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Positive influences of home food environment on primary-school children's diet and weight status: a structural equation model approach

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Abstract
Objectives: The mechanism by which the home food environment (HFE) influences childhood obesity is unclear. The present study aimed to investigate the relationship between HFE and childhood obesity as mediated by diet in primary-school children.
Design: Cross-sectional data collected from parents and primary-school children participating in the Obesity Prevention and Lifestyle Evaluation Project. Only children aged 9–11 years participated in the study. Matched parent/child data (*n* 3323) were analysed. Exploratory factor analysis underlined components of twenty-one HFE items; these were linked to child diet (meeting guidelines for fruit, vegetable and non-core food intakes) and measured child BMI, in structural equation modelling, adjusting for confounders.
Setting: Twenty geographically bounded metropolitan and regional South Australian communities.
Subjects: School children and their parents from primary schools in selected communities.
Results: In the initial exploratory factor analysis, nineteen items remaining extracted eight factors with eigenvalues >1·0 (72·4 % of total variance). A five-factor structure incorporating ten items described HFE. After adjusting for age, gender, socio-economic status and physical activity all associations in the model were significant ($P<0·05$), explaining 9·3 % and 4·5 % of the variance in child diet and BMI, respectively. A more positive HFE was directly and indirectly associated with a lower BMI in children through child diet.
Conclusions: The robust statistical methodology used in the present study provides support for a model of direct and indirect dynamics between the HFE and childhood obesity. The model can be tested in future longitudinal and intervention studies to identify the most effective components of the HFE to target in childhood obesity prevention efforts.

Keywords
Structural equation modelling
Childhood obesity
Home food environment
Exploratory factor analysis

A diet high in fruit and vegetables and low in non-core foods (those high in fat/sugar/energy and nutrient-poor) is essential to prevent excess weight gain[1]. However, a high proportion of children from Western countries do not meet recommendations for fruit and vegetable intake[2,3] and non-core foods are consumed excessively[4]. Since dietary behaviours track from childhood into adolescence and adulthood[5], targeting dietary behaviours early is important in long-term obesity prevention. Childhood obesity prevention efforts in primary-school children have predominantly targeted the school environment and there is a continued need to enhance understanding in other settings, particularly the home food environment (HFE)[6].

Based on the ecological systems theory[7], the HFE is determined by parents and represents the child's immediate food environment which acts to support or inhibit healthy eating[8]. It is likely that factors influencing child diet are unique at different age stages. While it is acknowledged that in early childhood the child is most dependent on parents with dietary intake largely influenced by the HFE[9,10], the contributing influences of the HFE in primary-school children as age increases are unknown. Competing influences such as the media, peers and school environments can also come into play[7].

A broad range of HFE components have been studied in relation to child dietary intake. In particular, availability

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and accessibility of fruit and vegetables, parental role modelling and parental intake were most consistently related to higher fruit and vegetable intake[10] and child feeding practices such as encouraging intake of healthy foods were related to lower BMI *Z*-scores[11]. Evidence is still inconsistent and limited for most HFE components, especially in relation to non-core foods and children's weight status[10,12]. Studies have conventionally examined individual components of the HFE, ignoring their unique contribution to the overall HFE[12]. Furthermore, although dietary intake is recognized as one of the major determinants of childhood obesity, little is known about its mediating role in the relationship between the HFE and childhood obesity. Many researchers have emphasized the need to develop strong conceptual models, based upon theories and innovative statistical analyses, to elucidate underlying dimensions and accurately assess mechanisms by which the HFE influences childhood obesity via the effects of mediators[6,13,14]. A recent study has documented this complexity of the HFE[15], but there remains a need to generate more compelling evidence in characterizing the HFE in relation to childhood obesity so that childhood obesity prevention efforts can target the most effective and powerful components.

Therefore, the aim of the present study was to explore the direct and indirect relationships between the HFE, child diet and BMI using baseline data collected for an obesity prevention programme.

Methods

Participants

The sample consisted of children in Years 4–6 (predominantly 9–11 years old) and their parents, from schools participating in the Obesity Prevention and Lifestyle (OPAL) Evaluation Project as part of the OPAL programme. OPAL is a large, multi-strategy, community-based childhood obesity prevention programme which commenced in 2009 in South Australia, funded by federal, state and local governments. It is based upon the Ensemble, Prévenons l'Obésité Des Enfants (EPODE) approach[16].

Details of recruitment and data collection for the OPAL Evaluation Project have been reported[17]. In brief, all eligible primary schools from within twenty intervention (OPAL communities) and twenty comparison communities in South Australia were invited to participate. OPAL communities were purposively selected based on higher density of children, higher levels of disadvantage and childhood obesity, and community and local government readiness, through an articulated commitment and contracted political buy-in from local government. This method of selection was used to include those communities at greatest disadvantage but who also had the capacity to deliver the OPAL programme. Comparison communities were matched as closely as possible on maternal education, geographical location, Index of Relative Socio-economic Disadvantage (a measure of socio-economic status based on a basket of income- and education-related measures) and population density of 0–18 year olds.

The recruitment process for obtaining evaluation data was as follows. Schools were invited to participate via a mailed information pack and a follow-up telephone call to the principal. For those schools that agreed to participate, information packs for parents of eligible children were delivered to the school and distributed to students. Parents completed either a hard copy or an online questionnaire. Students whose parents gave consent were measured for height, weight and waist circumference. Parents gave informed consent at the time of administration of the questionnaire and children gave assent before being measured. Details have been previously published[17].

Baseline data were collected between November 2011 and August 2013. A total of 428 schools in the targeted communities were invited to participate. All parents of primary-school children in Years 4–6 within the selected communities were eligible. Child and parent data were collected from schools and households, respectively. A total of 4637 children and 4446 parents were surveyed. Child and parent data were linked and we found 3323 cases in the matched data (Fig. 1).

All procedures involving human subjects were approved by the relevant human research ethics committees from the Flinders University Social and Behavioural Sciences Ethics Committee, the South Australian Department of Health Human Research Ethics Committee, the Aboriginal Health Council Human Research Ethics Committee, the Department of Education and Child Development, and Catholic Education, South Australia.

Measures

Demographics and physical activity (covariates)

The parent and child questionnaires sought details such as the child's age, gender, locality, parental education and estimated annual household income.

Physical activity was reported by parents as the number of times each week the child was involved in organized games, sports or dance outside school hours. As objective measures were not available, this item was used as a proxy variable to represent energy expenditure.

Child's weight status

Height and weight were measured by a trained researcher according to the protocols of the International Society for the Advancement of Kinanthropometry[18]. Final height and weight were determined as the mean of two measures or the median of three measures if the first two measures differed by more than 0·5 cm for height or 0·5 kg for weight. BMI was calculated as weight (in kilograms)

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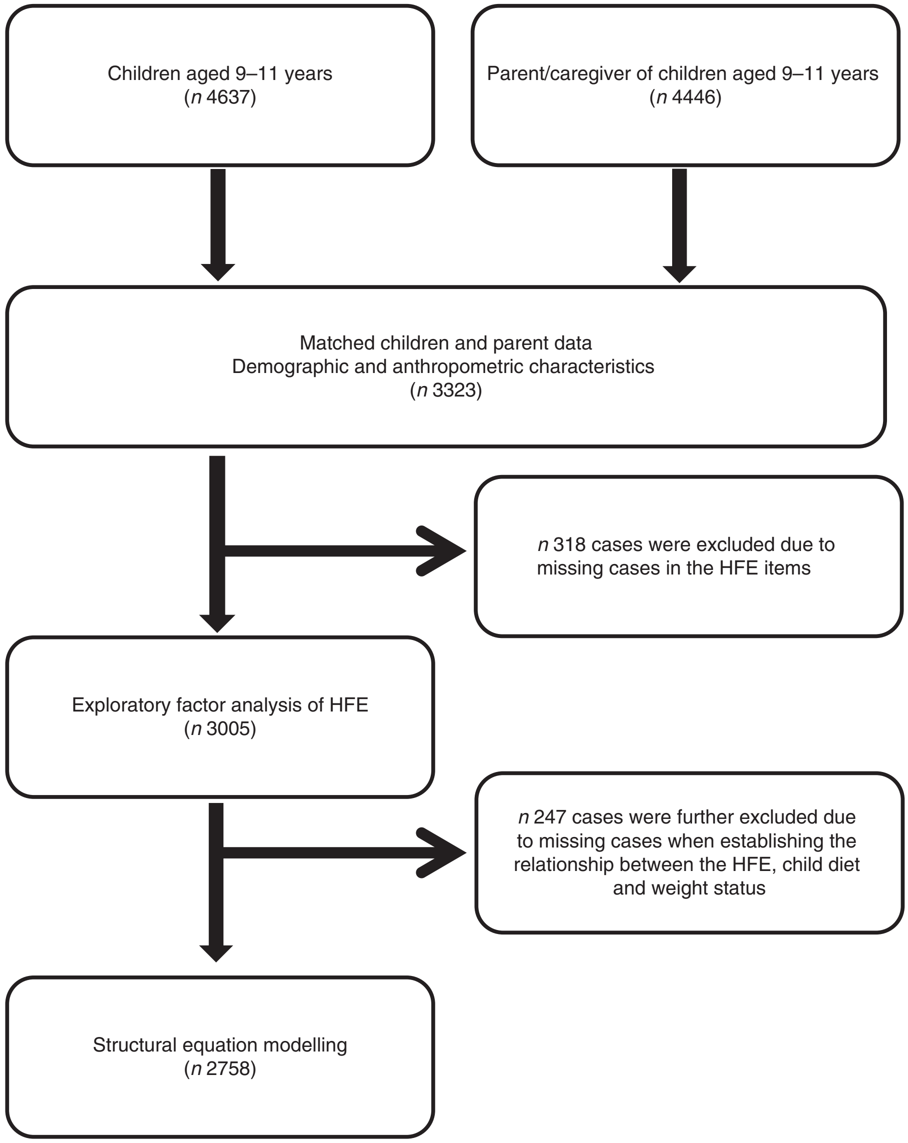

Fig. 1 Flowchart describing sample size in the current analyses (HFE, home food environment)

divided by height (in metres) squared and child BMI *Z*-score, adjusted for age and gender, was calculated using the UK 1990 reference data[19]. Children were categorized as underweight, healthy weight, overweight or obese using the International Obesity Task Force BMI cut-off points[20].

Child diet

Using serving sizes defined by the Australian Dietary Guidelines (serving size =75 g for vegetables, 150 g for fruit and 600 kJ for non-core foods)[21], child diet was estimated from ten questions regarding the number of typical servings of fruit (one item), vegetables (three items) and non-core foods (six items) consumed by the child the previous day. These items were adapted from existing valid and reliable tools[22–24]. Vegetable intake referred to all potato and other vegetables and legumes, excluding fried potato. The six items assessing child intake of sugar-sweetened beverages and sweet and savoury snacks such as confectionery, ice cream, cakes and potato crisps were converted from usual portion sizes (e.g. 50 g packet of crisps) to standardized non-core food servings of 600 kJ[21]. Servings for the six items were combined to give the child's overall non-core food intake. A cut-off point of >9 servings of fruit and vegetables (not including potato) for individual items was applied based on the spread of the data (many cases reporting 9 servings, much fewer for each value greater than 9) and that number of servings is likely to be overestimated[25]. The three food intake variables were dichotomized into whether the child met the diet guidelines (at least two servings of fruit, five servings of vegetables and two or fewer servings of non-core foods daily)[21]. These were scored as 0 = did not meet guidelines, 1 = met guidelines. Scores for each food group were summed to give the child's overall healthy diet score (possible score of 0 to 3).

Home food environment

The HFE was assessed using twenty-one items. These included parent report of the perceived importance of taste, cost, convenience, nutrition, serving size and weight control influencing food purchasing decisions (six items; 4-point Likert scale). Additionally, parental encouragement to eat fruit and vegetables (two items; 5-point Likert scale), using food as a reward and punishment (two items; 5-point Likert scale), frequency of cooking an evening meal and having vegetables at dinner (two items; 5-point Likert scale), frequency of parent eating with the child (one item; number of times/week), child helping to prepare food and snacking without permission (two items; 5-point Likert scale) were also included. Parental intake of fruit and vegetables (two items; number of servings/d) and knowledge of child's fruit and vegetable guidelines (two items; number of servings/d) were dichotomized into meeting guidelines or not. The negative item assessing parental use of food as a punishment for bad behaviour was reverse coded. Items used were adapted from existing valid and reliable tools[24,26] and were selected for the OPAL Evaluation Project as they have been defined as components of the HFE in previous studies[10–12].

Statistical analysis

Means and standard deviations were calculated for continuous data. Proportions were presented as percentages of the respective denominator. The normality assumption was checked visually by frequency histogram and normal *Q*–*Q* plot, and analytically by the Anderson–Darling test. All analysis was performed with two-tailed tests and the level of significance was set at $P<0{\cdot}05$.

Home food environment, child diet and weight status

A total of 318 cases were excluded due to missing cases in the HFE items. After excluding those with missing data, 3005 cases were available for analysis. Exploratory factor analysis (EFA) with twenty-one HFE items was first used to identify underlying constructs of the HFE. Data suitability was assessed using the Kaiser–Meyer–Olkin measure of sampling adequacy and Bartlett's test of sphericity. Kaiser–Meyer–Olkin value >0·5 and Bartlett's test of sphericity test the null hypothesis that no relationship exists between items[27]. Principal component analysis, Kaiser's criterion (eigenvalues >1·0), the scree test and parallel analysis were used to determine the number of factors retained[28]. Only items with absolute factor loadings >0·3 were retained[27].

Due to the ordinal nature of the HFE items, the polychoric correlation of the HFE items was calculated in the EFA. This measures the continuous normal distribution underlying ordinal data[29]. Polychoric correlation was also used to estimate the ordinal α coefficient (ordinal equivalent of Cronbach's α) to determine the internal consistency[30], with values greater than 0·60 being acceptable[27]. This was because EFA and Cronbach's α use Pearson correlation by default, which would inaccurately assume our ordinal items to be continuous[29,31]. These analyses were undertaken using the statistical software package IBM SPSS Statistics Version 22.0. Polychoric correlation and ordinal α were estimated using R Version 3.1.0.

Following EFA, the two-step approach[32] for structural equation modelling (SEM) was used to examine the association between the HFE, child diet and weight status. A total of 247 cases were further excluded due to missing cases when establishing the relationship between the HFE, child diet and weight status. After excluding those cases, 2758 cases were available for all variables used in the SEM analyses (Fig. 1). The first step, confirmatory factor analysis was used to test the measurement model and determine how well factors obtained from EFA fit within the second-order latent variable HFE[33]. In the second step, the corresponding structural equation model linking the HFE to child diet and weight status was tested and two pathways modelled to test the hypothesized direct and indirect relationships. The continuous measure of child BMI was used as a marker of child weight status and the overall child healthy diet score was used to represent child diet. Confounders included age, gender, locality, socio-economic status and physical activity due to the evidence on clustering of health behaviours[34].

Models were estimated with maximum likelihood estimation[35] and assessed using the following goodness-of-fit indices: χ^2 test of model fit, comparative fit index (CFI), root-mean-square error of approximation (RMSEA)[36] and goodness-of-fit index (GFI), where available. Cut-off values ≥0·9 have been suggested to indicate acceptable fit for CFI and GFI[37]. RMSEA value of less than 0·06 has been proposed as reasonable[37]. Model modification was specified based on reviewing item loadings, residual values and modification indices generated. Unstandardized and standardized regression coefficients with *P* values are presented. SEM was carried out using the software IBM AMOS™ Version 19.0.

Results

Sample characteristics

From the 428 schools invited to participate, a total of 4637 children and 4446 parents were surveyed. The child and parent information was collected from schools and households, respectively. Child and parent data were linked and found 3323 cases in the matched data.

Parent and child characteristics are presented in Table 1. Mean age of children was 10·7 (SD 0·9) years, 51 % were females and two-thirds (66·8 %) came from metropolitan regions. Socio-economic backgrounds were varied across

Table 1 Characteristics of parents and their primary-school children aged 9–11 years participating in the Obesity Prevention and Lifestyle (OPAL) baseline evaluation (total male + female = 3323)

Demographic and anthropometric variables	*n*†	Mean, SD or %
Child age (years), mean	3321	10·7
SD		0·87
Child gender		
Female	1704	51·3
Child BMI *Z*-score‡, mean	3101	0·317
SD		1·180
Child BMI (kg/m^2), mean	3103	18·50
SD		3·18
Child weight status§		
Healthy weight	2258	72·8
Overweight	508	16·4
Obese	147	4·7
Underweight	188	6·1
Child physical activity (d/week), mean	3291	2·25
SD		1·71
Socio-economic status‖		
Quintile 1	596	17·9
Quintile 2	635	19·1
Quintile 3	706	21·3
Quintile 4	654	19·7
Quintile 5	730	22·0
Locality		
Metropolitan	2220	66·8
Rural	1103	33·2
Parental education		
Up to Year 12 (high school)	1021	31·7
Technical or trade qualification	1153	35·8
Tertiary qualification or higher	1046	32·5
Parental estimated annual household income ($AU)		
0–35 000	498	15·2
35 001–70 000	802	24·5
70 001–100 000	700	21·3
>100 000	865	26·4
Did not respond	415	12·6

†*n* varies from 3227 to 3295 due to missing data.
‡UK 1990 cut-off points[19].
§International Obesity Task Force cut-off points[20].
‖Determined from Index of Community Socio-Educational Advantage (ICSEA) scores (cut-offs of 981/987/1062/1064). Higher quintile represents a more advantaged background.

income levels and fairly equally distributed across quintiles of disadvantage. A majority of children were of a healthy weight (72·8 %) and about 21 % were overweight or obese.

Children's dietary intake

Parents reported median child fruit intake and vegetable intake of 2·0 servings/d, with 63·1 % of the sample meeting the guideline of at least 2 servings/d for fruit and only 5·7 % meeting the guideline of at least 5 servings/d for vegetables. Median child non-core food intake was 3·0 servings/d with 39·0 % meeting the guideline of two or fewer servings of non-core foods daily. The proportion of children with a diet score of 0, 1, 2 and 3 was 18·2 %, 42·2 %, 33·6 % and 6·0 %, respectively.

Exploratory factor analysis

Initial EFA conducted on twenty-one items resulted in retention of nineteen items which extracted eight factors with eigenvalues >1·0 and explained 72·4 % of total variance. The Kaiser–Meyer–Olkin value (0·663) indicated sampling adequacy and Bartlett's test of sphericity ($P<0·01$) supported factorability of the data set ($\chi^2 = 16\,320·345$, df = 171, $P<0·001$)[27]. Communality values were low for two items ('How often can your child eat snacks and/or sweets without your permission?' and 'How often does your child help prepare food?') which were removed from subsequent analyses. Forced eight-factor rotated solutions were inspected for interpretability. Ordinal α revealed that three factors relating to parental knowledge of child fruit and vegetable guidelines, parental fruit and vegetable intake, and perceived importance of cost, convenience and taste had low internal consistency (ordinal $\alpha = 0·47$, 0·58 and 0·60, respectively). These three factors were excluded. The resultant five-factor solution with twelve items and explaining 53·7 % of total variance showed the best interpretability. Ordinal α coefficient values ranged from 0·61 to 0·80 (Table 2), supporting acceptable internal consistency of items within factors[27]. Factor loadings of the rotated Varimax solution and the proportion of total variance explained for each factor are shown in Table 2.

Structural equation modelling

Measurement model

Low correlations between the five factors allowed inclusion of the second-order latent variable HFE. High residual values indicated that model fit would be improved by removing two items ('Importance of nutrition' and 'I eat what I want my child to eat'). Consequently, the five-factor structure comprising ten items demonstrated an adequate fit to the data (χ^2/df = 104·1/30, $P<0·001$, CFI = 0·985, GFI = 0·992, RMSEA = 0·03; Fig. 2). All five factors were positively and significantly associated with the HFE and in the expected direction. Of these, 'Encouraging fruit & vegetable intake' and 'Cooking vegetables for dinner' were most strongly associated with the HFE. However, 'Instrumental feeding practices' was opposite to the hypothesized direction, which we expected to be negatively related to the HFE.

Structural equation model

When adjusted for age, gender, socio-economic status and physical activity, a more positive HFE was directly associated with a lower BMI in children. In addition, the HFE had a significant indirect effect on BMI through its positive effects on child's diet (Fig. 3). Associations in the model were in the hypothesized direction and were significant ($P<0·05$). Model goodness-of-fit was acceptable (χ^2/df = 521·1/94, $P<0·001$, CFI = 0·923, RMSEA = 0·041), with 9·3 % of total variance in child diet and 4·5 % of total variance in child's BMI accounted for by the model. Standard regression weights and significance values for the structural equation model are presented in Fig. 3.

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Table 2 Exploratory factor analysis with Varimax rotation and internal consistency for factors and their respective items

Factor/factor items	Factor loading	Variance explained (%)	Ordinal α
1. Encouraging fruit and vegetable intake (three items)		22·2	0·75
I eat food I want my child to eat	0·39		
I encourage my child to eat fruit	0·92		
I encourage my child to eat vegetables	0·90		
2. Importance of diet to weight (three items)		10·9	0·71
Importance of nutrition when purchasing food for family	0·55		
Importance of serving size when purchasing food for family	0·83		
Importance of weight control when purchasing food for family	0·86		
3. Eating meals with child (two items)		8·5	0·80
I sit with my child at mealtimes	0·88		
How many times a week does the primary and/or secondary caregiver eat the main meal of the day with your child/children?	0·90		
4. Instrumental feeding practices (two items)		7·7	0·72
I/we use food as a reward for good behaviour	0·87		
I/we withhold food as punishment for bad behaviour	0·88		
5. Cooking vegetables for dinner (two items)		6·5	0·61
How often do you or another adult in the house cook an evening meal?	0·83		
At home we have vegetables at dinner	0·63		

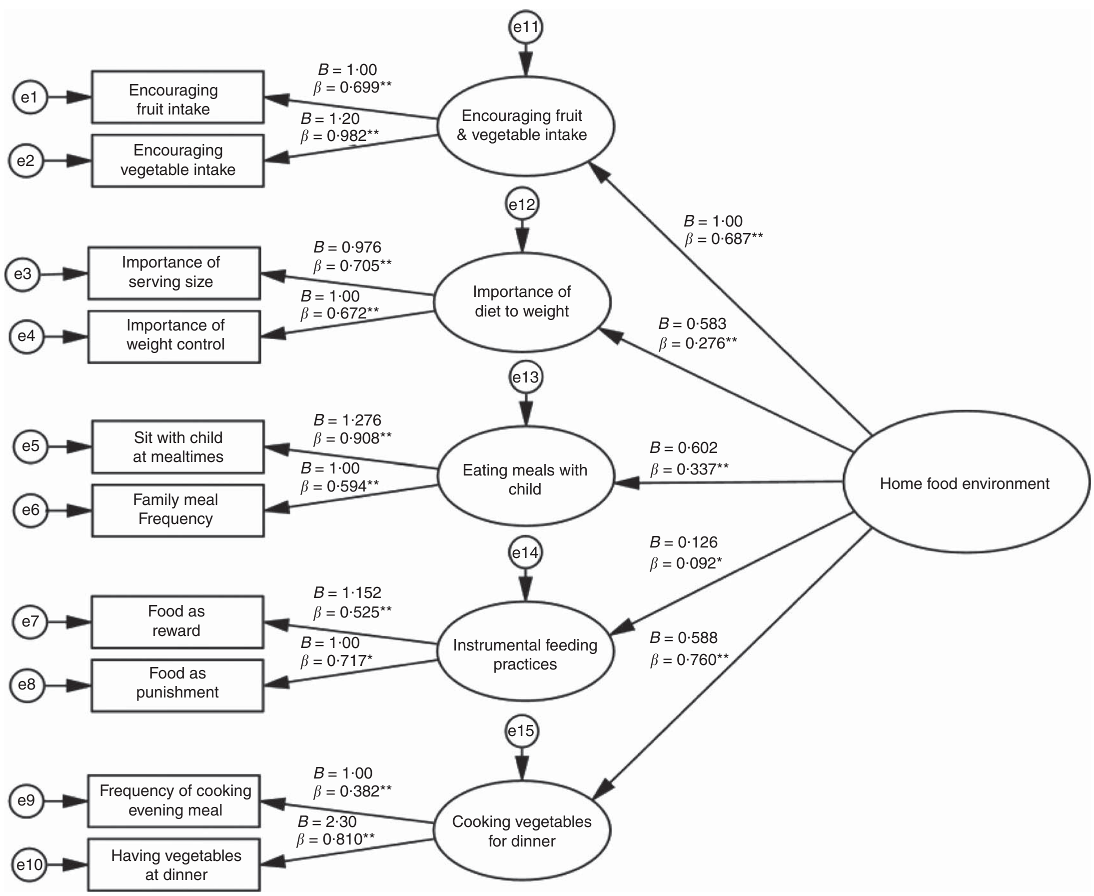

Fig. 2 Final measurement model between the home food environment, including measured variables (rectangles) and latent variables (ovals), and child diet and weight status using data collected from parents and their primary-school children aged 9–11 years participating in the Obesity Prevention and Lifestyle (OPAL) baseline evaluation (*n* 3323), South Australia, November 2011–August 2013 (*B*, unstandardized regression weight; *β*, standardized regression weight; e, error terms; $^{*}P<0·05$; $^{**}P<0·001$)

Discussion

This is one of few studies that have undertaken complex analyses in examining various components of the HFE simultaneously. Our hypothesized model was a good fit. We found that a more positive HFE may be protective of childhood obesity through two pathways: (i) the direct association between the HFE and child BMI; and (ii) indirectly through the mediation by child diet. After adjusting for confounders, a favourable HFE appears to be

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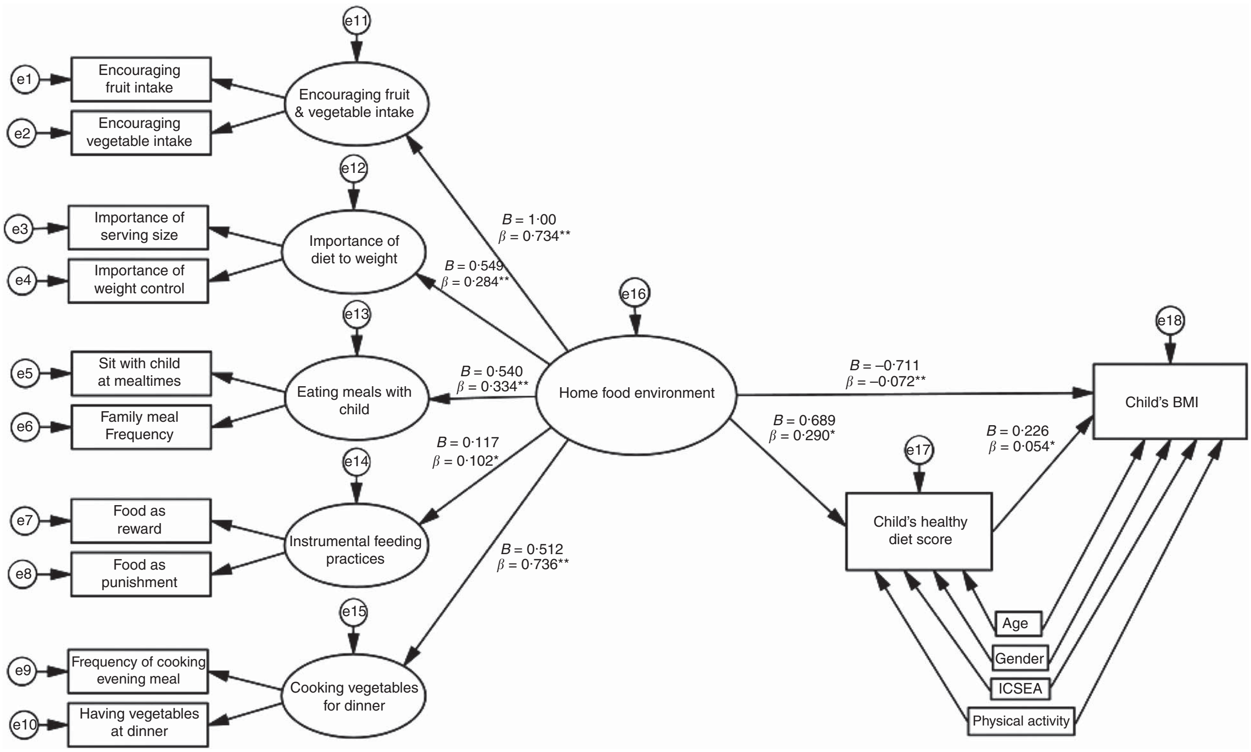

Fig. 3 Final structural equation model of the home food environment on child BMI as mediated by child diet using data collected from parents and their primary-school children aged 9–11 years participating in the Obesity Prevention and Lifestyle (OPAL) baseline evaluation (*n* 3323), South Australia, November 2011–August 2013 (rectangles, measured variables; ovals, latent variables; *B*, unstandardized regression weight; *β*, standardized regression weight; e, error terms; ICSEA, Index of Community Socio-Educational Advantage (measure of socio-economic status); $^*P<0{\cdot}05$; $^{**}P<0{\cdot}001$)

most strongly influenced by parental encouragement and parental facilitation through cooking vegetables for dinner.

'Encouraging fruit & vegetable intake' by parents was a strong component of the HFE influencing child diet. This supports previous studies showing parental influence on child diet through an encouraging feeding style[37]. Lack of parental encouragement and support is commonly cited as a major barrier to having a healthy diet by children[38]. According to Social Cognitive Theory[39], outcome expectancies are used to interpret information from the environment and directly influence behaviours. Therefore, encouragement may provide positive reinforcement by persuading and motivating children to consume more fruit and vegetables[40] and enabling the development of a positive attitude towards eating fruit and vegetables[41].

The two-item factor 'Cooking vegetables for dinner' was also strongly associated with the HFE. No published studies have specifically examined this factor as part of the HFE as it was a unique factor obtained from EFA in our study. Nevertheless, cooking vegetables during evening meals can facilitate consumption by increasing availability and accessibility[10]. Moreover, home-cooked meals which are usually more nutritious and balanced are mostly consumed at dinner[42]. It may also be that dinner time is a major setting for parents to role model and encourage healthy eating[43], reinforcing our previous discussion. These findings are important as the rising trend of maternal employment, particularly as children get older, is likely to change family meal routines and limit healthy eating at home. Parents frequently cite a busy lifestyle as a barrier to healthy eating[44], with children who do not eat family meals more likely to consume fried food away from home[43].

It was of interest that 'Instrumental feeding practices', describing parents' use of food as reward and punishment, was positively, although weakly, associated with the HFE in our study. Other studies have found instrumental feeding practices to be effective in promoting fruit and vegetable consumption in the short term through increased exposure and liking for the food, and that it can reinforce behaviour and facilitate a healthier diet[45]. However, Kroller and Warschburger[46] reported that such practices were associated with increased preference for and intakes of energy-dense and nutrient-poor foods such as desserts, sweet and savoury snacks, and sugar-sweetened beverages[47]. Such practices have been associated with weight gain in the long term[11,48]. Gray's neuropsychological model of reinforcement sensitivity postulated that parental use of such practices on their children can lead to an increased activation of impulsivity and reward responsiveness, leading to poor inhibition of food intake and an increased tendency to overeat[49].

It should be noted the sample size of these studies was less than 400 and they were conducted predominantly in early childhood. Thus, it may be that older children such as those in our study are less susceptible to the negative impacts of such practices. More longitudinal studies are needed to ascertain the impact that instrumental feeding practices have on child diet and weight in the long term.

Weak, positive associations with the HFE were obtained for the two factors 'Importance of diet to weight' and 'Eating meals with child', regarding parental perceptions and presence at mealtimes. Their role in the HFE is still unclear due to limited available research. Further studies are required to generate more compelling evidence.

The mechanisms for the direct association between the HFE and children's BMI are unclear, mainly due to the scant literature examining the overall HFE and those examining the association between composite measures of the HFE and child's weight status. Efforts to compare results were limited due to different operationalizations and combinations of HFE components examined. Most previous studies have examined individual aspects of the HFE and their association with dietary intake. Moreover, aspects of the HFE may not relate directly to the limited items of dietary intake that were assessed in the present study but may influence overall energy intake. It may be that parents who provide a healthier food environment may have healthier dietary and lifestyle practices, and their children are less sedentary, thus influencing BMI. One recent study in 157 children aged 5–10 years[15] used SEM to show that the overall HFE is positively associated with child's fruit and vegetable intake, but only child feeding practices and parent diet quality had an indirect effect on intake through the HFE. That study did not hypothesize the overall HFE or child's diet to be related to weight status. In another Australian study of 395 children aged 9–13 years a supportive HFE predicted child's fruit and vegetable intake[50]. Our study provided additional insight by including a measure of non-core foods as part of child diet. Schrempft *et al.*[51] found no significant association between the overall HFE and 4-year-old children's BMI, postulating that the influence of the HFE on BMI may emerge only later in development. Similarly, MacFarlane *et al.*[52] also found no associations with BMI in younger children (5–6 years), but some associations in older children (10–12 years), a finding which is consistent with our study. It should be noted that comparison of results was limited due to different operationalizations and combinations of HFE components examined.

Our findings are consistent with an ecological systems theory[7], showing that parents can influence their primary-school child's weight through the HFE they provide. The weak positive associations and relatively small proportion of explained variance in our study are in accordance with other studies which have also applied SEM and included a measure of BMI[46,53]. This low variance reflects the increasing autonomy and independence of the child and the influence of external environments as they grow older. While there may be other factors in the HFE that have not been explored in our model, our results provide important insight. To our knowledge, this is the first study to have investigated the direct and indirect effects between the HFE, child diet and weight status.

The present study is strengthened by its large community-based sample which is representative of all social strata and the objective anthropometric measures collected. The use of statistically robust SEM supports our model, which can form the basis for generating hypotheses to be tested in future studies. However, findings should be interpreted in light of study limitations. A limitation of the study is that data used were parent-reported and subject to social desirability bias. Dietary intake assessment was limited to only a few items and not necessarily reflective of overall energy intake. It is likely that inclusion of total intake (beyond the scope of the OPAL programme evaluation) in the model would result in a different outcome but we were not able to assess this. In addition, the physical activity variable used was a crude estimate of energy output as more objective measures (such as those derived from the use of accelerometers) were not available. The low response rate from primary-school parents and students also limits generalizability, and the cross-sectional design means that causality cannot be inferred. Lastly, our model may be limited as there may be other important aspects of the HFE not included.

The aetiology of childhood obesity is complex and multifactorial. Our study has shown the usefulness of applying innovative statistical analyses such as SEM to elucidate the dynamics within the HFE. Although its application in childhood obesity research has increased recently, more work is encouraged. It is imperative that future research be based upon an *a priori* theoretical or conceptual model to enhance understanding of mechanisms involved and provide consistency in defining the home environment. Such models should include a wider range of HFE components, such as parental rules and restrictions and child feeding styles, and also seek to examine associations between a composite measure of the home environment and BMI over a longer time period, while taking external influences into account, so as to generate more compelling evidence[13]. Most previous research has focused on the HFE among pre-school children but our results show that it is also important for primary-school children. Future childhood obesity prevention initiatives should involve parents and include strategies that promote a positive HFE, such as parenting practices that encourage fruit and vegetable consumption, and address barriers to the provision of home-cooked family meals.

Conclusion

Our results contribute to growing evidence on the role of the HFE in childhood obesity and have highlighted the important influence of the HFE on child diet, which affects childhood obesity risk. We provide support for a model of the direct and indirect dynamics within the HFE. A more positive HFE can be protective of childhood obesity and this can be through a healthier diet. Parents of primary-school children can facilitate healthy eating at home by cooking and eating home-cooked family meals with their child and by encouraging fruit and vegetable intake. Such practices have potential in preventing excessive weight gain.

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