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ORIGINAL ARTICLE

Breastfeeding is protective to diabetes risk in young adults: a longitudinal study

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Abstract
Aim It is unclear whether any breastfeeding or a certain duration of breastfeeding is protective against the development of diabetes in adult offspring.
Methods We followed a sub-sample of 3,595 offspring born in the Mater Hospital in Brisbane, Australia between 1981 and 1983 and for whom we had doctor diagnosed self-reported diabetes at age 21 years and maternal reported duration of breastfeeding at 6-month post-natal follow-up. Multiple logistic regression was used to examine the independent associations of duration of breastfeeding (never breastfeed, breastfed <4 months and breastfed ≥4 months) with offspring diabetes by age 21 years.
Results Of 3,595 young adults, 45 (1.25 %) developed diabetes by age 21 years. The odds ratio of experiencing diabetes was 0.58 (95 % CI 0.29, 1.16) for offspring who were breastfed <4 months, and it was 0.29 (95 % CI 0.13, 0.63), for offspring who were breastfed at least 4 months compared to the never breastfed offspring. Adjusting for potential confounding and mediating factors including maternal age, education, pre-pregnancy body mass index (BMI), smoking, offspring sports, TV and their BMI at 21 years did not substantially alter this association.
Conclusions Findings of this study suggest that infants who are breastfed for longer than 4 months have a substantial protective effect against the development of diabetes in young adulthood, which is independent of current BMI. Promoting breastfeeding for a minimum of 4 months may be a useful strategy for the prevention of diabetes among young adults.

Keywords Breastfeeding · Duration · Diabetes · Young adults

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Introduction

The sharply increasing prevalence of diabetes mellitus (DM) in most countries around the world over the last few decades represents a serious public health crisis for the twenty-first century. It is already the fourth or fifth leading cause of death in most developed countries, and there is substantial evidence that it is epidemic in many economically developing and newly industrialized countries [1]. There is consistent evidence that the increasing prevalence of overweight and obesity is the major driver of the DM epidemic [1, 2]. Considerable attention has focused on lifestyle factors such as healthy diets and regular physical activity to reduce overweight, obesity and DM.

One of the most important natural sources of early nutrition for infants is breast milk. Studies provide conflicting

results regarding the association between breastfeeding and DM. Some studies have shown that children who are breastfed are less likely to be obese in the short and long term [3, 4] and have a lower risk of developing DM [5]. Others, including a randomised controlled trial (randomization to breastfeeding promotion), found no association between breastfeeding and childhood adiposity, body fat and insulin-like growth factor [6, 7]. A systematic review and meta-analysis by Owen et al. [8] demonstrated that breastfeeding in infancy was associated with a reduced risk [odds ratio (OR) 0.61, 95 % CI 0.44, 0.85] of type 2 diabetes in later life. In their meta-analysis, seven individual studies were included, of which five reported no association [9–13] and two studies reported a positive association [14, 15]. In 2007, the World Health Organization (WHO) conducted a systematic review and meta-analysis to provide evidence on the long-term effects of breastfeeding [16]. They reported that individuals who were breastfed experienced lower mean blood pressure and total cholesterol and reduced risk of obesity. Although their pooled estimate in assessing the risk of type 2 diabetes reported a similar protective effect of breastfeeding as did Owen et al., recent studies provide conflicting findings with some showing no association [15] and others reporting positive associations [17, 18]. The association between breastfeeding and offspring DM remains inconclusive. These inconclusive results may reflect the fact that in most studies that have examined the association between breastfeeding and type 2 diabetes assessed breastfeeding as a simple dichotomous variable: breastfed and not breastfed. Therefore, these studies have not been able to take into account duration and intensity of breastfeeding, to accurately assess the association.

Most mothers intend to breastfeed and initiate exclusive breastfeeding although breastfeeding rates quickly plummet over time for various reasons [19]. At present, only 15–17 % of women in Australia and US typically breastfeed for the recommended duration of at least 6 months [20, 21]. Therefore, any potential benefits of breastfeeding are limited by inadequate duration in infancy. Most studies have not been able to examine the association between duration of breastfeeding and any future protective effect against adult diabetes in offspring. This study examined the association between the duration of breastfeeding and the risk of diabetes by the age of 21 years using a large community-based birth cohort study in Australia.

Methods

The study

The data used for this study came from the Mater-University of Queensland Study of Pregnancy (MUSP) cohort. MUSP is a large birth cohort study with a prospective follow-up of 7,223 mother–child pairs. Mothers were on average 18 weeks of gestation at the recruitment during the period 1981–1983 at the Mater Hospital in Brisbane, Australia. These mothers and their live singleton babies who were born at the study hospital and were not adopted before leaving hospital constituted the original mother–offspring cohort. Mother–offspring pairs were prospectively followed up at 3–5 post-delivery, 6-month, 5, 14 and 21 years post-partum. The current study is limited to a subsample of 3,595 offspring for whom we had information regarding doctor diagnosed self-reported diabetes at age 21 years and maternal reported duration of breastfeeding at the 6-month post-natal follow-up.

Each phase of the MUSP study was approved by the ethics committees of the University of Queensland and Mater Hospitals. We have also collected written informed consent from the mothers at all data collection phases of the study. At 14 and 21 years, offspring provided their own consent. Full details of the study participants and measurements have been previously reported [22, 23].

Measurements

Diabetes

At the 21-year follow-up, we used a self-administered questionnaire where respondent was asked “Have you EVER been told by a doctor that you have diabetes (high blood sugars)?” with response options “yes” or “no”. No information was collected regarding type of diabetes and current therapy for diabetes in this study.

Breastfeeding

Duration of breastfeeding was collected at 6-month follow-up of the mothers. At the ‘6-month’ follow-up time, some mothers were in fact between 4 and 6 months post-delivery (none were seen earlier than 4 months post-delivery). At the 6-month follow-up, mother was asked, “How long did you breastfeed?” with response options, “still breastfeeding”, “4–6 months”, “7 weeks to 3 months”, “2 weeks or less” and “not at all”. Because women were at least 4-months post-delivery when they responded to this question, there is a clear overlap between the last two categories and we therefore combined these into one category—breastfed for >4 months. To increase the statistical precision of the analyses, we have categorized breastfeeding as never, <4 months or ≥4 months. In 3–5 days post-delivery, mothers were asked, “Are you breastfeeding or intending to breastfeed your baby?” with response option “No” or “Yes”. We used this information as a sensitivity test of ever versus never breastfed.

Confounders or mediators

The potential confounders or mediators for the association between breastfeeding and diabetes were selected on the basis of a priori knowledge and recommended practice in epidemiology [24]. In the MUSP, available potential confounders are maternal age (in years), maternal educational attainment (did not complete secondary education, completed secondary education and completed further or higher education), parental ethnic origins (Caucasian, Asian, Aboriginal–Islander), maternal cigarette smoking during pregnancy (non smoker, 1–19 cigarettes per day, 20+ cigarettes per day), birth weight for gestation, parity (1, 2, 3 or more), hypertensive disorder in pregnancy and maternal pre-pregnancy body mass index (BMI).

The potential available mediators were adolescent television watching (weekdays/weekend: <1, 1–<3, 3–<5 and 5 or more h/day), sport participation (4–7; 0–3 days/week) and having meals together in the family (at least once a day, few times/once/less than once a week). Offspring BMI and waist circumference at 21 years were considered potential mediators. Offspring's height was measured to the nearest centimetre without shoes using a portable stadiometer (Road Rod 214 Portable Stadiometer, USA). Weight was measured with a scale accurate to 0.2 kg being used. The mean of the two measures of weight and height were used in all analyses. We used the WHO classification of BMI cutoffs [25]. Waist circumference (in cm) was measured using a standard protocol. We categorized waist circumference for males: <94 cm as normal, 94–<102 cm as overweight and 102 or more cm as obese; and for females: <80 cm as normal, 80–<88 cm as overweight and 88 or more cm as obese [26]. Details of measurements of all these confounders or mediators have been described and presented elsewhere [27, 28].

Statistical analysis

We compared the characteristics of respondents who provided information on breastfeeding at 6 month and diabetes at 21 years versus those who did not respond. We used the Chi-square test to assess statistical significance when the characteristics were categorical and *F* test when continuous. As we found no statistically significant difference of the association between breastfeeding and diabetes by male and female offspring, males and females were combined for all analyses.

We applied multiple imputation to impute missing items of the 3,595 sub-sample. All the multivariable analyses were conducted for the multiple imputation data to increase the statistical precision [29]. We used a series of multiple logistic regression to estimate the OR of experiencing diabetes by the age of 21 years, with adjustment for potential confounding and mediating factors. In Model 1, we did not adjusted for any factor other than breastfeeding duration. Model 2 was additionally adjusted for the potential confounding factors including maternal age, maternal educational levels, maternal smoking during pregnancy, parity, birth weight z-score for gestation, maternal pre-pregnancy BMI and hypertensive disorder in pregnancy. Model 3 was additionally adjusted for the mediating factors including adolescent television watching, participation in sports, and family meals. In the final model, we have further adjusted for offspring BMI at 21 years. Similarly, we repeated this analysis using a series of logistic regression to examine whether any breastfeeding duration (i.e. never breastfeed vs. breastfeed) predicted offspring diabetes at 21 years.

All analyses were undertaken using Stata version 12.0 (Stata inc., Texas).

Results

We found that participants who provided information compared to those who did not provide information about breastfeeding at 6-month follow-up and DM at 21 years were more like to be older, better educated, with higher incomes, Caucasian, non smokers during pregnancy, less likely to be of 3+ parity, more likely to be breastfed and have higher *Z*-score of birth weight for gestational age (Table 1). This was not associated with the pre-pregnancy BMI or hypertensive disorder in pregnancy.

Intention to breastfeed or breastfeeding at 3–5 days post-delivery and duration of breastfeeding reported at 6-month follow-up and the prevalence of diabetes by 21 years are presented in Table 2. Overall, in women who intended to breastfeed or breastfed at 3–5 days post-delivery, 95 % reported "ever breastfed" and 5 % reported "never breastfed" at the 6-months follow-up. To increase the statistical precision, we have presented main results for breastfeeding as never, <4 months or 4 months or more. Of 3,595 participants, 636 (17.69 %) were never breastfed, 1,366 (38.00 %) were breastfed <4 months, and the rest 1,593 (44.31 %) were breastfed for at least 4 months. By 21 years, 45 (1.25 %) young adults were found to have experienced diabetes. Duration of breastfeeding and offspring diabetes by 21 years is statistically significant (p value <0.05).

The risk of diabetes by the duration of breastfeeding is presented in Table 3. In the confounder-adjusted model (model 2 in Table 3), offspring who were breastfed <4 months had an OR for diabetes of 0.58 (95 % CI 0.29, 1.16) and for those who breastfed ≥4 months, the OR for diabetes was 0.30 (95 % CI 0.13, 0.70) by 21 years compared to the offspring who were never breastfed. This

Table 1 Comparison of the respondent characteristics who have been included versus those who have been excluded in the main analyses

Background factors	Respondents who provided information on breastfeeding at 6 m and diabetes at 21 year ($n = 3,595$)	Respondents who did not provide information on breastfeeding at 6 m and diabetes at 21 year ($n = 3,594$)	p value
Maternal education			
Did not complete secondary education	564 (15.79)	733 (20.57)	
Completed secondary education	2,283 (63.90)	2,307 (64.75)	
Completed further or higher education	726 (20.32)	523 (14.68)	<0.001
Family income at first clinic visit per year			
AUS $ <10,400	996 (29.17)	1,297 (39.28)	
AUS $ >10,399	2,419 (70.83)	2,005 (60.72)	<0.001
Maternal smoking during pregnancy			
None smoker	2,378 (66.74)	2,022 (56.80)	
1–19 cigarettes per day	934 (26.21)	1,174 (32.98)	
20+ cigarettes per day	251 (7.04)	364 (10.22)	<0.001
Parity			
1	1,459 (40.63)	1,468 (40.87)	
2	1,143 (31.83)	1,064 (29.62)	
3 or more	989 (27.54)	1,060 (29.51)	0.010
Race			
Caucasian	3,240 (92.70)	2,992 (85.95)	
Asian	127 (3.63)	178 (5.11)	
Aboriginal–Islander	128 (3.66)	311 (8.93)	<0.001
Hypertensive disorder in pregnancy			
Yes	327 (9.11)	303 (8.44)	
No	3,161 (90.89)	3,289 (91.56)	0.31
Duration of breastfeeding			
Never	636 (17.69)	794 (26.14)	
<4 months	1,366 (38.00)	1,224 (40.30)	
4 or more months	1,593 (44.31)	1,019 (33.55)	<0.001
Age at first clinic visit, mean (SD)	25.56 (5.02)	24.48 (5.14)	<0.001
Pre-pregnancy BMI kg/m^2, mean (SD)	21.95 (3.99)	21.83 (3.94)	0.21
Z-score of birth weight for gestational age	0.05 (0.97)	−0.02 (1.00)	0.01

* p indicates the significance level of the difference by characteristics of participants who have been included in the analyses versus participants who were not included in the analysis. We used an F test for a continuous data and a Chi-squared test for categorical data

association remains consistent even adjusting for TV watching, sports participation and family meals (model 3) in adolescent and current BMI at 21 years (model 4).

To examine whether any duration of breastfeeding is protective of young adult diabetes, we have repeated the same analysis as in Table 4 categorizing breastfeeding as never breastfed and breastfed. In the confounder-adjusted model (model 2 in Table 4), for offspring who were breastfed, the OR for diabetes was 0.45 (95 % CI 0.23, 0.87) by 21 years compared to the offspring who were never breastfed. This association was not explained by mediating factors including young adults current BMI or WC.

In further analysis, when we examined the association between breastfeeding intention or breastfeeding at 3–5 days post-delivery and offspring diabetes by 21 years, the association was robust and consistent.

Discussion

This study of the long-term protective effect of the duration of breastfeeding on the development of diabetes for young adult offspring is unique and timely because of the emerging public health concerns regarding overweight, obesity and diabetes in young adults. Using a long follow-up of a large community-based birth cohort study, we found that children who were ever breastfeed had a 58 % lower risk of diabetes by age 21 years compared to offspring who were never breastfed. This risk was much lower

Table 2 Breastfeeding and prevalence of diabetes

Breastfeeding duration	*n* (%)	*n* (%) diabetes by 21 years	*p* value*
Are you breastfeeding or intending to breastfeed your baby (3–5 days post-delivery)?			
No	623 (16.80)	16 (2.73)	
Yes	3,086 (83.20)	27 (0.90)	<0.001
Breastfeeding duration (at 6-month follow-up)			
Never	636 (16.69)	15 (2.36)	
2 weeks or less	379 (10.54)	8 (2.11)	
3–6 weeks	459 (12.77)	6 (1.31)	0.023
7 weeks to 3 months	528 (14.69)	5 (0.95)	
4–6 months	379 (10.46)	3 (0.80)	
Still breastfeeding	1,217 (33.85)	8 (0.66)	
Breastfeeding duration in three categories			
Never	636 (16.69)	15 (2.36)	
<4 months	1,366 (38.00)	19 (1.39)	0.005
4 or more months	1,593 (44.31)	11 (0.69)	

* *p* indicates the significance level of the difference between breastfeeding duration and diabetes prevalence using a Chi-squared test

Table 3 Odds ratio (95 % confidence interval) of diabetes among young adult offspring at 21 years by the duration of breastfeeding ($N = 3{,}595$)

Model	Odds ratio of having diabetes at 21 years (95 % CI)		
	Did not breastfed	<4 months	4 or more months
Model 1	1.00	0.58 (0.29, 1.16)	0.29 (0.13, 0.63)
Model 2	1.00	0.58 (0.29, 1.16)	0.30 (0.13, 0.70)
Model 3	1.00	0.58 (0.29, 1.17)	0.31 (0.13, 0.72)
Model 4	1.00	0.60 (0.30, 1.22)	0.32 (0.14, 0.75)

Model 1: unadjusted

Model 2: adjusted for maternal age, smoking during pregnancy, maternal education, parity, birth weight *z*-score for gestation, maternal pre-pregnancy BMI and hypertensive disorder in pregnancy

Model 3: model 2 plus adolescent TV watching, sport participation and family meals

Model 4: model 3 plus offspring BMI at 21 years

(71 %) for those children who were breastfed for at least 4 months. Potential important confounders including birth weight z-score for gestation and maternal pre-pregnancy obesity or mediating factors including adolescent television watching, sport participation and 21 years BMI did not explain this prospective association. The findings of this study confirm the suggestion that breastfeeding may have a long-term protective effect against the development of diabetes in young adults.

Results of this study support the evidence provided by the systematic review and meta-analyses [8, 16]. Two meta-analyses reported that for offspring who were ever

Table 4 Odds ratio (95 % confidence interval) of diabetes among young adult offspring at 21 years by those who were never breastfed and those who were breastfed

Model	Odds ratio of having diabetes at 21 years (95 % CI)	
	Never breastfed	Breastfed
Model 1	1.00	0.42 (0.23, 0.79)
Model 2	1.00	0.45 (0.23, 0.87)
Model 3	1.00	0.45 (0.23, 0.88)
Model 4	1.00	0.47 (0.24, 0.92)

Model 1: unadjusted

Model 2: adjusted for maternal age, smoking during pregnancy, maternal education, parity, birth weight *z*-score for gestation, maternal pre-pregnancy BMI and hypertensive disorder in pregnancy

Model 3: model 2 plus adolescent TV watching, sport participation and family meals

Model 4: model 3 plus offspring BMI at 21 years

breastfed the OR for diabetes was 0.61 (95 % CI 0.44, 0.85) [8] and 0.63 (95 % CI 0.45–0.89) [16], respectively. These results are quite consistent with our own estimate of adjusted OR 0.47 (95 % CI 0.24, 0.92). This small difference could be mainly due to heterogeneity of the studies in the meta-analyses. This may include different age groups of women and offspring, prospective versus retrospective collection of breastfeeding data, self-reported versus clinical diagnosis of type 2 diabetes, and consideration of the varying confounding factors. Additionally, we have shown an association between being breastfed for at least 4 months and a much lower risk of diabetes (OR 0.32, 95 % CI 0.14, 0.75) compared to no breastfeeding. Importantly, this association was robust and independent of a range of confounding or mediating factors including offspring current obesity status.

There are multiple pathways including biological, environmental and health behavioural which could explain why breastfeeding is protective for offspring diabetes. Human breast milk contains substances including γ-linolenic acid (GLA), arachidonic acid (AA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) that promote the maturation of the immune system which protect again developing diabetes [30]. One possible mechanism for the protective effects of breastfeeding on offspring diabetes risk is the pathway of breastfeeding being protective for obesity. This may be mediated by bioactive substances in breast milk that enhance satiety and energy balance, preventing excess weight gain during childhood and thus protecting against the development of type 2 diabetes [31]. However, recent systematic reviews and meta-analyses have suggested that the effect of breastfeeding on obesity prevalence and mean BMI is small [31, 32]. This weak relationship between breastfeeding and obesity could be the reason that when adjusted for current BMI, the

association between breastfeeding and young adult diabetes remains robust. Alternatively, it is entirely likely that much of the diabetes reported by adults at 21 years was type 1 diabetes, that may or may not be obesity induced. In this case, the role of breastfeeding and the prevention of type 1 diabetes need to be further explored. However, the role of obesity as an intermediate mechanism remains uncertain [32]. Another mechanism could be that breastfed mothers are health conscious and lead a healthy life style during their child's development. Subsequently their offspring might also adopt that healthy lifestyle and as a result be at lower risk to experience diabetes. However, adjustment for adolescents TV watching, sports participation and family meals together did not attenuate the associations.

This study has a few notable limitations. The interpretation of the results is limited by the measurement of both breastfeeding and young adult diabetes. The breastfeeding variable did not distinguish full from partial breastfeeding (i.e. supplementing breastfeeding with infant formula or solid foods) and the breastfeeding duration variable was not aligned with the WHO/UNICEF recommendation of 6 months exclusive breastfeeding [33]. However, the rates of breastfeeding seen in this study (Table 1) are consistent with another Australian study at similar time period [34]. Diabetes for the young adults was self-reported though we did ask them whether it was doctor diagnosed. It was unclear whether the participants were reporting type 1 or type 2 diabetes, although we mentioned high sugar level while asking the question. As diabetes was not clinically diagnosed, it is not possible to know whether this protective role of breastfeeding is for type 1 diabetes and/or type 2. The prevalence of diabetes at 21 years was 1.25 %. We found no other Australia based study that reported prevalence of diabetes for the same age group. The closest we found was the Busselton study that reported the prevalence of diabetes in aged 25–34 (1981 data standardized against the 1998 Australian population) was 1.3 % [35] and in the AusDiab (1998 data) for the same age group was 0.3 %, with an abnormal glucose tolerance prevalence of 5.7 % [35, 36]. The latter finding is unusual, as for every other age group the diabetes prevalence recorded in AusDiab was higher than for Busselton, and may be due to the under-representation of this age group for the biomedical examination in AusDiab, or the higher socioeconomic status of responders [36].

The attrition rates in the MUSP cohort were considerable though comparable to similar long follow-up studies such as the British Birth Cohort study. Interestingly, the MUSP has 99 % baseline data for all participants, enabling us to compare the characteristics of respondents who remain with those lost to follow-up. Overall, we found the sub-sample who remained in the study compared to those lost to follow-up were more likely to be socio-demographically advantaged. Our results would be biased if the associations between exposure and outcome were s non-existent or in the opposite direction with those lost to follow-up, which is unlikely. We used a variety of methods including multiple imputation and inverse-probability weighting to examine potential attrition bias [37, 38]. Overall, we have found no substantial impact of attrition on effect estimates.

The MUSP sample comprises effectively all consecutive births of public patients in the Mater Hospital over 3 years during 1981–1983, ensuring a broad cross-section of mid to lower socio-economic status women and births. Women were not selected on the basis of any physical or social characteristic. The MUSP is not representative of all Australian families. However, there is no biological reason to suspect that the associations we found in this study should differ substantially from other populations. Many of the epidemiological associations that inform public health practice are based upon cohort studies that are similarly unrepresentative of the overall population. Examples include the effect of smoking on lung cancer and cardiovascular disease (a cohort of British male doctors [39]) and the effect of cholesterol on cardiovascular disease (the Framingham study of middle-aged US adults [40]).

In conclusion, this study suggests that while any breastfeeding has some protective effect against the development of young adult diabetes, a longer duration (in our data characterized as at least 4 months) may provide stronger long-term protection against the development of young adult offspring diabetes. Ideally, future studies should use the WHO/UNICEF recommendation of 6 months exclusive breastfeeding [33] and examine whether this recommend duration of breastfeeding has most protective effect against development of diabetes. Importantly, this protective effect was not explained by the development of obesity. The sharply increasing prevalence of diabetes around the world including Australia represents a serious public health crisis in the twenty-first century. Although there is no unique solution to this problem, promoting breastfeeding for a minimum of 4 months may be a useful strategy for the prevention of diabetes among young adults.

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Conflict of interest Abdullah Al Mamun, Michael J. O'Callaghan, Gail M. Williams, Jake M. Najman, Leonie Callaway and Harold D. McIntyre declare that they have no conflict of interest.

Ethical standard Ethical clearances were obtained from the University of Queensland Human Ethics committee.

Human and animal rights disclosure All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2008 (5).

Informed consent disclosure Informed consent was obtained from all participants for being included in the study.

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