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ORIGINAL ARTICLE **Epidemiology of Allergic Disease**

Fast food consumption counters the protective effect of breastfeeding on asthma in children?

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Summary

Background Fast food consumption and childhood asthma have rapidly increased in recent decades. During the same period there has been an increased rate of prolonged breastfeeding.
Objective To evaluate if fast food consumption was associated with asthma in children, and if the proposed protective effect of breastfeeding on asthma was altered by fast food consumption.
Methods This case–control study included 246 children with allergist-diagnosed asthma and 477 non-asthmatic controls at age 8–10 years. Information on fast food consumption and exclusive breastfeeding was obtained from questionnaire data. The association between asthma and fast food consumption was evaluated. Asthma in relation to exclusive breastfeeding was also evaluated, taking into account fast food consumption as a modifying factor.
Results Children with asthma were more likely to consume fast food than children without asthma [crude odds ratio (OR) 1.70, 95% confidence interval (CI) 1.23–2.34]. In comparison to prolonged exclusive breastfeeding ($\geqslant 12$ weeks), asthma was positively associated with short-term exclusive breastfeeding (< 12 weeks) in children who never or occasionally consumed fast food (crude OR 1.84, 95% CI 1.09–3.11), but not in children who frequently consumed fast food (crude OR 1.07, 95% CI 0.72–1.61). The *P*-value for this interaction (0.109) was borderline. Children with high fast food consumption who were exclusively breastfed < 12 weeks as infants, had greater than a twofold risk of asthma compared with infants who had been exclusively breastfed for a longer time period and who did not become high consumers of fast food in later childhood. These findings were not affected after final adjustment of confounders and covariates.
Conclusion Fast food consumption is associated with asthma in children and potentially counteracts the protective effect of prolonged breastfeeding on asthma. This may explain the paradoxical phenomenon of parallel increased rates of prolonged breastfeeding and asthma in children.

Keywords asthma, breastfeeding, fast food consumption, sex
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Introduction

The recent epidemic of asthma and allergy has been attributed to a package of changes in the Western lifestyle, including diet [1]. One aspect of this changed diet has been frequent consumption of fast food [2]. The consumption of fast food in children has increased from 2% of total energy in the late 1970s to 10% in the 1990s [3]. Fast food has been associated with wheezing symptoms in children, particularly in boys [4, 5], but the relationship between fast food consumption and asthma is not confirmed yet.

Over the same time period we have seen increased rates of prolonged breastfeeding [6, 7]. Prolonged breastfeeding has been associated with reduced risk of asthma in toddlers, pre-schoolchildren and pre-adolescents [8–10], although results are not conclusive [11, 12]. Despite the

proposed protective effect of breastfeeding on asthma, we are puzzled by the parallel increased rates of prolonged breastfeeding and childhood asthma in recent years [13]. In this study, we hypothesized that fast food consumption was a modifying factor in the relationship between prolonged breastfeeding and asthma in children, i.e. the protective effect of breastfeeding may be altered by fast food consumption.

Methods

This was a nested case–control study of the Study of Asthma, Genes and the Environment (SAGE) project, which comprised 246 children with paediatric allergist-diagnosed asthma and 477 non-asthmatic controls at 8–10 years of age. Subjects' recruitment and response were detailed in a flow chart in our previous study [14]. In short, a one-page survey on child health was sent to families of all children born in 1995 in the province of Manitoba, Canada ($n = 12\,566$), who were identified from a provincial health registry. All children with parent-reported asthma in the past 12 months ($n = 388$) were selected for recruitment out of 3598 participants. Control children ($n = 450$) were randomly selected from the remaining participants stratified by family income (low/high) and residence location (urban/rural) to ensure a balanced representation. Selected children were subsequently invited to undergo a clinical assessment. Following clinical assessment for asthma by a paediatric allergist (A. B. or J. L.), there were ultimately 246 children with allergist-diagnosed asthma and 477 children without asthma among those who participated in the clinical examination ($n = 723$). Eighty percent of the children with allergist-diagnosed asthma were from the parent-reported asthma group, and the other 20% were from the initial control children. In allergist-confirmed control children, the proportion from the initial control children and from the parent-reported asthma group was 81% and 19%, respectively. Doctor diagnosis of asthma in our study was based on the Canadian Asthma Consensus Guidelines in which symptoms and variable airway obstruction were two major criteria [15]. To confirm the asthma diagnosis, paediatric allergists conducted a physical examination for chest symptoms (hyperinflation, wheeze, prolonged expiration and decreased breath sounds) and a standardized history, including questions on cough, wheeze, shortness of breath, response to current medications (i.e. bronchodilators, corticosteroids), and the presence of other allergic conditions (e.g. allergic rhinitis, atopic dermatitis and food allergies). Survey respondents were representative of the full birth cohort children in terms of child sex, child asthma status and history of maternal asthma [14]. Children who underwent the clinical examination were also not different from survey respondents in terms of these characteristics, indicating that non-response bias would not pose a problem in this study.

Information on fast food consumption and exclusive breastfeeding was obtained from questionnaires answered by the parents of participants at the clinical examination. Fast food consumption was defined as 'eating burgers/fastfood' more than once or twice per week on average in the last 12 months compared with children who never or occasionally ate burgers/fastfood. The information of exclusive breastfeeding was derived from a question on the time of introduction of formula/cow's milk, 'when did your child first have any formula/cow's milk?' Exclusive breastfeeding was categorized as a dichotomous variable in our previous study [10], i.e. exclusive breastfeeding < 12 weeks and exclusive breastfeeding $\geqslant 12$ weeks. The cut-off of 12 weeks coincided with the value used in a meta-analysis of studies on the effect of breastfeeding on asthma development [16]. The reliability of maternal report of breastfeeding was studied in the previous study [10] by comparison with information on breastfeeding collected after birth in a sample of 40 SAGE children who had also participated in a prospective intervention study in 1995 [17]. Sixty-five percent of this survey information was in agreement with the prospective information using the 12-week cut-off value. In addition, there was no significant difference in the percentage of agreement between asthmatic and control children, indicating that there were no systematic recall errors [10]. The study was approved by the Health Research Ethics Board, University of Manitoba.

Statistics

The association between asthma and fast food consumption was determined in all children and by gender (Table 1). In addition, the association between asthma and exclusive breastfeeding < 12 weeks was assessed in children who consumed fast food vs. children who did not or occasionally consumed fast food. The interaction between exclusive breastfeeding and fast food consumption in relation to asthma was formally tested. Contrasts were also implemented to test the effect of fast food consumption in children with exclusive breastfeeding $\geqslant 12$ weeks vs. children with exclusive breastfeeding < 12 weeks. The odds ratio (OR) for each category of exclusive breastfeeding in combination with fast food consumption was obtained from logistic regression analyses, using exclusive breastfeeding $\geqslant 12$ weeks and no fast food consumption as the reference group (Table 2). Maternal asthma, sex, urban/rural location, First Nations origin and exclusive breastfeeding were associated with asthma at a significance level of $P < 0.1$ [14], whereas overweight (BMI $\geqslant$ 85th percentile of age–sex specific [14, 18] or low family income ($<$ CAD$ 30 000 annual) was not associated with asthma ($P > 0.2$ for both). First Nations origin

Table 1. The association between fast food consumption and asthma: a comparison between asthma cases and controls

	Cases		Controls		Crude OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR* (95% CI)
	n	%	*n*	%			
All (cases/controls = 243/472)							
Fast food	160	65.8	251	53.2	1.70 (1.23–2.34)	1.63 (1.17–2.27)	1.59 (1.13–2.25)
Boys (cases/controls = 146/252)							
Fast food	100	68.5	130	51.6	2.04 (1.33–3.13)	1.97 (1.26–3.09)	2.06 (1.29–3.30)
Girls (cases/controls = 97/220)							
Fast food	60	61.9	121	55.0	1.33 (0.81–2.16)	1.32 (0.80–2.18)	1.32 (0.78–2.23)

Fast food consumption: eating burgers/fastfood more than once or twice per week in the last 12 months. There were missing values on fast food consumption in eight children. Adjusted OR: First Nations origin and exclusive breastfeeding were adjusted as confounders in the multivariate logistic regression analyses.

*Adjusted OR: additional adjustments were made including sex, maternal asthma, location, overweight and family income besides the adjustment for First Nations origin and exclusive breastfeeding.

CI, confidence interval; OR, odds ratio.

Table 2. The interactive role of exclusive breastfeeding and fast food consumption: a comparison between asthma cases and controls in all children, and in boys and girls separately

	Exclusive breastfeeding (weeks)	Fast food	Cases		Controls		Crude OR (95% CI)	Adjusted OR (95% CI)	Adjusted OR* (95% CI)
			n	%	*n*	%			
All	⩾12	No	42	18.0	137	30.5	1.00	1.00	1.00
	<12	No	39	16.7	69	15.4	1.84 (1.09–3.11)	1.95 (1.15–3.30)	1.87 (1.08–3.23)
	⩾12	Yes	76	32.5	125	27.8	1.98 (1.27–3.10)	2.03 (1.30–3.19)	1.96 (1.23–3.11)
	<12	Yes	77	32.9	118	26.3	2.13 (1.36–3.34)	2.41 (1.52–3.82)	2.32 (1.44–3.73)
Boys	⩾12	No	21	15.1	73	30.8	1.00	1.00	1.00
	<12	No	24	17.3	39	16.5	2.14 (1.06–4.32)	2.19 (1.08–4.44)	2.09 (1.00–4.34)
	⩾12	Yes	44	31.7	67	28.3	2.28 (1.23–4.23)	2.34 (1.26–4.35)	2.24 (1.17–4.27)
	<12	Yes	50	36.0	58	24.5	3.00 (1.62–5.54)	3.55 (1.88–6.71)	3.91 (2.01–7.61)
Girls	⩾12	No	21	22.1	64	30.2	1.00	1.00	1.00
	<12	No	15	15.8	30	14.2	1.52 (0.69–3.36)	1.67 (0.75–3.73)	1.53 (0.65–3.59)
	⩾12	Yes	32	33.7	58	27.4	1.68 (0.87–3.24)	1.73 (0.89–3.33)	1.83 (0.92–3.64)
	<12	Yes	27	28.4	60	28.3	1.37 (0.70–2.68)	1.50 (0.76–2.96)	1.26 (0.61–2.57)

Information on exclusive breastfeeding or fast food consumption was missing in 40 children. Adjusted OR: First Nations origin was adjusted as a confounder in the multivariate logistic regression analyses.

*Adjusted OR: additional adjustments were made including sex, maternal asthma, location, overweight and family income besides the adjustment for First Nations origin.

CI, confidence interval; OR, odds ratio.

and exclusive breastfeeding were associated with fast food consumption in the control children without asthma at a significance level of $P<0.01$, whereas maternal asthma, sex, location, overweight or family income were not ($P\geqslant 0.5$). First Nations origin and exclusive breastfeeding were adjusted as the confounding factors in the relationship between fast food consumption and asthma (Table 1), as the two factors were significantly associated with both the outcome (asthma) and the exposure (fast food consumption) variables [19]. First Nations origin was thus the only confounding factor adjusted in the relationship between asthma and exclusive breastfeeding in combination with fast food consumption (Table 2). Previous studies have also shown that Aboriginal people are less likely to be breastfed for a longer period, and more likely to consume fast food and have chronic diseases [20, 21]. All statistical analyses were performed with STATA, release 9.0 (College Station, TX, USA).

Results

Children with asthma were more likely to consume fast food than children without asthma (65.8% vs. 53.2%, $P=0.001$). An association between asthma and fast food consumption was found in boys but not in girls (Table 1), but the difference between sexes did not reach statistical significance ($P_{\text{interaction term}}=0.19$). The adjustment for the confounding factors (exclusive breastfeeding and First

Nations origin) did not change the significant association between asthma and fast food consumption [adjusted OR 1.63, 95% confidence interval (CI) 1.17–2.27]. When additional adjustment was performed for sex, maternal asthma, location, overweight and family income, the association between fast food consumption and asthma remained (adjusted OR 1.59, 95% CI 1.13–2.25).

Asthma was significantly associated with exclusive breastfeeding <12 weeks in children who never or occasionally consumed fast food (crude OR 1.84, Table 2, $P=0.022$). However, asthma was not significantly associated with exclusive breastfeeding <12 weeks in children who consumed fast food (crude OR 1.07, 95% CI 0.72–1.61, $P=0.732$, contrast statement). There was a suggestion of an interaction between exclusive breastfeeding and fast food consumption in relation to asthma ($P_{\text{interaction term}}=0.109$). Among children who were exclusively breastfed $\geqslant 12$ weeks, fast food consumption was significantly associated with an increased risk of asthma (crude OR 1.98, Table 2), but it was not associated with a risk of asthma in children who were exclusively breastfed <12 weeks (crude OR 1.15, 95% CI 0.71–1.88). In addition, exclusive breastfeeding <12 weeks with fast food consumption had the highest risk of asthma (crude OR 2.13, Table 2) as compared with exclusive breastfeeding $\geqslant 12$ weeks with no fast food consumption. The adjustment for the confounding factor (First Nations origin) did not affect the results (Table 2). Nor were the results affected by also adding sex, maternal asthma, location, overweight or family income in the final adjustment. After the final adjustment, asthma was significantly associated with exclusive breastfeeding <12 weeks in children who never or occasionally consumed fast food (adjusted OR 1.87, Table 2); it continued not to be associated with exclusive breastfeeding <12 weeks in the presence of fast food consumption (adjusted OR 1.19, 95% CI 0.76–1.84, contrast statement). After stratification by sex in the final adjustment, asthma was associated with a significantly increased OR (adjusted OR 2.09, Table 2) for exclusive breastfeeding <12 weeks in boys who never or occasionally consumed fast food, and with a non-significantly increased OR (adjusted OR 1.53, Table 2) in girls who never or occasionally consumed fast food.

Discussion

We found that paediatric allergist-diagnosed asthma was significantly associated with fast food consumption in children, which was in keeping with previous studies that reported an association between fast food intake and childhood wheezing symptoms [4, 5]. Our findings also hint an interaction between breastfeeding and fast food consumption in relation to asthma. Asthma was positively and significantly associated with short-term exclusive breastfeeding in children who never or occasionally consumed fast food. This association disappeared in those children who frequently consumed fast food. Moreover, these associations did not differ statistically between boys and girls.

Our study contributes evidence to the association between fast food consumption and childhood asthma [22], but a cause–effect relationship cannot be established due to the retrospective study design. However, the findings are biological plausible. Fast food contains a high sodium content that can increase the risk for bronchial hyperresponsiveness and wheezing symptoms in children [23]. The high fat content in fast foods and the lack of antioxidants may also play a role in the pathogenesis of asthma [24, 25].

To our knowledge this is the first study to evaluate the interaction between breastfeeding and fast food consumption in relation to asthma. Prolonged breastfeeding has been shown to have a protective effect on asthma in young children [8, 9]. Our study suggests that the protective effect of long-term breastfeeding on asthma may be negated by fast food consumption. While fast food consumption may be a proxy for many other factors, the counteractive effect of fast food intake could not be explained by the status of First Nations origin, sex, maternal asthma, location, overweight or family income in our study. Long duration of breastfeeding may exert its protective effect against asthma via the reduction of lower respiratory infections, modulation of T-helper type-1 (Th1) and Th2 balance or other mechanisms during the first years of life and later on [26–28]. In addition, breast milk compared with cow's milk and home-prepared foods contains low sodium [29]. Frequent fast food consumption may diminish this protection and even act as an independent risk factor for asthma through the lack of antioxidant properties or its high salt and fatty acid content [23–25]. Further research is warranted to study if there are common pathophysiologic mechanisms involved in the counteractive effect between prolonged breastfeeding and fast food consumption.

We acknowledge several limitations in our study. The survey response was modest, but survey participants did not differ from the cohort population in terms of child sex, child asthma status and maternal asthma. Control children (without parent-reported asthma) were representative in terms of urban and rural location, and family income. Location, family income and other important factors were also included in the final model adjustments. Data on exclusive breastfeeding and fast food consumption were obtained retrospectively, with the potential for recall bias. However, we determined recalled information on breastfeeding to be well correlated with prospectively collected data on breastfeeding in a subset of children in a previous study [10]. Information on fast food consumption was ascertained for the past year, but we assumed a similar pattern of fast food consumption in previous years,

consistent with longitudinal studies which show fast food intake to be constant in children until adolescence, at which time rates start to increase [30]. As children's liking for foods high in fat is shaped at an early age through exposure to foods present and consumed by family members, it is conceivable that schoolchildren who frequently consume fast food were more likely to be exposed to fast food in early life [31].

We interpret the marginal *P*-value of interaction term as being suggestive of an interaction between exclusive breastfeeding and fast food consumption. This is supported by our ability to detect an almost twofold increased risk of asthma subsequent to short-term exclusive breastfeeding in the small group of children without fast food consumption ($n = 287$). However, the association lost its statistical significance in the presence of fast food consumption, even though this group of children was larger in size ($n = 396$). Secondly, after full adjustment of confounders and covariates, short-term exclusive breastfeeding remained to be associated with asthma among children who never or occasionally consumed fast food, but this increased risk of asthma was not seen in children who consumed fast food. Further, children with high fast food consumption who were exclusively breastfed < 12 weeks as infants, had the highest risk of asthma compared with infants who had been exclusively breastfed for a longer time period and who did not become high consumers of fast food as they grew older.

Finally, our study was strengthened by the careful classification of cases and controls. Asthma was diagnosed on clinical investigation by one of two paediatric allergists (A. B. or J. L.) and was based on a standardized history and physical examination. We undertook this careful phenotyping in order to minimize misclassification that may be more of a problem with a questionnaire-based approach. However, due to the retrospective case–control study design, cautions should be taken when generalizing our results. We hope that our study will stimulate further research in this area to either confirm or dispute our findings and interpretations.

In conclusion, we have shown that fast food consumption is associated with asthma in children. We speculate that fast food consumption may counter the protective effect of prolonged breastfeeding on asthma. This may explain the observed paradoxical phenomenon of parallel increased rates of prolonged breastfeeding and asthma in recent decades.

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