaoka M, Norback D. Diet among Japanese female university students and asthmatic symptoms, infections, pollen and furry pet allergy. *Respir Med*. 2008;102:1045–1054.
[23] Uddenfeldt M, Janson C, Lampa E, et al. High BMI is related to higher incidence of asthma, while a fish and fruit diet is related to a lower—results from a long-term follow-up study of three age groups in Sweden. *Respir Med*. 2010;104:972–980.
[24] Varraso R, Fung TT, Barr RG, Hu FB, Willett W, Camargo CA. Prospective study of dietary patterns and chronic obstructive pulmonary disease among US women. *Am J Clin Nutr*. 2007;86:488–495.
[25] Varraso R, Fung TT, Hu FB, Willett W, Camargo CA. Prospective study of dietary patterns and chronic obstructive pulmonary disease among US men. *Thorax*. 2007;62:786–791.
[26] Varraso R, Kauffmann F, Leynaert B, et al. Dietary patterns and asthma in the E3N study. *Eur Respir J*. 2009;33:33–41.
[27] Rosenkranz RR, Rosenkranz SK, Neessen KJJ. Dietary factors associated with lifetime asthma or hayfever diagnosis in Australian middle-aged and older adults: a cross-sectional study. *Nutr J*. 2012;11:1–12.
[28] Shi Z, Yuan B, Wittert GA, et al. Monosodium glutamate intake, dietary patterns and asthma in Chinese adults. *PlosOne*. 2012;7:e51567.
[29] Pekkanen J, Sunyer J, Anto JM, Burney P. European Community Respiratory Health Study. Operational definitions of asthma in studies on its aetiology. *Eur Respir J*. 2005;26:28–35.
[30] Black PN, Sharpe S. Dietary fat and asthma: is there a connection? *Eur Respir J*. 1997;10:6–12.
[31] Schreck R, Albermann K, Baeuerle PA. Nuclear factor kappa B: an oxidative stress-responsive transcription factor of eukaryotic cells (a review). *Free Radic Res Commun*. 1992;17:221–237.
[32] Blackwell TS, Christman JW. The role of nuclear factor-kappa B in cytokine gene regulation. *Am J Respir Cell Mol Biol*. 1997;17:3–9.
[33] Zaknun D, Schroecksnadel S, Kurz K, Fuchs D. Potential role of antioxidant food supplements, preservatives and colorants in the pathogenesis of allergy and asthma. *Int Arch Allergy Immunol*. 2012;157:113–124.
[34] Ardern KD. Dietary salt reduction or exclusion for allergic asthma. *Cochrane Database Syst Rev*. 2004;3:CD000436.

[35] Pogson Z, McKeever T. Dietary sodium manipulation and asthma. *Cochrane Database Syst Rev*. 2011;3:CD000436.
[36] Weber RW, Hoffman M, Raine DA Jr, Nelson HS. Incidence of bronchoconstriction due to aspirin, azo dyes, non-azo dyes, and preservatives in a population of perennial asthmatics. *J Allergy Clin Immunol*. 1979;64:32–37.
[37] Freedman BJ. Sulphur dioxide in foods and beverages: its use as a preservative and its effect on asthma. *Br J Dis Chest*. 1980;74:128–134.
[38] Ibero M, Eseverri JL, Barroso C, Botey J. Dyes, preservatives and salicylates in the induction of food intolerance and/or hypersensitivity in children. *Allergol Immunopathol (Madr)*. 1982;10:263–268.
[39] Kewalramani A, Bollinger ME. The impact of food allergy on asthma. *J Asthma Allergy*. 2010;3:65–74.
[40] Kau AL, Ahern PP, Griffin NW, Goodman AL, Gordon JI. Human nutrition, the gut microbiome and the immune system. *Nature*. 2011;474:327–336.
[41] Muegge BD, Kuczynski J, Knights D, et al. Diet drives convergence in gut microbiome functions across mammalian phylogeny and within humans. *Science*. 2011;332:970–974.
[42] Chakir J, Shannon J, Molet S, et al. Airway remodeling-associated mediators in moderate to severe asthma: effect of steroids on TGF-beta, IL-11, IL-17, and type I and type III collagen expression. *J Allergy Clin Immunol*. 2003;111:1293–1298.
[43] Molet S, Hamid Q, Davoine F, et al. IL-17 is increased in asthmatic airways and induces human bronchial fibroblasts to produce cytokines. *J Allergy Clin Immunol*. 2001;108:430–438.
[44] Lee YK, Menezes JS, Umesaki Y, Mazmanian SK. Proinflammatory T-cell responses to gut microbiota promote experimental autoimmune encephalomyelitis. *Proc Natl Acad Sci U S A*. 2011;108(suppl 1):4615–4622.
[45] Atarashi K, Tanoue T, Oshima K, et al. Treg induction by a rationally selected mixture of Clostridia strains from the human microbiota. *Nature*. 2013;500:232–236.
[46] Esplugues E, Huber S, Gagliani N, et al. Control of TH17 cells occurs in the small intestine. *Nature*. 2011;475:514–518.
[47] Trompette A, Gollwitzer ES, Yadava K, et al. Gut microbiota metabolism of dietary fiber influences allergic airway disease and hematopoiesis. *Nat Med*. 2014;20:159–166.
[48] Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. *Nat Rev Immunol*. 2011;11:85–97.
[49] Ali Assad N, Sood A. Leptin, adiponectin and pulmonary diseases. *Biochimie*. 2012;94:2180–2189.
[50] Lugogo NL, Kraft M, Dixon AE. Does obesity produce a distinct asthma phenotype? *J Appl Physiol*. 2010;108:729–734.
[51] Sideleva O, Suratt BT, Black KE, et al. Obesity and asthma: an inflammatory disease of adipose tissue not the airway. *Am J Respir Crit Care Med*. 2012;186:598–605.
[52] Sexton P, Black P, Metcalf P, et al. Influence of Mediterranean diet on asthma symptoms, lung function, and systemic inflammation: a randomized controlled trial. *J Asthma*. 2013;50:75–81.
[53] Wood LG, Garg ML, Smart JM, Scott HA, Barker D, Gibson PG. Manipulating antioxidant intake in asthma: a randomized controlled trial. *Am J Clin Nutr*. 2012;96:534–543.
[54] Ma J, Strub P, Lavori PW, et al. DASH for asthma: a pilot study of the DASH diet in not–well-controlled adult asthma. *Contemp Clin Trials*. 2013;35:55–67.
[55] Alm JS, Swartz J, Lilja G, Scheynius A, Pershagen G. Atopy in children of families with an anthroposophic lifestyle. *Lancet*. 1999;353:1485–1488.
[56] Cross ML, Stevenson LM, Gill HS. Anti-allergy properties of fermented foods: an important immunoregulatory mechanism of lactic acid bacteria? *Int Immunopharmacol*. 2001;1:891–901.
[57] Willett WC. Recall of remote diet. In: Willett WC, ed. *Nutritional Epidemiology*. New York: Oxford University Press; 1998:148–156.