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Parent–child dietary intake resemblance in the United States: Evidence from a large representative survey

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Abstract

We studied the association in dietary intakes and patterns between parents (aged 20–65 years) and their children (aged 2–18 years), using nationally representative data collected by the US Department of Agriculture (USDA) in the Continuing Survey of Food Intake by Individuals 1994–96. We analyzed two 24-h recall dietary data for 1061 fathers, 1230 mothers, 1370 sons and 1322 daughters. All analyses adjusted for sampling design complexity. We assessed multivariate-adjusted parent–child correlations in selected nutrients, food groups and overall dietary quality assessed using the new USDA 2005 Healthy Eating Index score (HEI_n). The parent–child correlations were weak or moderate (0.20–0.33) for most intake measures. There were clear patterns of interaction with gender dyads in the intakes of calcium and dairy products ($P < 0.05$ for dyad × parental intake), whereby multivariate-adjusted correlations in mother–daughter or mother–child dyads were significantly stronger compared to their father–child counterparts. The reverse was true for multivariate-adjusted correlations in HEI_n. Hispanics and other ethnic groups had significantly stronger resemblance than Non-Hispanic whites and blacks in soft drinks and HEI_n. Resemblance in general was stronger among older children, though the reverse was true when considering agreement in HEI_n's upper quintile. The influence of family income and parental education on the resemblance was small. In conclusion, parent–child dietary resemblance in the US is relatively weak, and varies by nutrients and food groups, and by the types of parent–child dyad and population groups. Factors other than parental eating behaviors seem to play an important role in affecting American young people's dietary intake.

Keywords

USA; Dietary; Diet; Dietary quality; Ethnicity; Food intake; Familial aggregation; Child; CSFII; Gender

Introduction

Children and adolescents adopt certain dietary behaviors that previous studies suggested might track into adulthood (Mikkila, Rasanen, Raitakari, Pietinen, & Viikari, 2005; Wang, Bentley, Zhai, & Popkin, 2002). It is therefore important to promote healthy eating among the youth to prevent occurrence of chronic conditions later on in life, particularly obesity, cardiovascular disease, type 2 diabetes and certain types of cancer. There are many ways by which promoting healthy eating among the youth can be achieved. However, one of the suggested means was to educate the parents and enhance their awareness about their own health as well as that of

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their children. This approach assumes that parents are gate keepers and role models for their children and that their nutrition beliefs and behaviors may actually influence that of their offspring.

However, there is a growing body of evidence suggesting that dietary intake among the youth moderately resembles that of their parents, particularly in the United States. To date, around fifteen such studies have been conducted (Adelekan & Adeodu, 1997; Cullen, Lara, & de Moor, 2002; Feunekes, de Graaf, Meyboom, & van Staveren, 1998; Feunekes, Stafleu, de Graaf, & van Staveren, 1997; Fisher, Mitchell, Smiciklas-Wright, & Birch, 2002; French, Story, & Jeffery, 2001; Galloway, Fiorito, Lee, & Birch, 2005; Laskarzewski et al., 1980; Oliveria et al., 1992; Park, Yim, & Cho, 2004; Patterson, Rupp, Sallis, Atkins, & Nader, 1988; Perusse et al., 1988; Rossow & Rise, 1994; Stafleu, Van Staveren, de Graaf, Burema, & Hautvast, 1994; Vauthier, Lluch, Lecomte, Artur, & Herbeth, 1996), seven of which were carried out in non-representative samples within the United States, limiting the ability to produce national population estimates. A number of published studies support familial resemblance in dietary patterns (Adelekan & Adeodu, 1997; Laskarzewski et al., 1980; Oliveria et al., 1992; Patterson et al., 1988; Perusse et al., 1988; Rossow & Rise, 1994; Stafleu et al., 1994; Vauthier et al., 1996). On the other hand, other studies show that the association is either very weak or non-existent (Cullen et al., 2002; Feunekes et al., 1997, 1998). The weak or non-existent association is likely related to young people's eating patterns being affected by a myriad of complex factors, with the influence of parents and the family environment being only one of them (French et al., 2001; Popkin, 2006; Vereecken, Inchley, Subramanian, Hublet, & Maes, 2005).

To our knowledge, previous research has not examined familial resemblance in dietary intake using nationally representative data in the United States. Most previous studies are based on small and local samples, and the possible regional and between-group differences in the resemblance between child and parental dietary intakes could affect our understanding of the relationships at the national level. It is crucial to assess such an interrelationship to guide future dietary intervention programs which to date have been strictly targeted towards schools among other non-household settings (Abdel Gawwad, Fetohy, Fiala, Al Orf, & Al Saif, 2006; Agozzino, Esposito, Genovese, Manzi, & Russo Krauss, 2007; Fahlman, Dake, McCaughtry, & Martin, 2008; Janega et al., 2004; Nicklas et al., 1997; Podrabsky, Streichert, Levinger, & Johnson, 2007). Although previous studies assessed nutrient and food group associations between family members, none have evaluated associations in overall diet quality.

The present study examined parent–child dietary pattern interrelationships, using nationally representative data in the United States, and thus produced national population estimates. We estimated correlations in selected nutrients and food groups and assessed the intra-familial clustering in healthy behaviors through adherence to dietary guidelines (i.e., overall dietary quality) between parent and child dyads. We also tested for effect modification of these associations by selected individual and household-level characteristics.

Methods

Continuing Survey of Food Intakes by Individuals (CSFII) data

Data from the US Department of Agriculture (USDA) Continuing Survey of Food Intakes by Individuals (CSFII) 1994–96 were used (US Department of Agriculture ARS, 1994–96). A nationally representative multi-stage stratified sample of 16,103 non-institutionalized persons aged 0–90 years contained information about dietary intake (by one or two nonconsecutive 24-h recalls that were 3–10 days apart); socioeconomic, demographic and health parameters (Tippett & Cypel, 1997).

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Study population

Parent sample—Among the 16,103 CSFII respondents, we included 9872 who were 20 years or older and had complete data on day 1 of recall. We excluded those over the age of 65 years ($n = 2127$) and those who completed only one 24-h dietary recall ($n = 414$), which resulted in a final sample of 7331 adults (aged 20–65 years, 3721 men and 3610 women) aged 20–65 years with both 24-h dietary recalls completed. Among them, 86% ($n = 6303$) were either the respondent or the spouse of the respondent and hence were eligible to be included in our sample. All other adults were other family members including, those having a "child of head of household" status. The rationale for excluding elderly subjects was that their dietary intake might differ from those of other family members due to illness or general poorer health.

Children sample—Children were matched to the adult sample by family relationship. Only those children aged 2–18 years with both completed 24-h recalls were considered. After matching parents with their children, the final sample sizes were: 2291 parents (1061 fathers and 1230 mothers) in 1473 households; 2692 children (1370 sons and 1322 daughters); a total of 4244 parent–child dyads (1156 mother–son, 1128 mother–daughter, 982 father–son and 978 father–daughter).

Measures

Dietary intakes and dietary quality indicators—Average dietary intakes from the two 24-h recalls were used. Among young children aged 2–9 years old in the total CSFII 1994–96, proxy response was the common pattern (only 7% and 11% self-reported dietary intake on days 1 and 2, respectively). Food groups and nutrients considered included energy (kcal/day), fat (g/day and % of energy), saturated fat (g/day and % of energy), cholesterol (milligrams; mg/day), sodium (mg/day), fiber (g/day), calcium (mg), sugars and candies (g/day), fruits and vegetables (g/day), dairy products (g/day), total and unsweetened soft drinks (g/day). The food groups and nutrients were selected because of their important influence on health outcomes, including obesity and the metabolic syndrome.

Diet quality index (HEI_n)—To assess the overall quality of diet, we applied the newly revised USDA 2005 Healthy Eating Index (HEI_n) (Britten, Marcoe, Yamini, & Davis, 2006; U.S. Department of Agriculture (USDA), 2005), which incorporated the new dietary recommendations and energy-adjusted all of its individual components (Britten et al., 2006). For many of the food group criteria, Mypyramid serving estimates rather than grams were used as made available by the USDA website http://www.ars.usda.gov/Services/. The HEI_n score consisted of the sum of scores on twelve components covering dietary recommendations in terms of nutrient and food group intakes scored differently (0–5 to 0–20) according to their importance to overall dietary quality. These were developed based on a large body of evidence as outlined elsewhere (Britten et al., 2006). Appendix A table shows the criteria used for scoring each component. The HEI_n could range between 0 and 100. Likely HEI_n is a more robust measure of overall diet pattern (and dietary quality) than intakes of individual nutrient or food groups.

Other covariates—Covariates in our statistical models were included as either potential confounders or effect modifiers as they were previously shown to affect dietary intakes, particularly among adults (Beydoun, Powell, & Wang, 2008; Beydoun & Wang, 2008a, 2008b): age and gender of parents and children, parental ethnicity (Non-Hispanic whites, Non-Hispanic blacks, Hispanics, Others), education (years), employment status (yes vs. no), smoking status (current smoker vs. not), self-rated health (poor health vs. not), physical activity (currently sedentary, measured as never or rarely participates in vigorous activities vs. not), and number of chronic conditions (>2 conditions vs. not), household poverty income ratio category (PIR: 0–129 (poor); 130–299 (near poor) and 300+ (not poor)), and contextual factors including geographical region and degree of urbanization. Other covariates in children included reported body mass index (BMI) and reported physical activity (same categories as for adults).

Statistical analysis

All analyses were conducted using survey-related commands in STATA release 9.0 (STATA, 2005), which take complex sampling design into account (multi-stage stratified cluster as opposed to simple random sample) and produce nationally representative estimates of means, proportions and regression coefficients as well as correct estimates of standard errors (Lohr, 1999). In all tests, $P < 0.05$ was considered as statistically significant.

We first conducted descriptive analysis to describe the populations' characteristics. Further, and to best examine commonality in dietary intakes between parents and children, we estimated adjusted Pearson correlation coefficients as approximated by the linear regression coefficient between parent and child intakes expressed as standardized *z*-scores. This approach allows us to account for complex survey design effects and also to control for potential confounders.

We also estimated overall observed agreement, expected agreement and the kappa estimate, which measures agreement beyond that by chance and is calculated based on observed agreement, expected agreement and the weight matrix. We presented both unweighted and weighted agreement percentages and their corresponding kappa values (Agresti, 1996; Wang & Wang, 2003). Note that the weight matrix and kappa estimation formula are presented in Appendix B. Next, we conducted multivariate logistic regression analysis to test whether "high diet quality" (top quintile or 80th percentile of HEI_n) among parents predicted "high diet quality" among their children (i.e., agreement of having a healthy diet) controlling for other covariates.

Further, we assessed effect modifications, e.g., by gender of parents and children based on a statistically significant interaction term (DQI_{pj} × dyad) introduced into the multivariate regression models for parent–child associations. The other potential effect modifiers we tested included ethnicity, children's age, household income (PIR), and parental education. In all tests, a *P*-value of <0.05 was considered as statistically significant including interaction terms.

Results

Study population characteristics

The distribution of selected characteristics of parents and children stratified by gender is presented in Table 1. In general, fathers were older than mothers by an average of two years, and an appreciably higher percentage of them were employed (93% vs. 67%). In terms of health factors, while men had a higher BMI, women were more likely to assess their physical activity level as sedentary. Among children, mean age and BMI did not differ by gender, although physical activity did, with a higher proportion of girls reporting a sedentary lifestyle.

Dietary intake and patterns

The mean and standard errors of nutrients, food groups and the HEI_n are presented for each selected family member according to status and gender (Table 2). In general, fathers had higher energy intake and absolute intakes of most foods and nutrients as well as relative intake of fat and saturated fat compared to mothers ($P < 0.05$), with the exceptions of sugars and candies and unsweetened soft drinks ($P > 0.05$). In addition, HEI_n indicated a higher overall quality of dietary intake among mothers compared to fathers (50.3 vs. 48.4; $P < 0.05$). While gender differences between parents were significant for all nutrients, food groups and the HEI_n, differences between sons and daughters in intake of some nutrients (fat and saturated fat as %

energy), food groups (sugars and candies, fruits and vegetables and unsweetened soft drinks) and the HEI_n were non-significant ($P > 0.05$).

Parent–child dietary correlations: overall and subgroup analysis

Parent–child correlations are presented as standardized regression coefficients adjusted for design complexity in Table 3. Analysis was further stratified first by gender dyads and second by selected characteristics including parents' ethnicity, child's age and the household's PIR group. It is worth noting that in many cases, adjustment for potential confounders yielded markedly attenuated correlations (by >10%), when comparing crude to adjusted correlation coefficients (data not shown). Overall, parent–child correlations in nutrient and food group intakes ranged from <0.10 for fat and saturated fat as % energy to around 0.30 for fruits and vegetables and cholesterol intakes. Most of the multivariate-adjusted correlations were in the weak to moderate range (0.20–0.30). In addition, the adjusted correlation between parents and children in terms of overall diet quality was 0.26 ($P < 0.01$). Looking at the relationship by gender dyads, there were clear patterns of interaction in the case of calcium and intake of dairy products as well as the overall diet quality index (HEI_n). In most of these cases, except the HEI, mother–child multivariate-adjusted correlations were significantly stronger compared to their father–child counterparts ($P < 0.05$ for dyad × dietary $intake_{(parent)}$ interaction term). For all other nutrients and food groups, interaction patterns by gender dyads were less evident and non-significant.

When examining the strength of dietary pattern correlations between parents and their children across ethnic groups, we found that for overall dietary quality (HEI), Hispanics and "other" ethnicities had a significantly higher correlation compared to Non-Hispanic whites and blacks (0.36 and 0.38 vs. 0.30 and 0.27 respectively; $P < 0.10$ for ethnicity × dietary $intake_{(parent)}$ interaction term). Ethnic group differences in the strength of parent–child correlations were also noted for total soft drinks, in which the "other" minority groups had the strongest correlation.

The strength of parent–child correlations varied by child's age as well for most nutrients, food groups and for the diet quality index, with few exceptions such as fruits and vegetables and sugars/candies. In most cases, children older than 10 years had a stronger association in dietary intake with their parents compared to younger children aged 2–10 years. The difference in correlation was particularly marked in the case of total and saturated fat intakes. However, when looking at overall diet quality, child–parent correlation in HEI_n was similar for both younger and older children with no statistically significant interactions observed.

In terms of poverty income ratio, only one statistically significant interaction was noted for unsweetened soft drinks. In particular, the non-poor segment of the population exhibited a stronger parent–child correlation compared to the poor and near poor groups.

Agreement in diet quality between parents and children based on quintiles: overall and subgroup analysis

Table 4 shows the observed and expected agreement between parents' and children's HEI scores based on quintiles. In general, the agreement was weak as indicated by both the weighted and unweighted results (note both have accounted the complex sampling design effects). The observed unweighted agreement percentages ranged between 26.0% for father-son dyad and 33.1% for 'other ethnic groups,' while the weighted ones were 58.9% for father–son dyad and 64.6% for Hispanics. Weighted kappa estimates varied between groups and were the lowest among "other ethnic group" (kappa = 0.07) and highest among Hispanics (kappa = 0.28). All weighted kappas were significantly different from zero.

Further, we studied the child–parent association in high diet quality (top quintile, i.e., having a healthy diet) and adjusted for potential confounders using logistic regression models (see Table 4). In general, the odds for children having a healthy diet (HEI_n score range: 61–81) increased by three-folds when their parents did so (HEI_n score range: 62–86). There was a significant effect modification by age of the child, with the odds of upper quintile concordance between parents and children being increased to a greater extent among young children aged 2–10 years compared to older children (>10 years) (4.05 vs. 1.55, $P < 0.05$ for $age_{(\text{-child})} \times HEI_{(\text{parent})}$ interaction term). All other comparisons of odds ratios across strata did not indicate any interactions.

Testing potential influence of parental education on the child–parent dietary intake resemblance

We suspected that parental educational attainment might influence dietary choices and food habits, and thus, might affect the child–parent dietary intake resemblance. We tested parental education in years as an effect modifier, but overall, our analyses indicated that the influence of parental education on the resemblance was small. The interaction term with parental dietary intake in our models was significant only for cholesterol intake indicating that the correlation becomes significantly stronger but only by a value of 0.02 ($P = 0.039$) with each year of educational attainment by parents. We also conducted this analysis separately for each dyad. However, only for father–son pairs, father education significantly reduced the correlation by −0.03 per year ($P = 0.025$) for total saturated fat.

Discussion

To our knowledge, the present study is the first attempt to assess familial resemblance in dietary intakes and patterns, particularly between parents and their children using nationally representative data collected in the U.S. It is also the first to examine differences in the resemblance between groups (e.g., different parent–child dyads, ethnicity, family income, child age) in various dietary intake variables including nutrients, food groups and overall dietary quality.

Our study has several main interesting findings. First, the results provide insight into the extent of parental influence on children's dietary behaviors and suggest that there is moderate resemblance, in terms of correlations, in intake and patterns within families. Similar to findings from previous studies of selected samples, the correlations are moderate (0.20–0.33) (Adelekan & Adeodu, 1997; Cullen et al., 2002; Feunekes et al., 1997, 1998; Fisher et al., 2002; French et al., 2001; Galloway et al., 2005; Laskarzewski et al., 1980; Oliveria et al., 1992; Park et al., 2004; Patterson et al., 1988; Perusse et al., 1988; Rossow & Rise, 1994; Stafleu et al., 1994; Vauthier et al., 1996), suggesting that young people's dietary intakes are influenced by other factors in addition to household factors (Boutelle, Fulkerson, Neumark-Sztainer, Story, & French, 2007; Hanson, Neumark-Sztainer, Eisenberg, Story, & Wall, 2005), such as community and school (Fitzgibbon & Stolley, 2004; Jahns, Siega-Riz, & Popkin, 2001; Zizza, Siega-Riz, & Popkin, 2001) food environments, peer influence (Salvy, Romero, Paluch, & Epstein, 2007), television viewing and programs (Ayala et al., 2007; Boynton-Jarrett et al., 2003; Matheson et al., 2004), as well as individual factors such as self-image and self-esteem (Satia, Kristal, Curry, & Trudeau, 2001; Shariff & Yasin, 2005). For example, one of the earliest US studies estimated correlations between one parent and one of their children (aged 6–19 years) in terms of several nutrient intakes and found them to be 0.26, 0.30, 0.15 and 0.22 for energy, carbohydrates, saturated fat and polyunsaturated fat intake ($P < 0.05$), respectively (Laskarzewski et al., 1980). Another more recent US study (Oliveria et al., 1992) indicated that correlations were particularly high in fat intake as well as monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs) when mother–child relationships were considered (*r* ranged between 0.31 and 0.51, $P < 0.05$). These correlations were shown to be weaker in father–child associations ($r = 0.10–0.22$) with the exception of saturated fat ($r = 0.40$, $P < 0.05$).

In addition, our study suggested variations in the differences of HEI across gender dyads and in the correlations in terms of food groups and nutrients both across gender dyads and other selected characteristics. In particular, there were clear patterns of interaction in the case of intakes of calcium and dairy products, whereby in mother–daughter or mother–child in general, multivariate-adjusted correlations were significantly stronger compared to their father–child counterparts. Several prior studies came to similar conclusions (Feunekes et al., 1998; Laskarzewski et al., 1980; Oliveria et al., 1992; Park et al., 2004; Stanton, Fries, & Danish, 2003). At least two previous studies found that daughters had a higher resemblance to their parent's diet compared to sons (Feunekes et al., 1997; Park et al., 2004). Other studies found no consistent patterns across macronutrients in terms of gender dyads (Vauthier et al., 1996). However, our findings indicated that the mother–daughter association in overall diet quality as measured by HEI_n was the weakest among the four dyads, particularly after adjusting for child and parent characteristics.

We also found ethnic differences – Non-Hispanic blacks consistently exhibiting a weaker association than other ethnicities, specifically for soft drinks and the overall HEI_n score. This may suggest that environmental factors may be more influential on certain ethnic groups especially in terms of overall diet quality, rendering the association between parents and their children weaker than expected in the total population. Another study came to a similar conclusion whereby white parent–child dyads had significantly higher correlations of fat and fiber intakes compared to African-American dyads (Stanton et al., 2003). However, an earlier study reported that parent–child nutrient correlations were particularly strong when parents were black mothers aged over 40 years, particularly in the case of energy and carbohydrate intakes (Laskarzewski et al., 1980). It is possible that the parent–child resemblance in dietary intakes might have changed over time, and the changes vary across population groups.

Our findings based on this national sample show that the influence of family SES on the parent–child resemblance in dietary intakes is small. We found no effect modification by family income and a very weak one by parental education independently of other demographic factors for HEI_n, in which most of the food groups and nutrients were considered. To test effect modification of parental educational attainment independently of income, we further conducted a sensitivity analysis for all parent–children dyads as well as for each dyad separately. Overall, the results suggested that its influence on the resemblance was small and only significant for cholesterol intake.

Furthermore, we suspected that older children tend to have weaker resemblance with their parents' intakes compared to their younger counterparts due the greater possible influence of factors outside the home based on some previous studies (Adelekan & Adeodu, 1997; French et al., 2001; Laskarzewski et al., 1980; Oliveria et al., 1992; Patterson et al., 1988; Perusse et al., 1988; Popkin, 2006; Rossow & Rise, 1994; Stafleu et al., 1994; Vauthier et al., 1996; Vereecken et al., 2005). In fact, stronger peer influence may affect children as they grow older and reach the adolescent stage who then experience more autonomy from parents when making eating choices (Worthington-Roberts & Rodwell Williams, 1999). In our study, we only found a stronger resemblance in older children when HEI_n was assessed as a binary variable in terms of agreement between parents and children within the upper quintile. However, for some other individual nutrients and food groups, older children tended to have stronger multivariate-adjusted correlations, and there was no marked age interaction in terms of continuous HEI_n. These mixed findings may be due to the fact that HEI includes a large number ($n = 12$) of components and the related cutpoints were energy-adjusted, while most individual food groups and nutrients considered were not. Day-to-day measurement errors might affect the individual dietary variables more seriously than on the HEI, and the influences varied by nutrients and food groups. Those findings also suggest that if one only focused on selected individual food groups or nutrient intakes (particularly those unadjusted for total energy intake) as opposed to overall dietary quality, the conclusion regarding familial dietary resemblance may be biased. This sheds important insight on the interpretation of previous findings and helps to guide future studies.

Finally, our analysis for parents and children separately also shows that their average intakes of several nutrients including fiber and calcium were inadequate while some were too high (e.g., sodium) compared to the 2005 daily recommended intakes (DRIs) (Institute of Medicine, 2005). The related DRI for fiber was 19–38 g/day for children depending on gender/age group and 21–38 g/day among adults depending on gender/age group; for calcium, 600–1000 mg/day for children and 800–1500 mg/day for adults; and for sodium, 700 mg per 1000 kcal diet, i.e. around 1540 mg for a 2200 kcal diet.

Our study has a number of strengths. First, we used nationally representative data and obtained population estimates of correlations that account for sampling design complexity. Second, we examined resemblance using the new 2005 USDA Healthy Eating Index, which helps assess overall dietary quality. Finally, we assessed effect modification by gender parent–child dyads and selected SES characteristics with sufficient sample heterogeneity to test effect modifiers.

Despite its strengths, our study had its limitations. First, the data were approximately 10 years old, but available data from other more recent nationally representative surveys such as the National Health and Nutrition Examination Surveys do not allow for such analyses based on the released data. We suspect it is unlikely that the association might have changed substantially over time over the past two decades, and our findings could assist in testing such time trends in parent–child correlations once more recent comparable data become available. Second, dietary intake was based solely on two 24-h recalls. Random intra-individual variations across the targeted time frame of intake may attenuate parent–child correlations (Oliveria et al., 1992). In addition, for very young children (2–6 years), reporting of dietary intakes was mostly by proxy which may bias the results away from the null (Tippett & Cypel, 1997). However, our results suggest that younger children, at least for most food groups and nutrients selected, are less likely to follow their parental dietary patterns than older one. Hence, the interaction with age may be even stronger. Despite these expectations, one has to be cautious about the impact of misreporting of dietary intake by parents for smaller children as well as the measurement error in reporting dietary intake by older children which may not be uncorrelated with their actual true intake. In addition, dietary intake cannot be assumed to be of equal validity across childhood and adulthood (Baxter, Hardin, Royer, Guinn, & Smith, 2008). Finally, clustering at the household level may have occurred given that each parent may be linked to more than one child per household. A sensitivity analysis was conducted whereby the cluster variable considered was the household. Our findings were similar, but we preferred to present the results with PSU as the cluster variable. We could not adjust for both.

Our findings have a number of important public health implications. First, the overall weak to moderate parent–child resemblance (r: 0.2–0.33) in food groups, nutrients and HEI_n scores suggests that interventions targeting at parents could only have a moderate effect in improving their children’s diet. In particular, such interventions would be most effective when targeted at mothers, minority groups, and as early as possible in childhood (i.e. at ages 2–10 years). Economic well–being does not seem to affect the resemblance, indicating that intervention should be applied to all economic segments of the population (e.g., not only the low-SES families).

In conclusion, our findings based on nationally representative data show that the overall parent–child resemblance in dietary intakes in the United States is relatively weak, and is not as strong as many previously believed. And the resemblance varies by nutrients and food groups, and by types of parent–child dyad and population groups (e.g., age, ethnicity, and SES). Factors other than parental eating behaviors seem to play an important role in affecting American young people's dietary intakes. Future studies should attempt to investigate how parental diet-related knowledge, perceptions and self-efficacy may impact children's diet and whether parental dietary intake is acting as a mediator. Ethnic differences in parent–child dietary intakes' associations should be further explored in terms of differential effects of environmental and non-household factors such as peer pressure and their impact on children's dietary behavior in those ethnic groups in which correlations were found to be weak.

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Appendix

Appendix A

New 2005 USDA Healthy Eating Index - components and standards for scoring.[a]

Component	Maximum points	Standard for maximum score	Standard for minimum score of zero
Total fruit (includes 100% juice)	5	≥0.8 cup equiv. per 1000 kcal	No fruit
Whole fruit (not juice)	5	≥0.4 cup equiv. per 1000 kcal	No whole fruit
Total vegetables	5	≥1.1 cup equiv. per 1000 kcal	No vegetables
Dark green and orange vegetables and legumes[b]	5	≥0.4 cup equiv. per 1000 kcal	No dark green or orange vegetables or legumes
Total grains	5	≥3.0 oz equiv. per 1000 kcal	No grains
Whole grains	5	≥1.5 oz. equiv. per 1000 kcal	No whole grains
Milk[c]	10	≥1.3 cup equiv. per 1000 kcal	No milk
Meat and beans	10	≥2.5 oz equiv. per 1000 kcal	No meat or beans
Oils[d]	10	≥12 g per 1000 kcal	No oil
Saturated fat	10	≤7% of energy[e]	≥15% energy
Sodium	10	≤0.7 g per 1000 kcal[e]	≥2.0 g per 1000 kcal
Calories from solid fat, alcohol, and added sugar (SoFAAS)	20	≤20% of energy	≥50% energy

[a]Intakes between the minimum and maximum levels are scored proportionately, except for saturated fat and sodium (see note e).

[b]Legumes counted as vegetables only after meat and beans standard is met.

[c]Includes all milk products, such as fluid milk, yogurt, and cheese.

[d]Includes nonhydrogenated vegetable oils and oils in fish, nuts, and seeds.

[e]Saturated fat and Na get a score of 8 for the intake levels that reflect the 2005 Dietary Guidelines, 10% of energy from saturated fat and 1.1 g Na/1000 kcal, respectively.

Appendix B. Measuring agreement using weighted kappa

Kappa provides a measured of agreement between raters and the degree to which they concur in their respective sorting of *N* items into *k* mutually exclusive categories (Agresti, 1996). In our case, we are studying agreement across quintiles of the Healthy Eating Index (HEI) between parents and their children. When categories are ordinal (e.g. quintiles), it is possible to use weights depending on the distance between the misclassified quintiles of parents and those of their children. To obtain observed, expected probability of agreement and weighted kappa estimates for quintiles, the following steps are needed:

(i) Construct a linear weight matrix for kappa estimation: W

1	0.75	0.25	0	0
0.75	1	0.75	0.25	0
0.25	0.75	1	0.75	0.25
0	0.25	0.75	1	0.75
0	0	0.25	0.75	1

$$\text{Linear weight} = 1 - \frac{|\text{dist}|}{\text{dist}_{\text{max}}}$$

Where $\text{dist}_{\text{max}} = k - 1 = 5 - 1 = 4$ for quintiles; dist can range between 0 and 4.

$$\text{Kappa}_{(\text{weighted})} = \left(\text{Agreement}_{(\text{observed prop.})} - \text{Agreement}_{(\text{expected prop.})}\right) / \left(1 - \text{Agreement}_{(\text{expected prop.})}\right)$$

(ii) Construct the agreement observed matrix: O

	Parents				
Children	$P11$	$P21$	$P31$	$P41$	$P51$
	$P12$	$P22$	$P32$	$P42$	$P52$
	$P13$	$P23$	$P33$	$P43$	$P53$
	$P14$	$P24$	$P34$	$P44$	$P54$
	$P15$	$P25$	$P35$	$P45$	$P55$

(iii) Construct the agreement "chance expected" matrix: E

$$P_1 = P_2 = P_3 = P_4 = P_5 = 0.20$$

$P_1 \times P_1 = 0.04$	0.04	0.04	0.04	0.04
0.04	$P_2 \times P_2 = 0.04$	0.04	0.04	0.04
0.04	0.04	$P_3 \times P_3 = 0.04$	0.04	0.04
0.04	0.04	0.04	$P_4 \times P_4 = 0.04$	0.04

0.04	0.04	0.04	0.04	$P_5 \times P_5 = 0.04$

(iv) Calculate P_{observed}, P_{expected} and weighted kappa:

$$\begin{aligned} P_{\text{observed}} &= O \times W = P_{11} \times 1 + P_{12} \times 0.75 + P_{13} \times 0.25 + \cdots \\ P_{\text{expected}} &= E \times W \\ &= 0.04 \times 1 + 0.04 \times 0.75 + 0.04 \times 0.25 + \cdots = 0.50 \end{aligned}$$

$$\text{Kappa}_{(\text{weighted})} = \left(P_{\text{observed}} - P_{\text{expected}}\right) / \left(1 - P_{\text{expected}}\right)$$

Note: observed and expected P were also weighted by the sample weight.

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Table 1

Demographic and health characteristics of study sample: mean or % ± SEM[a]; CSFII 1994–96.

	Both (*n* = 2291)	**Male (*n* = 1061)**	**Female (*n* = 1230)**
Parent and household characteristics			
Age (years)	37.5 ± 0.2	38.7 ± 0.3	36.4 ± 0.3*
Ethnicity (%)			
NH white	72.7 ± 2.6	75.0 ± 3.1	70.8 ± 2.4*
NH black	10.9 ± 1.6	8.6 ± 1.9	12.7 ± 1.6
Hispanic	12.6 ± 2.2	12.4 ± 2.5	12.8 ± 2.0
Other ethnicity	3.8 ± 0.5	3.9 ± 0.6	3.7 ± 0.5
Education (years)	13.5 ± 0.1	13.7 ± 0.1	13.4 ± 0.1
Employment status (% employed)	78.3 ± 1.0	92.6 ± 10.1	66.6 ± 1.7*
Poverty income ratio (PIR %)			
Poor (PIR:0–130)	16.3 ± 1.2	10.5 ± 0.8	21.0 ± 1.6*
Near poor: (PIR: 131–300)	46.7 ± 1.8	50.0 ± 2.1	44.9 ± 1.8
Not poor (PIR > 300)	36.9 ± 2.1	40.5 ± 2.3	34.1 ± 2.0
Smoking status (% current)	22.9 ± 1.0	24.9 ± 1.4	21.4 ± 1.1
BMI (kg/m^2)	26.2 ± 0.1	27.0 ± 0.2	25.5 ± 0.2*
Physical exercise (% sedentary)	31.5 ± 1.4	24.9 ± 1.6	36.9 ± 1.9*
Self-rated health (% poor)	1.2 ± 0.2	0.9 ± 0.3	1.5 ± 0.3
Chronic disease (% >2 reported)[b]	5.3 ± 0.7	6.7 ± 1.4	4.1 ± 0.5
Child characteristics	(*n* = 2692)	(*n* = 1370)	(*n* = 1322)
Age (years)	10.8 ± 0.1	10.8 ± 0.2	10.9 ± 0.1
BMI (kg/m^2)	20.2 ± 0.1	20.3 ± 0.1	20.0 ± 0.2
Physical exercise (% sedentary)	6.1 ± 0.8	3.8 ± 0.8	8.5 ± 1.2*

*$P < 0.05$ based on ANOVA (continuous characteristics) or χ^2 (categorical characteristics) tests comparing means or proportions across parent–child gender dyads.

[a]SEM: standard error of the mean.

[b]Conditions included diabetes, high blood pressure, heart disease, cancer, osteoporosis, high blood cholesterol and stroke.

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Table 2

Nutrient, selected food group intakes and overall diet quality of parents and children: mean ± SEM; CSFII 1994–96.

	Parents (*n* = 2291)	**Father (*n* = 1061)**	**Mother (*n* = 1230)**	**Children (*n* = 2692)**	**Son (*n* = 1370)**	**Daughter (*n* = 1322)**
HEI_n	49.5 ± 0.5	48.4 ± 0.5	50.3 ± 0.5	48.9 ± 0.4	48.4 ± 0.4	49.4 ± 0.5*
Energy (kcal/d)	2040.8 ± 25.7	2479.3 ± 37.0	1685.3 ± 23.9	1992.7 ± 27.7	2239.4 ± 38.3	1730.7 ± 26.1
Fat (g/d)	77.1 ± 1.2	94.9 ± 1.9	62.7 ± 1.1	72.1 ± 1.1	81.6 ± 1.7	62.0 ± 1.0
Fat (% kcal)	33.4 ± 0.2	34.0 ± 0.2	32.9 ± 0.3	32.2 ± 0.1	32.4 ± 0.4	32.0 ± 0.2*
Saturated fat (g/d)	25.9 ± 0.4	31.8 ± 0.6	21.1 ± 0.4	26.0 ± 0.4	29.5 ± 0.6	22.2 ± 0.4
Saturated fat (% kcal)	11.2 ± 0.1	11.4 ± 0.1	11.0 ± 0.1	11.7 ± 0.1	11.8 ± 0.1	11.5 ± 0.1 *
Cholesterol (mg/d)	266.4 ± 5.3	330.1 ± 7.5	214.8 ± 5.8	220.6 ± 4.4	248.8 ± 6.5	190.7 ± 5.2
Sodium (mg/d)	3425.8 ± 48.8	4223.9 ± 72.8	2778.8 ± 39.7	3154.9 ± 50.8	3564.0 ± 68.4	2720.5 ± 40.3
Calcium (mg/d)	754.4 ± 10.3	886.2 ± 15.2	647.6 ± 12.1	902.4 ± 16.7	1027.9 ± 24.6	769.2 ± 15.0
Fiber (g/d)	15.7 ± 0.3	18.7 ± 0.4	13.2 ± 0.2	13.2 ± 0.2	14.5 ± 0.3	11.7 ± 0.3
Sugar and candies (g/d)	21.4 ± 1.0	22.3 ± 1.5	20.6 ± 1.0*	36.9 ± 1.5	38.1 ± 2.3	35.6 ± 2.0*
Fruits & vegetables (g/d)	351.2 ± 8.3	386.3 ± 10.7	322.6 ± 8.5	302.4 ± 8.8	316.7 ± 11.9	287.2 ± 9.6*
Dairy products (g/d)	213.2 ± 6.0	236.8 ± 10.3	194.0 ± 6.3	447.1 ± 11.6	512.4 ± 17.9	377.7 ± 10.2
Total soft drinks (g/d)	404.2 ± 14.5	455.8 ± 17.8	362.4 ± 15.5	318.1 ± 12.1	367.4 ± 15.3	267.4 ± 11.0
Unsweetened soft drinks (g/d)	122.4 ± 10.1	108.9 ± 11.1	133.4 ± 12.1 *	27.4 ± 3.0	25.1 ± 3.8	29.8 ± 4.5*

* $P > 0.05$ based on *t*-test for the null hypothesis of equality of means between genders. All other gender comparisons were statistically significant ($P < 0.05$).

Table 3

Association between children and parents' diet: multivariate-adjusted standardized regression coefficients[b,c] stratified by gender dyads and selected characteristics; CSFII 1994–96.

	By gender dyads					By selected characteristics							
	Parent–child (n = 4244)	**Father–son (n = 982)**	**Father–daughter (n = 978)**	**Mother–son (n = 1156)**	**Mother–daughter (n = 1128)**	**NH white (n = 2914)**	**NH black (n = 492)**	**Hispanic (n = 658)**	**Other (n = 180)**	**Child 2–10 y (n = 2399)**	**Child > 10 y (n = 1845)**	**PIR 0–300 (n = 3028)**	**PIR > 300 (n = 1216)**
HEI_n	0.26	0.29	0.28	0.28	0.18*[a]	0.24	0.20	0.31	0.28*[a]	0.31	0.19*	0.28	0.21*
Energy (kcal/d)	0.22	0.29	0.14	0.23	0.26	0.21	0.25	0.22	0.35	0.11	0.33[a]	0.22	0.17
Total fat (g/d)	0.24	0.27	0.18	0.28	0.24	0.22	0.25	0.28	0.56	0.11	0.40[a]	0.23	0.20
Fat (%kcal)	0.01*	0.01*	0.02*	−0.04*	0.02*	0.01*	−0.06*	0.03*	0.03*	−0.05	0.06*[a]	−0.03*	0.10*
Saturated fat (g/d)	0.23	0.21	0.20	0.28	0.24	0.21	0.26	0.32	0.53	0.10	0.39[a]	0.21	0.17
Saturated fat (% kcal)	0.02*	0.01*	0.05*	−0.02*	0.03*	0.04*	−0.11*	0.04*	0.05*	−0.03*	0.08[a]	−0.00*	0.09*
Cholesterol (mg/d)	0.30	0.32	0.20	0.47	0.31	0.32	0.26	0.17	0.35	0.23	0.41[a]	0.28	0.23
Sodium (mg/d)	0.21	0.24	0.18	0.25	0.25	0.21	0.11*	0.18	0.43	0.12	0.30[a]	0.21	0.16
Calcium (mg/d)	0.20	0.14	0.14	0.30	0.30 [a]	0.19	0.18	0.32	0.20*	0.16	0.24[a]	0.19	0.16
Fiber (g/d)	0.26	0.31	0.18	0.33	0.28	0.25	0.22	0.33	0.21	0.21	0.32[a]	0.30	0.17
Sugar and candies (g/d)	0.21	0.13	0.22	0.23	0.27	0.20	0.26	0.25	0.42	0.20	0.22	0.22	0.13
Fruits & vegetables (g/d)	0.29	0.29	0.21	0.31	0.37	0.28	0.22	0.36	0.17*	0.32	0.25	0.28	0.25
Dairy products (g/d)	0.17	0.10	0.10	0.30	0.30[a]	0.16	0.13*	0.28	0.11*	0.14	0.20	0.16	0.20
Total soft drinks (g/d)	0.16	0.19	0.09*	0.20	0.19	0.14	0.18*	0.30	0.56[a]	0.09	0.20[a]	0.14	0.21
Unsweetened soft drinks (g/d)	0.17	0.09*	0.19	0.22	0.18	0.18	0.32*	−0.01*	0.80	0.13	0.23	0.09*	0.31[a]

* $P > 0.05$ for null hypothesis that $r = 0$.

[a] $P < 0.05$ for null hypothesis that interaction term $Intake_p$ × [gender dyad] or $Intake_p$ × [selected characteristic] = 0. Only multivariate-adjusted models were tested for interaction by gender dyads and selected characteristics.

[b] Regression coefficients are adjusted by: age of the parents and children, gender of parents and children, child's BMI and physical activity, parental ethnicity, educational level, employment status, poverty income ratio, smoking status, BMI, physical exercise, self-rated health and # of chronic conditions, geographical region and urbanization. When effect modification was studied by one of these variables, the variable was removed from the model as confounder to avoid multicollinearity. In all models, design complexity was accounted for.

[c] Regression coefficients are interpreted as Pearson correlations coefficients because each dietary variable (e.g. parent and child) was transformed into a z-score.

Table 4

Observed and expected percentage agreement,[a] kappa, and multivariate logistic regression[b] for agreement in high diet quality between parents and their children (HEI_n > 80th percentile), stratified by gender dyads and selected characteristics; CSFII 1994–96.

Characteristics	Overall agreement, weighted (sum of all 5 quintiles)			Overall agreement, unweighted (sum of all 5 quintiles)			Healthy diet (Q5) agreement	
	Observed agreement (%)	Expected agreement (%)	Weighted kappa	Observed agreement (%)	Expected agreement (%)	Unweighted kappa	OR	95% CI
Total population	61.1	50.0	0.22	29.1	20.0	0.11	2.71*	(1.91, 3.84)
Gender dyads								
Father–son	58.9	50.7	0.16	26.0	20.2	0.07	3.66*	(2.02, 6.64)
Father–daughter	61.0	50.2	0.22	28.1	20.4	0.10	3.78*	(2.20, 6.48)
Mother–son	61.8	50.5	0.23	31.2	20.2	0.14	2.71*	(1.68, 4.67)
Mother–daughter	62.3	49.6	0.25	30.2	20.0	0.13	1.90*	(1.22, 2.93)
Selected characteristics								
Non–Hispanic white	60.1	50.4	0.19	28.7	20.0	0.11	2.61*	(1.71, 3.97)
Non–Hispanic black	61.9	50.8	0.22	28.4	21.1	0.09	3.00*	(1.73, 5.20)
Hispanic	64.6	50.8	0.28	31.0	21.3	0.12	2.81*	(1.49, 5.28)
Others	60.9	57.8	0.07	33.1	30.3	0.04	1.95	(0.56, 6.80)
Child age								
Child's age: 2–10 y	61.7	49.9	0.24	29.4	20.0	0.12	4.05*[c]	(2.45, 6.68)
Child's age: > 10 y	60.2	49.9	0.21	28.6	19.9	0.11	1.55*	(1.01, 2.36)
Family income								
PIR: 0–300	60.9	49.8	0.21	28.7	20.0	0.11	3.09*	(2.07, 4.60)
PIR > 300	60.9	49.8	0.22	29.7	20.0	0.12	2.17*	(1.30, 3.67)

*$P < 0.05$ for null hypothesis that $\beta = \ln(OR) = 0$.

[a]The unweighted agreement estimates were calculated taking into account sample weight only and thus expected agreement was close to 4% × 5 = 20%. The weighted agreement estimates were calculated took into account of sample weight and weight matrix, and thus the expected agreement was not 4% × 5 = 20% but rather closer to 50% (see Appendix B). A kappa > 0.2 indicated a weak agreement and a kappa > 0.3, moderate agreement.

[b]Regression coefficients (interpreted as ln(OR)) are adjusted by: age of the parents and children, gender of parents and children, child's BMI and physical activity, parental ethnicity, educational level, employment status, poverty income ratio, smoking status, BMI, physical exercise, self-rated health and # of chronic conditions, geographical region and urbanization. When effect modification was studied by one of these variables, the variable was removed from the model as confounder to avoid multicollinearity. In all models, design complexity was accounted for.

[c]$P < 0.05$ for null hypothesis that interaction term $HEI_n p$ × [gender dyad] or $HEI_n p$ × [selected characteristic] = 0.