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Familial intergenerational and maternal aggregation patterns in nutrient intakes in the Lifeways Cross-Generation Cohort Study

Aakash Shrivastava[1], Celine Murrin[1], Mary Rose Sweeney[2], Patricia Heavey[1] and Cecily C Kelleher[1,*] for the Lifeways Cohort Study Steering Group

[1]National Nutrition Surveillance Centre and Health Research Board Centre for Diet and Health Research, UCD School of Public Health, Physiotherapy and Population Science, Woodview House, University College Dublin, Belfield, Dublin 4, Ireland: [2]School of Nursing, Dublin City University, Dublin, Ireland

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Abstract

Objective: The current study prospectively examines the intra-uterine hypothesis by comparing maternal, paternal and grandparental lineage influences on children's diet and also maternal–child aggregation patterns during pregnancy and early childhood.
Design: Prenatal dietary information was available for expectant mothers, fathers and up to four grandparents through a detailed validated semi-quantitative FFQ. At 6-year follow-up, when children averaged 5 years of age, dietary information was re-collected for mothers and a subset of maternal grandmothers using the same FFQ. Child's FFQ version was used for children. Anthropometric and socio-demographic variables were also collected.
Settings: Three-generation familial cohort representative of the contemporary Irish national population.
Subjects: Children aged 5 years (n 567) and their parents and grandparents.
Results: Associations for energy, macronutrient and fibre intakes were compared using Pearson's correlations, intra-class correlations (ICC) and linear regression models, adjusted for energy and potential confounders. Significant, moderate-strength positive correlations were observed for nutrient intakes in children's nuclear families (ICC (range) = 0·22–0·28). The father–child associations (r (range) = 0·13–0·20) were weaker than the mother–child associations (r (range) = 0·14–0·33). In general, associations were stronger for maternal postnatal intake–child intake than for maternal prenatal intake–child intake, except for percentage of energy from fat (adjusted $\beta = 0{\cdot}16$, 95% CI 0·05, 0·26; $P = 0{\cdot}004$), which was stronger for maternal prenatal intake, specifically in non-breast-fed children (adjusted $\beta = 0{\cdot}28$, 95% CI 0·12, 0·44; $P = 0{\cdot}001$). Among all grandparents, correlations were significant only for maternal grandmother–mother pairs (r (range) = 0·10–0·36). Significant positive ICC were observed for nutrient intakes of maternal grandmother–mother–child triads (ICC (range) = 0·12–0·27), not found in paternal lines.
Conclusions: These findings suggest that maternal-environment programming influences dietary intake.

Keywords
Prenatal–postnatal
Diet
Intergenerational
Familial aggregation

The extent to which food and nutrient patterns cluster or aggregate within families has considerable public health importance. This is particularly relevant given the rising trends in early childhood obesity and subsequent related chronic disease patterns, now seen in most countries worldwide. Yet there is a relative paucity of research on familial aggregation of dietary intake. Wang *et al.*[1] recently performed a systematic review on parent–child resemblances for food and nutrient intakes. Their search resulted in only twenty-four studies of satisfactory methodological quality suitable for review. Of these, very few were from large national samples and most were from the American continent.

Nuclear families may share common genes and home environment. Twin studies on dietary preferences and intake have shown that genetic predisposition partially explains this familial resemblance[2–5]. Parents may also influence children's food preferences and behaviour through food provision, role modelling and parenting patterns[6,7]. In their review, Wang *et al.*[1] concluded that there was a definite parent–child intake resemblance, although it was weak to moderate in strength. The same was true for studies on pre-school children[8–10].

Not many studies have paternal dietary information and only a few with such data have analysed paternal–child intakes separately from maternal–child intakes, to

*Corresponding author: Email Cecily.kelleher@ucd.ie

allow parent-of-origin comparisons. Also, to our knowledge there is only one three-generation study on familial intake which again was limited to examining the female line (maternal grandmothers, mothers and daughters) as adults[11]. Although these studies discussed the possibility of the genetic and home-environmental influence as explanations for their observed familial dietary aggregation patterns, they did not sufficiently explain why the maternal-line associations in their findings were stronger.

Studies comparing the influence of prenatal and postnatal maternal dietary intake on offspring's diet are also rare. Brion *et al.*[12], the first to publish such a comparison, showed that maternal macronutrient intake at 32 weeks of pregnancy and 47 months postnatally were each positively associated with their children's intake, and further, that these maternal associations were stronger than those for paternal intake. They also demonstrated that associations of maternal prenatal–child intake patterns were greater than maternal postnatal–child intake, specifically for protein and fat. This Avon Longitudinal Study of Parents and Children (ALSPAC) concluded that these findings demonstrated evidence of *in utero* programming of children's dietary behaviours.

The aim of the present analysis was to examine patterns of familial aggregation of dietary intake in the Lifeways Cross-Generation Cohort Study. We sought to examine the existence, direction and magnitude of parental associations for children's dietary intake when they averaged 5 years of age. To assess intra-uterine influences on offspring's diet, we first contrasted the parent-of-origin associations, the maternal–child associations with paternal–child associations for dietary intake. These comparisons were made for maternal and paternal intake measured during pregnancy and additionally for maternal intake 5 years postnatally. Second, we compared the associations of child's dietary intake with maternal intake during pregnancy and maternal intake 5 years postnatally. Finally we tested our hypothesis on intra-uterine influences by comparing aggregation in maternal and paternal lines of three generations.

Methods

Lifeways is a three-generation Irish familial cohort that was established in 2001–2003; the recruitment procedure has been described previously[13]. The *a priori* purpose of establishing the cohort was to examine familial and cross-generation influences on early childhood development over the first 5 years of children's lives, studying development and health in a life course perspective.

In brief, mothers were recruited at first booking visit during pregnancy in two regional maternity hospitals in Galway (west) and Dublin (east) at which point (time 1, T1) they completed a health status questionnaire, including a semi-quantitative food frequency (SQFFQ) instrument containing 149 food and drink items developed for surveillance purposes in the Republic of Ireland from the European Prospective Investigation into Cancer and Nutrition (EPIC)[14]. This instrument was validated for an Irish adult population using food diary and protein biomarker studies[15]. Each expectant mother provided information on her habitual diet since conception. In the health status questionnaire each mother also provided information on her pre-pregnancy anthropometric measures, including height and weight, and other sociodemographic variables. It was aimed, if possible, to include the mother's male partner and at least one grandparent. These adults also completed the same questionnaire as the mothers at baseline and, along with anthropometric measures and other sociodemographic variables, provided information on their habitual diet for the last year. Live infants were subsequently added to the cohort with hospital maternity linkage information.

In 2007–2008, when these children averaged 5 years of age (time 2, T2), mothers were asked to repeat the health assessment questionnaire, including reported dietary intake, and to provide information also on their child's health status, including dietary intake. Mothers responded for themselves on the same SQFFQ as earlier and again reported habitual diet for the previous year. The SQFFQ containing fifty-two food and drink items used for assessment of children's diet was comparatively different and adapted from the UK National Diet and Nutrition Survey of 4·5-year-old children[16]. The mothers' and children's SQFFQ were also validated in the Lifeways study using a 7 d weighed food diary in a sub-sample prior to follow-up stage of the study[17]. Mothers also provided information on children's breast-feeding and child-care attendance (both institutional and non-institutional). Mothers and children were offered an examination for height, weight and waist circumference as well, to a standardised protocol[17]. At the same time in 2007–2008, a repeat dietary assessment with the same adult SQFFQ instrument was undertaken for a sub-set of these grandparents residing in the greater Dublin area only. Figure 1 provides a schematic representation of data collection relevant to this analysis.

Nutrient conversion was undertaken using McCance and Widdowson's food composition tables[18] with the program FFQ_Software© version 1·0 developed specially by the National Nutrition Surveillance Centre, School of Public Health and Population Science, University College Dublin. Dietary data were logarithm-transformed to improve normality. Pearson's correlations were undertaken for energy, macronutrient and fibre intakes in cohort member pairs (dyads), in unadjusted, adjusted for energy and fully adjusted models. Family correlation is a well-accepted behavioural genetic strategy design to analyse if behavioural traits run in families[19]. Energy adjustments were made by the multivariate nutrient density model method[20,21]. Nutrients were converted to nutrient densities by computing percentage of energy for

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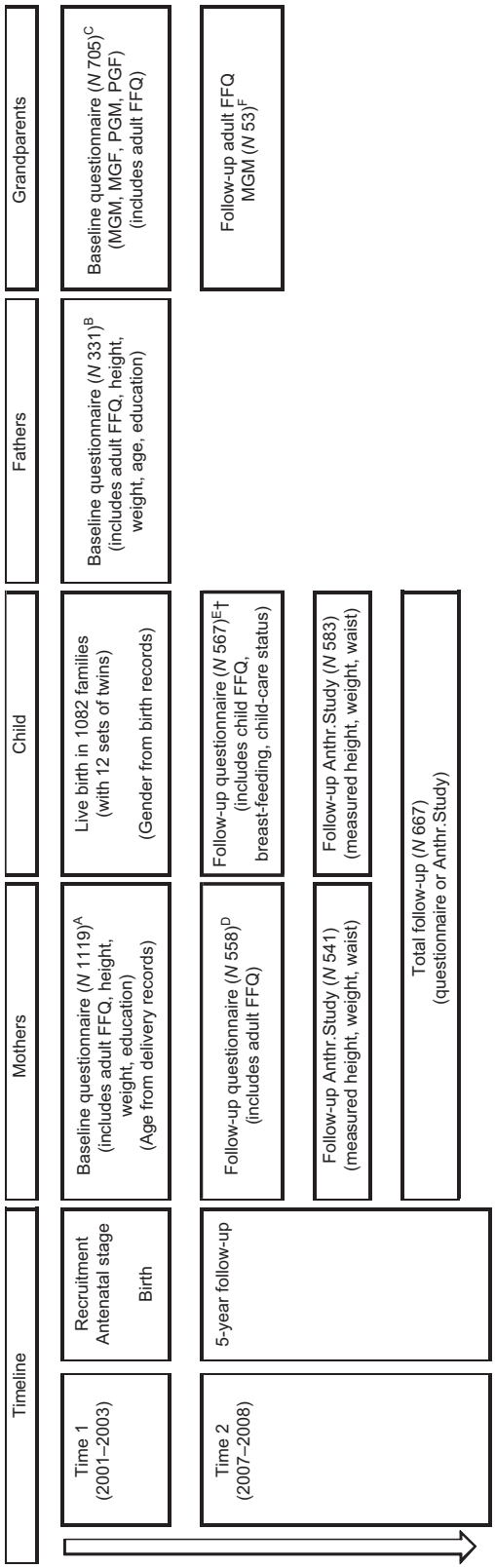

Fig. 1 Schematic representation of time points and data available for analysis ([A–F]corresponding figures in Table 1; †core analysis group; MGM, maternal grandmothers; MGF, maternal grandfathers; PGM, paternal grandmothers; PGF, paternal grandfathers; Anthr.Study, anthropometric measurement study)

protein, fat and carbohydrate and dividing fibre intake by 4184 kJ (1000 kcal). Statistical tests were computed on nutrient densities with additional adjustment for energy variables as covariates. The fully adjusted model additionally adjusted for children's gender, height, BMI and for parents' age, height, BMI and education level.

Intra-class correlations (ICC) were also undertaken, first for three nuclear family triads (that of child and parents, mother and her parents and father and his parents) and second for extended families, in maternal and paternal lines (child, parent, grandparents). ICC implied that intake among individuals of the same family (within-family) was more alike than among individuals from different families (between-family)[22,23].

Finally, maternal prenatal–postnatal comparisons were made using independent linear regression models for children's energy intake and each of the nutrients. The regression of children's intake for the nutrient of interest (dependent variable) *v.* mothers' prenatal (T1) and postnatal (T2) intake for the same nutrient (independent variables) was performed. Similar to correlations analyses, linear regressions were computed in unadjusted, energy-adjusted and fully adjusted models. These fully adjusted models additionally adjusted for the use of child-care services and were also stratified for breast-fed and non-breast-fed children. All analyses were made using the SPPS statistical software package version 15·0.

Ethical approval for the Lifeways study was obtained from ethical committees of Coombe University Hospital, Dublin, University College Dublin, the Irish College of General Practitioners and University College Hospital Galway, Ireland.

Results

Of 1119 expectant mothers with FFQ data, 1082 gave birth to a live infant; 331 fathers, 283 maternal grandmothers, 163 maternal grandfathers, 163 paternal grandmothers and ninety-six paternal grandfathers also participated in the dietary study at the antenatal stage (T1). In the 2007–2008 follow-up (T2), 558 mothers, 567 children and fifty-three maternal grandmothers provided dietary information. As the participation of grandparents at follow-up (T2) was small, for the purposes of the present analysis only maternal grandmothers were considered. Exact numbers of available FFQ for participating family member pairs (dyads) from singleton baby (non-twin) families are given in Table 1.

Table 2 gives the unadjusted correlations for energy (kJ), protein, fat, carbohydrate and fibre intakes (all in grams) between all family member dyads of this cohort. It shows clear relationships in intakes for the child's nuclear family, i.e. mother–child–father. Only the father's fat intake was not correlated with that of his child. The positive correlations in the child's nuclear family were of

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Table 1 Number of FFQ data (*N*) and FFQ pairs (*n*) available at antenatal (T1) and follow-up (T2) stages: Lifeways Cross-Generation Cohort Study, Republic of Ireland

	Mothers (T1; *N* 1119)[A]	Mothers (T2; *N* 558)[D]	Fathers (T1; *N* 331)[B]	MGM (T1; *N* 283)[C]	MGM (T2; *N* 53)[F]	MGF (T1; *N* 163)[C]	PGM (T1; *N* 163)[C]	PGF (T1; *N* 96)[C]
Children (T2; *N* 567)[E]/(*N* 552 singleton children)†	*n* 545	*n* 551	*n* 229					
Mothers (T1)			*n* 329	*n* 282	*n* 53	*n* 161		
Mothers (T2)			*n* 234	*n* 181	*n* 37			
Fathers (T1)							*n* 86	*n* 51

T1, prenatal stage time point; T2, follow-up stage time point (postnatal); MGM, maternal grandmothers; MGF, maternal grandfathers; PGM, paternal grandmothers; PGF, paternal grandfathers; *N*, total FFQ in sample; *n*, FFQ in pairs (dyads) for singleton baby families.
[A–F]Corresponding figures in Fig. 1.
†Core analysis group.

Table 2 Pearson's correlations for energy, macronutrient and fibre intakes in family member dyads (unadjusted model): Lifeways Cross-Generation Cohort Study, Republic of Ireland

	Mother (T1)–child (T2)		Mother (T2)–child (T2)		Father (T1)–child (T2)		Mother (T1)–father (T1)	Mother (T2)–father (T1)
	r	95 % CI	*r*	95 % CI	*r*	95 % CI	*r*	*r*
Energy (kJ)	0·19**	0·11, 0·27	0·31**	0·24, 0·39	0·13*	0·00, 0·26	0·19**	0·24**
Protein (g)	0·18**	0·10, 0·26	0·27**	0·19, 0·35	0·13*	0·00, 0·26	0·12*	0·13*
Fat (g)	0·15**	0·07, 0·23	0·25**	0·17, 0·33	0·05	−0·08, 0·18	0·17**	0·22**
Carbohydrate (g)	0·14**	0·06, 0·23	0·27**	0·19, 0·35	0·15*	0·02, 0·27	0·19**	0·22**
Fibre (g)	0·15**	0·07, 0·23	0·33**	0·25, 0·40	0·20**	0·07, 0·32	0·21**	0·18**

	MGM (T1)–mother (T1)	MGM (T1)–mother (T2)	MGM (T2)–mother (T1)	MGM (T2)–mother (T2)	MGF (T1)–mother (T1)	PGM (T1)–father (T1)	PGF (T1)–father (T1)
	r	*r*	*r*	*r*	*r*	*r*	*r*
Energy (kJ)	0·06	0·08	0·25(*)	0·29(*)	−0·04	−0·24	−0·09
Protein (g)	0·10*	0·11	0·30*	0·34*	−0·06	−0·18	−0·05
Fat (g)	0·07	0·06	0·27*	0·36*	0·05	−0·16	−0·18
Carbohydrate (g)	0·06	0·08	0·12	0·26	−0·08	−0·25	0·08
Fibre (g)	0·15*	0·12	−0·05	0·18	−0·04	−0·08	0·17

T1, prenatal stage time point; T2, follow-up stage time point (postnatal); MGM, maternal grandmothers; MGF, maternal grandfathers; PGM, paternal grandmothers; PGF, paternal grandfathers.
(*)$P < 0·1$, *$P < 0·05$, **$P < 0·01$ (all two-tailed).

modest strength, but statistically significant, with the mother–child correlations ($r \leq 0{\cdot}33$) being stronger than the father–child ones ($r \leq 0{\cdot}20$). The positive correlations observed for mother–child dietary intake were generally stronger for the maternal postnatal (T2) diet (r (range) = 0·25–0·33) than for the maternal prenatal (T1) diet (r (range) = 0·14–0·19). There was also a direct pattern of association between the maternal grandmother's dietary intake and that of her daughter at both time points ($r \leq 0{\cdot}36$). Other than the maternal grandmothers, none of the other grandparents showed any significant dietary associations.

Table 3 shows the correlations for energy-adjusted nutrient intakes. The correlations were largely reduced or weakened in this model. However, the mother–child correlations ($r \leq 0{\cdot}23$) were still stronger than those for the father ($r \leq 0{\cdot}14$) and again generally stronger associations were observed with maternal postnatal (T2) intake ($r \leq 0{\cdot}23$). The exception was fat intake, which appeared to be borderline significant only for the maternal prenatal (T1) intake ($r = 0{\cdot}08$, $P = 0{\cdot}08$). Maternal grandmother's fat ($r = 0{\cdot}15$) and fibre ($r = 0{\cdot}25$) intakes continued to correlate positively and significantly with those of her daughter.

Table 4 shows the correlations in the fully adjusted models for the child's nuclear family. Mother–child positive and statistically significant correlations (r (range) = 0·11–0·29) were still seen at both time points, but the father–child correlations attenuated and were no longer statistically significant. The maternal postnatal intake–child intake positive correlations were again stronger than for maternal prenatal intake–child intake as seen with energy ($r = 0{\cdot}29$), protein ($r = 0{\cdot}11$) and fibre ($r = 0{\cdot}21$). However, the maternal prenatal intake–child intake was now significantly stronger for fat ($r = 0{\cdot}17$, $P = 0{\cdot}001$).

Table 5 shows the ICC results in family triads and extended three-generation family lines. A statistically significant homogeneity was found in energy and nutrient intakes of the child's nuclear family (ICC (range) = 0·22–0·28) and also in the mother's nuclear family (ICC (range) = 0·08–0·19) although they were comparatively weaker in strength. However, there were no significant correlations for the father's family. When comparisons were made in extended family lines involving only parents and grandparents of concerned lineage, the ICC were again significant in the maternal family line (ICC (range) = 0·09–0·19) and not in the paternal family line (ICC (range) = −0·04 to 0·09). Finally, removal of the maternal grandfather from the maternal family line further improved the correlation strengths for maternal grandmother–mother–child triads, suggesting a stronger homogeneity in their dietary intake (ICC (range) = 0·12–0·27). These findings confirm familial resemblances in dietary intake patterns for the children's nuclear family and also in the maternal line, but not the paternal line.

Table 6 shows the unadjusted, energy-adjusted and fully adjusted linear regression models. The models show

Table 3 Partial correlations for energy-adjusted macronutrient and fibre intakes in children's nuclear family member dyads and maternal grandmother–mother dyads (energy-adjusted model): Lifeways Cross-Generation Cohort Study, Republic of Ireland

	Mother (T1)–child (T2)	Mother (T2)–child (T2)	Father (T1)–child (T2)	Mother (T1)–father (T1)	Mother (T2)–father (T1)
	Partial *r*	Partial *r*	Partial *r*	Partial *r*	Partial *r*
% Energy from protein	0·12**	0·15**	0·02	0·07	0·07
% Energy from fat	0·08(*)	0·03	0·02	0·14*	0·18*
% Energy from carbohydrate	0·05	0·06	0·14	0·06	0·12(*)
Fibre (g/4184 kJ†)	0·10*	0·23**	0·14*	0·25**	0·15*
	MGM (T1)–mother (T1)	MGM (T1)–mother (T2)	MGM (T2)–mother (T1)	MGM (T2)–mother (T2)	
	Partial *r*	Partial *r*	Partial *r*	Partial *r*	
% Energy from protein	0·01	0·00	0·08	0·17	
% Energy from fat	0·15*	0·12(*)	0·09	0·29(*)	
% Energy from carbohydrate	0·08	0·06	0·00	0·05	
Fibre (g/4184 kJ†)	0·25**	0·14(*)	−0·09	−0·18	

T1, prenatal stage time point; T2, follow-up stage time point (postnatal); MGM, maternal grandmothers.
(*) $P < 0{\cdot}1$, * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$ (all two-tailed).
†4184 kJ = 1000 kcal.

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Table 4 Partial correlations for fully adjusted† energy, macronutrient and fibre intakes in children's nuclear family member dyads (full adjusted model): Lifeways Cross-Generation Cohort Study, Republic of Ireland

	Mother (T1)–child (T2)	Mother (T2)–child (T2)	Father (T1)–child (T2)
	Partial *r*	Partial *r*	Partial *r*
Energy (kJ)	0·15**	0·29**	0·11
% Energy from protein	0·06	0·11*	−0·01
% Energy from fat	0·17**	0·06	−0·02
% Energy from carbohydrate	0·10(*)	0·07	0·03
Fibre (g/4184 kJ‡)	0·08	0·21**	0·14(*)

(*)$P < 0·1$, *$P < 0·05$, **$P < 0·01$ (all two-tailed).
†Adjusted for children's characteristics: gender, height (cm), BMI (kg/m^2); parents' characteristics: age (years), height (cm), BMI (kg/m^2), education status; and additional adjustment of energy (kJ) of children and parents.
‡4184 kJ = 1000 kcal.

the same pattern as seen in correlation models for the maternal–child dyads. The maternal–child intake associations were generally stronger for maternal postnatal (T2) diet (energy, percentage of energy from protein, energy-adjusted fibre), with exception of the percentage of energy from fat which, although modest in magnitude, was statistically highly significant for maternal prenatal (T1) intake (adjusted $\beta = 0·16$, 95% CI 0·05, 0·26; $P = 0·004$). In the fully adjusted models the child gender variable and the variable on history of child-care use were neither statistically significant nor made appreciable change to observed associations.

The fully adjusted models stratified by history of breast-feeding showed that maternal–child intake associations for percentage of energy from protein were observed only for breast-fed children and the associations for energy and energy-adjusted fibre were also relatively stronger for breast-fed children. These associations were again stronger with maternal postnatal intake. However, the earlier observed association between maternal prenatal–child intake for percentage of energy from fat was seen only in non-breast-fed children (adjusted $\beta = 0·28$, 95% CI 0·12, 0·44; $P = 0·001$).

Discussion

We have shown statistically significant patterns of dietary intake associations of moderate strength and positive direction in the children's nuclear family in this contemporary birth cohort study. The findings are consistent with the direction and magnitude of association reported by Wang *et al.*[1] in their recently published meta-analysis on parent–child resemblance studies for dietary intake, suggesting a coherent familial association, not just by chance. The magnitude of the association is also consistent with a meta-analysis on parent–child resemblance studies for food preferences, instead of intakes[24].

We have also demonstrated that maternal–child dietary resemblances are stronger than paternal–child dietary resemblances at both prenatal and postnatal time points. The fact that the mother provided responses for both herself and her child could be a possible reason for stronger maternal associations. However, a number of methodological steps, as detailed in the 'Methods' section, were taken to minimise the possibility of such a bias. First, the study employed FFQ of different design for mothers and children, so a mother could not easily replicate in one section what was recorded in another; the adult and child FFQ versions were adopted from different international studies. Second, at the follow-up stage, a subset of mothers' and children's FFQ was validated using a 7 d weighed food diary under the supervision of researchers making home visits. The maternal–paternal differences were also observed at the prenatal time point, recorded by both parents 6 years before the mothers recorded the child's intake; thus it is unlikely that the stronger maternal–child associations are solely due to bias.

The finding of stronger maternal–child dietary resemblances is again consistent with that of previous comparable studies[8,23,25,26]. Demonstrating a stronger maternal association compared with paternal association in itself suggests a unique relationship between mother and child, potentially an intra-uterine mechanism of influence on offspring's outcome.

It might be argued that mothers' influence on children in their shared home environment would be stronger than that of fathers. However, we were able to further validate our finding by contrasting associations for parental exposure at the time of pregnancy itself. Davey-Smith *et al.* contend that demonstrating that maternal exposure during pregnancy has a stronger influence than paternal exposure is a robust method of showing that child outcome is due to intra-uterine exposure[27,28]. For the first time, we have shown this effect using maternal and paternal dietary intake both measured at 14–20 weeks of pregnancy and the resulting parent–child intake association patterns were consistent with those that Brion *et al.*[12] demonstrated with ALSPAC cohort data, contrasting maternal intake at 32 weeks' gestation with paternal intake 47 months postnatally.

We also found that maternal prenatal–offspring fat intake associations were stronger than maternal postnatal–offspring fat intake associations, a finding in respect to fat

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Table 5 Intra-class correlations (ICC) between nuclear family triads and extended maternal *v.* paternal family lines: Lifeways Cross-Generation Cohort Study, Republic of Ireland

	Child's family		Mother's (T1) family		Mother's (T2) family		Father's family	
	Mother (T2)–father (T1)–child (T2) (*n* 229)		MGM (T1)–MGF (T1)–mother (T1) (*n* 122)		MGM (T1)–MGF (T1)–mother (T2) (*n* 85)		PGM (T1)–PGF (T1)–father (T1) (*n* 37)	
	ICC	95 % CI	ICC	95 % CI	ICC	95 % CI	ICC	95 % CI
Energy (kJ)	0·28*	0·19, 0·36	0·08(*)	−0·02, 0·20	0·08(*)	−0·04, 0·23	−0·01	−0·17, 0·20
Protein (g)	0·23*	0·15, 0·32	0·11*	0·00, 0·22	0·14*	0·01, 0·28	0·09	−0·09, 0·31
Fat (g)	0·22*	0·14, 0·31	0·09*	0·00, 0·21	0·08(*)	−0·04, 0·22	−0·04	−0·20, 0·17
Carbohydrate (g)	0·25*	0·17, 0·33	0·08(*)	−0·02, 0·20	0·10(*)	−0·03, 0·24	−0·01	−0·17, 0·21
Fibre (g)	0·26*	0·17, 0·34	0·19*	0·08, 0·31	0·15*	0·02, 0·29	0·13	−0·05, 0·36
	Four members maternal line		Four members paternal line		Three members maternal line			
	MGM (T1)–MGF (T1)–mother (T2)–child (T2) (*n* 45)		PGM (T1)–PGF (T1)–father (T1)–child (T2) (*n* 26)		MGM (T1)–mother (T2)–child (T2) (*n* 45)			
	ICC	95 % CI	ICC	95 % CI	ICC	95 % CI		
Energy (kJ)	0·12*	0·00, 0·29	−0·05	−0·16, 0·14	0·24*	0·06, 0·44		
Protein (g)	0·09(*)	−0·03, 0·25	−0·05	−0·16, 0·14	0·12(*)	−0·05, 0·32		
Fat (g)	0·09(*)	−0·03, 0·25	−0·04	−0·15, 0·15	0·22*	0·04, 0·42		
Carbohydrate (g)	0·12*	0·00, 0·29	−0·04	−0·16, 0·15	0·18*	0·00, 0·38		
Fibre (g)	0·19*	0·05, 0·36	0·09	−0·06, 0·31	0·27*	0·08, 0·46		

T1, prenatal; T2, postnatal; MGM, maternal grandmothers; MGF, maternal grandfathers; PGM, paternal grandmothers; PGF, paternal grandfathers.
(*)$P < 0{\cdot}1$, *$P < 0{\cdot}05$ (all two-tailed).

Table 6 Results from regression of children's energy, macronutrient and fibre intakes *v.* mothers' prenatal and mothers' postnatal energy, macronutrient and fibre intakes (*n* 544): Lifeways Cross-Generation Cohort Study, Republic of Ireland

Unadjusted model (*n* 544)			Energy-adjusted model (*n* 544)			Fully adjusted model (*n* 419)†			Breast-fed fully adjusted (*n* 217)†,‡			Not breast-fed fully adjusted (*n* 190)†,‡		
	Std β	*P*		Std β	*P*	Std β	95 % CI	*P*	Std β	95 % CI	*P*	Std β	95 % CI	*P*
Child's energy (kJ)														
Mother's T1 energy (kJ)	0·115	0·007				0·096	−0·00, 0·19	0·055	0·078	−0·06, 0·22	0·27	0·131	−0·01, 0·28	0·07
Mother's T2 energy (kJ)	0·282	0·000				0·259	0·16, 0·35	0·000	0·275	0·14, 0·42	0·000	0·221	0·08, 0·36	0·003
Child's protein (g)			Child's % energy from protein											
Mother's T1 protein (g)	0·116	0·007	Mother's T1 % energy from protein	0·087	0·05	0·063	−0·04, 0·16	0·21	0·017	−0·12, 0·15	0·81	0·076	−0·08, 0·23	0·34
Mother's T2 protein (g)	0·236	0·000	Mother's T2 % energy from protein	0·120	0·008	0·087	−0·02, 0·19	0·09	0·161	0·02, 0·30	0·02	0·052	−0·11, 0·22	0·54
Child's fat (g)			Child's % energy from fat											
Mother's T1 fat (g)	0·085	0·05	Mother's T1 % energy from fat	0·077	0·10	0·158	0·05, 0·26	0·004	0·051	−0·10, 0·20	0·51	0·278	0·12, 0·44	0·001
Mother's T2 fat (g)	0·225	0·000	Mother's T2 % energy from fat	−0·003	0·96	0·011	−0·10, 0·12	0·84	0·044	−0·12, 0·21	0·60	0·001	−0·15, 0·15	0·99
Child's carbohydrate (g)			Child's % energy from carbohydrate											
Mother's T1 carbohydrate (g)	0·091	0·03	Mother's T1 % energy from carbohydrate	0·041	0·34	0·072	−0·03, 0·17	0·15	0·068	−0·07, 0·21	0·29	0·038	−0·11, 0·19	0·61
Mother's T2 carbohydrate (g)	0·250	0·000	Mother's T2 % energy from carbohydrate	0·047	0·27	0·060	−0·04, 0·16	0·22	0·073	−0·06, 0·21	0·34	0·078	−0·07, 0·23	0·30
Child's fibre (g)			Child's fibre (g/4184 kJ§)											
Mother's T1 fibre (g)	0·056	0·19	Mother's T1 fibre (g/4184 kJ§)	0·045	0·38	0·037	−0·09, 0·16	0·56	0·089	−0·09, 0·27	0·33	−0·03	−0·23, 0·17	0·77
Mother's T2 fibre (g)	0·312	0·000	Mother's T2 fibre (g/4184 kJ§)	0·286	0·000	0·265	0·14, 0·39	0·000	0·247	0·04, 0·46	0·02	0·227	0·05, 0·41	0·01

Std β, standardized β coefficient (standard deviation change in child's intake for 1-unit standard deviation change in mother's intake); T1, prenatal time point; T2, postnatal (follow-up stage) time point.
P values are two-tailed.
†Adjusted for children's characteristics: gender, height (cm), BMI (kg/m^2), attended child care; mothers' characteristics: age (years), height (cm), BMI (kg/m^2), education status; and additional adjustment of energy (kJ) of children and mothers.
‡Stratified by breast-fed status.
§4184 kJ = 1000 kcal.

intake also demonstrated by the ALSPAC study[12]. Thus the present study is the second one to compare maternal prenatal–postnatal dietary intake influence on her young child's dietary intake. Our study has an added advantage of investigating this effect in children of pre-school age compared with ALSPAC study data on children of primary school age.

Our analysis provided a further addition to the previous literature by stratifying these prenatal–postnatal associations by children's breast-feeding status. The stratified analysis revealed that dietary intake associations for mother–child resemblances were generally stronger for breast-fed children. The exceptional association noted for maternal prenatal fat intake was observed only in non-breast-fed children. Not breast-feeding and consuming fatty foods are both unhealthy behaviours and a possible explanation for the association may be that mothers practising unhealthy behaviours themselves go on to feed fatty foods to their children.

However, the ALSPAC investigators[12] contended that the stronger maternal prenatal associations suggested maternal *in utero* programming of children's preferences. The finding that maternal prenatal fat-rich diet may program the offspring's preferences for fat intake has also been demonstrated in a number of animal studies[29–33], which suggest that maternal prenatal intake of fatty palatable foods permanently alters offspring's *in utero* development and expression of central neural (reward related) and endogenous systems involved in regulation of preferences and intake of palatable (junk) foods[29–32], even at the expense of protein- and fibre-rich foods[32]. The relevance of fat is attributed to a possible mechanistic explanation that its palatability is an important driving stimulus in dietary intake regulation[32]. Evidence suggests that infants' taste and preferences are formed with the earliest sensory perceptions from amniotic fluid (prenatal) or breast milk (postnatal) of the mother[34–36]. On the contrary, there is also evidence that formula feed flavours[37,38] and post-weaning feeding practices influence children's conditioning of tastes[36,39]. Thus it may be difficult to disentangle these early-life effects from one another.

However, our analysis showed predominantly maternal postnatal associations for energy, energy-adjusted protein and fibre suggesting that mothers' influence through the shared food environment is still substantial.

The current study is the first three-generation family cohort analysis to demonstrate clear patterns of aggregation of dietary intake distinguishable along maternal and paternal lines. A key strength of the study was the inclusion of grandparents from both lineages, with maternal grandmothers' dietary information available at two time points. While numbers were modest, none the less the effects were consistently suggestive of a positive association in maternal grandmother–daughter dietary intake. Stafleu *et al.*[11] had similarly shown a maternal-line association for maternal grandmothers' food, energy, fat and fatty acid intakes, although they did not have other grandparents' dietary data for comparisons. The observed association could be explained by a shared childhood environment where grandparents shape their children's (parents') dietary preferences, specifically with grandmothers imparting cookery skills to their daughters (mothers). However, this was unlikely to provide the full explanation considering that these grandparents and parents would usually live apart as adults. Moreover, our analyses did not demonstrate any association with paternal grandparents and also the homogeneity in intake among maternal grandmother–mother–child triads only improved with removal of maternal grandfathers from the maternal family line, suggesting again the maternal programming hypothesis of transmission. The maternal grandmother–mother–child relationship is unique in sharing an additional pathway of the womb in addition to other possible genetic and environmental pathways. Barker, the seminal author in this field, postulates that a 100-year period of nutritional flow from maternal grandmother and mother through the intra-uterine route may influence the health for life of an individual[40]. An explanation for the fetal origins hypothesis is that maternal provisioning is a conduit to transfer nutritional history of matrilineal ancestry to the fetus in order to align offspring to characteristics of the matrilineal line[41,42].

Wang *et al.*[1] in their systematic review devised a seven-component score for judging methodological quality of the family resemblance studies (sample size of dyads, age range of children, method of dietary assessment, foods and nutrients analysed, types of parent–child pairs analysed, adjustment for confounders, and representativeness of sample). Based on these criteria, our study has a number of strengths.

It is a nationally representative cohort, whose socio-demographic profile has shown a match to Irish national survey data[43]. However, interpretation should be circumspect in that, as in most cohorts, there was a loss to follow-up over the period of 6 years, which may have introduced a degree of self-selection bias.

To our knowledge, our study is unique in having collected independent data in the same families for both prenatal and postnatal maternal dietary intake, and also data from fathers and all grandparent lines, probably unprecedented for paternal grandparents. It is also unique in collecting prenatal data for both parents at the same time point in early pregnancy.

Postnatal data were collected when the child was at a critical age, at school entry point. This allowed for a proper estimation of familial influence, before the children's diet would become influenced by the environment beyond the immediate family sphere. Although children may utilise institutional or non-institutional child-care services before regular school starts, adjustment for this variable in our study did not attenuate the observed pattern in mother–child associations.

We employed a validated detailed FFQ which has been widely used internationally and which was specifically validated against a 7 d food diary in this cohort. Although maternal postnatal and other cohort members' dietary data were collected for their habitual diet for the preceding year, the maternal prenatal dietary data were collected for a relatively shorter duration of 3–4 months because it was intended to estimate her diet during pregnancy. It may be argued that this difference in reference period might have induced a bias, with maternal recall for her prenatal dietary data being relatively better. However, since maternal–paternal differences were not only seen with maternal prenatal but also with maternal postnatal and grandparental data, it is unlikely that this difference in recall period had any noteworthy impact. Furthermore, the maternal postnatal diet with a longer recall period actually showed stronger associations than the maternal prenatal diet.

Finally, our analyses were adjusted for a variety of potential confounders, including BMI and measures of social position.

The current study does have some acknowledged limitations. It is a relatively small cohort which may have implications for power considerations, but judging by sample sizes of most parent–child resemblance studies, our sample size was satisfactory. In fact if we applied the chosen criteria of Wang *et al.*[1] for rating studies by sample size in their systematic review, our study qualifies for the highest score by this criterion.

Clearly only a third of fathers provided dietary data and not all grandparents participated. A comparison of non-responders *v.* responders on sociodemographic profile showed that, as expected, responders had a relatively better social status; but all the same the anthropometric parameters of nutritional status were not different in both groups[17]. Arguably this difference might have influenced the findings in that the most closely knit families participated. It is possible therefore that there is some systematic bias at play which could influence our outcome in producing stronger correlations for participating families, but this is unlikely to influence the differential patterns of associations observed in the matrilineal and patrilineal lines. Also, our findings with this prospective contemporary familial cohort are very consistent with findings from other published studies.

This cohort in a former analysis demonstrated a familial aggregation in BMI, with stronger effects of maternal-line influence[44] and in the present analysis, even after controlling for familial BMI, we show a familial aggregation in dietary intake with similar patterns of stronger maternal-line influence. As the familial dietary associations are independent of BMI, the observed familial dietary aggregations are not attributable to familial resemblances in anthropometric measures[45].

The Lifeways cohort has previously also shown a relationship between maternal diet during pregnancy and childhood asthma when the children averaged 3 years of age[46], so the cohort patterns of significant maternal gestational influence on outcomes of her child have been consistent over time.

The long-term significance for child health must be speculative. A systematic review and meta-analysis has shown that early-life shared environment does not necessarily translate into a significant contribution to childhood outcomes, such as obesity[47]. While in our analyses the correlations are consistent and statistically significant, they explain only a modest amount of the variance in the fully adjusted models. Children's development is influenced not just by maternal nutrition but also placental development and longer-term energy storage[40,48]. Davey Smith[49] has cautioned that there are inherent methodological difficulties in distinguishing heritable, shared and non-shared environmental characteristics in family studies and noted especially that few studies track these familial associations into adulthood. None the less, if programming in early years is as critical as some authors suggest, then these early dietary patterns are important to document and good-quality mother and child health programmes that focus on child development beginning with the prenatal stage are warranted. Although still rare, some initiatives of such programmes are available (http://www.preparingforlife.ie/)[50]. Barker contends that the critical 1000 d period from pregnancy until the child is 2 years old is of profound importance, but also that grand-maternal influences are strongly influential[40,51–53]. In conclusion, our study provides empirical cross-generation evidence in human families that that is certainly the case in relation to nutrient intakes.

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follow-up, interpretation of the current analysis and to providing critical revision of the paper. C.C.K. has been the principal investigator of all sweeps of the Lifeways cohort study since its establishment, oversaw the present analysis and contributed to the interpretation of findings and drafting of the paper. Each author has seen and approved the contents of the submitted manuscript. The authors greatly appreciate the participation of the Lifeways cohort families.

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