Parent-child nutrient intake interrelationships in school children ages 6 to 19: The Princeton School District Study[1-3]

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ABSTRACT Interrelationships between nutrient intakes (dietary cholesterol, total carbohydrate, saturated and polyunsaturated fat, and total calories) of parents and children were examined in 294 families (60 black, 234 white) which included at least one parent and one child in the Princeton School survey of parents and their children, ages 6 to 19. The nutrient data were collected by means of the standardized Lipid Research Clinics' collaborative 24-hr dietary recall; simple correlations and analysis of covariance were used to assess parent-child nutrient intake (per kg body weight) relationships. There were significant positive simple correlations between nutrient intake of parents and children for total carbohydrate ($r = 0.28$, $P < 0.0001$), saturated fat ($r = 0.15$, $P < 0.01$), polyunsaturated fat ($r = 0.19$, $P < 0.001$), and calories ($r = 0.24$, $P < 0.0001$); parents' intake of cholesterol did not correlate with that of their children ($r = 0.004$, $P > 0.1$). By analysis of covariance with adjustment for sex, race, age, and recall group, the parent-child association of cholesterol intake was significant ($P = 0.001$), and the remaining parent-child nutrient intake relationships were congruent with those observed by simple correlations. The proportion of variation of the children's nutrient intake accounted for by parental nutrient intake varied from a low of 23% for parent-child cholesterol intakes (all parents-all children) to a high of 97% for carbohydrate intake in black fathers over age 40 and their children. The multiple R^2s for black parents-black children for nutrient intakes were higher than those for white parents-white children for carbohydrate, saturated fat, and calories. Close parent child nutrient interrelationships not only suggest that a considerable portion of lipid-lipoprotein variability may be nutritionally-environmentally determined, but may contribute to clustering of coronary heart disease risk factors in families. *Am. J. Clin. Nutr.* 33: 2350–2355, 1980.

Multiple studies have demonstrated familial aggregation of risk factors associated with coronary heart disease (CHD), including blood pressure, lipids, and lipoproteins (1–5). In the recently reported study of Framingham parents and their children (5), it was concluded that a considerable proportion of parent-child lipid and lipoprotein correlations could be accounted for by genetic, as compared to transient environmental factors. In twin studies (6, 7), the total impact of genetic differences on low density lipoprotein cholesterol (C-LDL) was estimated by Christian et al. (6) to be about 30% of total population variability. Dichorionic identical twins, moreover, have lower twin-twin lipoprotein correlations than monochorionic twins (7). This points to the importance of genetic and/or intrauterine influences on C-LDL levels as

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an important source of population variation. Chase et al. (3) have provided strong evidence for familial aggregations of plasma cholesteral levels and have emphasized the importance of distinguishing the amount of genetic and environmental contributions to these familial aggregations.

Although parent-child (1–5) and twin studies (6, 7) point to an important familial-genetic role in the determination of lipids and lipoproteins, Brunner et al. (8) reported that levels of total cholesterol, triglyceride, and the percentage of total cholesterol as β-lipoproteins were equaly variable within and between families in an Israeli kibbutz settlement in which all residents ate the same foods prepared in a single communal kitchen as opposed to eating as family units, with separate family kitchens. The study of Brunner et al. (8) supported strongly a view that a considerable portion of lipid and lipoprotein variability is nutritionally and environmentally determined. The important contribution of nutritional practices relating to plasma lipids and lipoproteins and ultimately to CHD in population groups has recently been detailed by Stamler (9).

In our recent study of relationships beween nutrient intakes and plasma lipids and lipoproteins in 1669 school children ages 6 to 19 (10, 11), we reported that nutrient intake had a numerically small but highly significant association with plasma lipids and lipoproteins in children; as such, pediatric nutrient intake may have importance relative to the pediatric genesis of atherosclerosis (10–12). Our findings of significant inverse correlations in 6- to 19-year-old school children between the dietary P/S ratio, total carbohydrate, and sucrose to plasma C-LDL and high density lipoprotein cholesterol (C-HDL) emphasize the direct association of nutrient intake with the major atherogenic (C-LDL) and antiatherogenic (C-HDL) lipoproteins (10, 11). Frank et al. (13), have reported (in 10-year-old Louisiana children) that plasma total cholesterol was negatively correlated with dietary carbohydrate while plasma triglyceride was positively correlated with sucrose intake.

This study was designed to assess potential interrelationships between nutrient intake of parents and children.

Materials and methods

Study population

As previously described (10, 11, 14), the Princeton School survey of the Cincinnati Lipid Research Clinic characterized lipid and lipoprotein levels in a biracial population (75% white, 24% black) of school children, and a randomly selected subset of their parents. At an initial visit, fasting blood tests were restricted to cholesterol and triglyceride determinations (10, 11, 14, 15). At a second visit (visit 2) approximately 6 weeks later, a 15% random subset of the initial population independent of lipid results (random recall group), was restudied along with an additional subset, approximately 10%, dependent on the upper decile of lipid levels, (the hyperlipidemic recall group) (10, 11, 14, 15).

To obtain the largest number of parents and children we selected (from both recall groups) all families with at least one parent and one child (n = 294). There were the following numbers of children by 2-year age groups: ages 6 to 7, n = 44; ages 8 to 9, n = 38; ages 10 to 11, n = 40; ages 12 to 13, n = 59; ages 14 to 15, n = 48; ages 16 to 17, n = 53; ages 18 to 19, n = 12. For adults by 10 year age groups there were the following: ages 20 to 29, n = 21; ages 30 to 39, n = 146; ages 40 to 49, n = 99; ages 50 to 59, n = 24; ages 60 to 69, n = 4.

For children, there were 136 males and 158 females, for adults, 154 males and 140 females. There were 60 black families and 234 white families.

In families with more than one parent and/or more than one child, we arbitrarily chose the oldest parent and/or the oldest child (n = 96); hence, there were 198 families from which we had data for only one parent and one child. There were 60 black families and 234 white families.

Collection of nutrient data

At visit 2 the nutrient data were collected on all subjects by means of the standardized Lipid Research Clinics' collaborative 24-hr dietary recall (10, 11, 15–17). The collection of the nutritional data, certification of nutritionists to ensure uniformity in the determination of nutrient vectors, and central error checking and nutrient composition computaion have all been previously described (10, 11, 15–17). Data collection took place 6 days per week, spanning 12 months per year from October 1973 through October 1975. The recall was carried out on the day of the visit 2 sampling and covered the 24-hr period before the 12-hr fasting period. Dietary recalls were obtained on all days of the week except Sunday. A proportion of the children (26%) were seen on Mondays to reflect weekend food patterns in accordance with the collaborative protocol (10, 11, 15–17). Of the 294 families, dietary recalls were obtained in both parents and children on the same day in 99 families and on different days (within a week) for 195 families.

The 24-hr recall of the collaborative Lipid Research Clinics' program (10, 11, 15–17) was used instead of 7- or 9-day diet records (17–20) with full awareness of the potential sources of error and problems in evaluation of 24-hr diet recall (10, 11, 17–20). The very large sample size at visit 2 in the collaborative Lipid Research Clinics' program (15) and the related cost of nutriton data acquisition and analysis essentially mandated the 24-hr recall. Despite the methodological limitations of the 24-hr recall, in the Cincinnati Lipid Research Clinic's pediatric cohort, the 24-hr recall proved to be adequate for demonstrating highly significant partial correlation coefficients between nutrient intake and plasma lipids and lipoproteins (10, 11).

Statistical methods

The parent-child nutrient interrelationships were examined using nutrients per kg to best examine commonality of dietary composition between parents and children. The data were also assessed as nutrients per 1000 kcal, with similar but less consistent results than nutrients per kg, which were subsequently used. Simple correlations (both Pearson and Spearman) (21) between nutrient intakes of a parent and his/her child (per kg body weight) were obtained for dietary cholesterol, total carbohydrates, saturated fat, polyunsaturated fat, and calories (Table 1). Analysis of covariance (22) was used to further assess parent-child nutrient intake per kg interrelationships. The nutrient intakes of the children served as the response variable. The sex, race, recall group, and age group of the parents were the main effects (classification variables) in the analysis of covariance. The age groups were determined from cluster analysis to allow their use as a classification rather than a continuous variable. Covariables included the child's age, sex, recall group, and the parent's nutrient intake. The interactions of both the main effects and covariables commonly considered in an analysis of covariance were also included in the model.

Each nutrient was analyzed separately. For each nutrient, parent-child relationships were displayed in varying formats only if the effect of the parent's nutrient intake on the child's nutrient intake differed for one or more of the main effects (sex, race, recall group, and age group) (Table 2). For example, the association of the parent's cholesterol intake with that of their children's intakes was similar for all parents' sex, age, race, and recall groups (Table 2). The association of the mother's polyunsaturated fat intake with her child's intake differed from the association of the father's polyunsaturated fat intake with his child's, hence, results were reported by parents' sex (Table 2). The parent-child relationship of carbohydrate intake differed for each race and age group, and also by sex for black parents over age 40, with display, therefore, of these differing main effect groups separately (Table 2). The caloric intake relationship was similar for all fathers but differed among race and age groups in mothers (Table 2).

Results

Parent-child nutrient interrelationships, simple correlation coefficients

Table 1 displays simple Pearson and Spearman correlation coefficients for the following parent-child dyads: one parent-one child; oldest parent-oldest child, all parents-all children. All of the correlation coefficients were positive. There were significant correlations between all parents and all children for intake of total carbohydrates, saturated and polyunsaturated fat, and calories whether Pearson or Spearman correlations were used (Table 1). Significant correlations were also obtained in the one parent-one child group for total carbohydrate, saturated and polyunsaturated fat, and calories. A similar pattern of relationships was observed for the oldest parent-oldest child group with the exception of insignificant parent-child relationships for polyunsaturated fat intake.

Parent-child nutrient interrelationships, analysis of covariance

Table 2 summarizes the effect of parents' nutrient intake on children's nutrient intake using analysis of covariance. The *P* value for the parent-child relationship, and the multiple R^2 statistic (the proportion of variation of the children's nutrient intake accounted for by the explanatory variables) are given for each group, and for each nutrient, as described in the "Methods" section. All of the parent-child nutrient interrelationships were positive.

TABLE 1
Correlations between nutrient intake/kg of parents and children

	Cholesterol	Total carbohydrate	Saturated fat	Polyunsaturated fat	Calories
One parent-one child (n = 198)					
Pearson	−0.002	0.208[a]	0.148[b]	0.184[a]	0.210[a]
Spearman	−0.004	0.296[c]	0.154[b]	0.218[a]	0.256[d]
Oldest parent and/or oldest child (n = 96)					
Pearson	0.165	0.255[a]	0.319[d]	0.126	0.309[a]
Spearman	0.017	0.230[b]	0.140	0.116	0.210[b]
All parents, all children (n = 294)					
Pearson	0.056	0.223[c]	0.224[c]	0.164[a]	0.250[c]
Spearman	0.004	0.276[c]	0.154[a]	0.191[d]	0.241[c]

[a] $P < 0.01$. [b] $P < 0.05$. [c] $P < 0.0001$. [d] $P < 0.001$.

TABLE 2
Parent-child nutrient interrelationships, analysis of covariance

Parent-child group	n	Nutrient	Parent-child association (P)	Multiple R^2
All parents—all children	294	Cholesterol	0.001	0.23
White parents >44 yr old—children	61	Carbohydrate	0.02	0.38
White parents 34–44 yr old—children	133	Carbohydrate	NS[a]	0.38
White parents <34 yr old—children	40	Carbohydrate	NS	0.35
Black fathers >40 yr old—children	11	Carbohydrate	0.0004	0.97
Black mothers >40 yr old—children	12	Carbohydrate	0.006	0.92
Black parents <40 yr old—children	37	Carbohydrate	NS	0.56
All mothers—all children	140	Polyunsaturated fat	0.0007	0.33
All fathers—all children	154	Polyunsaturated fat	NS	0.24
White mothers >44 yr old—children	19	Saturated fat	NS	0.59
White mothers 34–44 yr old—children	60	Saturated fat	NS	0.32
White mothers <34 yr old—children	23	Saturated fat	0.03	0.47
All white fathers, RR[b] group—children	67	Saturated fat	NS	0.54
All white fathers, HR[c] group—children	65	Saturated fat	0.01	0.40
Black mothers >40 yr old—children	12	Saturated fat	0.04	0.65
Black mothers <40 yr old—children	26	Saturated fat	NS	0.61
All black fathers, RR group—children	9	Saturated fat	NS	0.50
All black fathers, HR group—children	13	Saturated fat	NS	0.66
White mothers >44 yr old—children	19	Calories	NS	0.35
White mothers 34–44 yr old—children	60	Calories	NS	0.47
White mothers <34 yr old—children	23	Calories	0.002	0.67
Black mothers >40 yr old—children	12	Calories	0.01	0.81
Black mothers <40 yr old—children	26	Calories	NS	0.61
All fathers—all children	154	Calories	NS	0.51

[a] $P > 0.1$. [b] Random recall. [c] Hyperlipidemic recall.

The parent-child association for cholesterol intake was significant, $P = 0.001$ (Table 2). Polyunsaturated fat intake of children was closely associated with that of their mothers ($P = 0.0007$), but not with that of the fathers' intake (Table 2). Carbohydrate intake in parents and their children was significantly associated in children of white parents older than 44, children of black fathers older than 40, and children of black mothers older than 40 (Table 2). The caloric and saturated fat intake of white mothers younger than 34 was significantly related to that of their children (Table 2). The caloric and saturated fat intake of black mothers older than age 40 was related to that of their children (Table 2).

The saturated fat intake of hyperlipidemic recall fathers had a significant association with the saturated fat intake of their children (Table 2).

For polyunsaturated fat, saturated fat, and caloric intakes, mother-child nutrient interrelationships were stronger than those of fathers and children.

If the parent-child nutrient intake relationships differed between recall groups, our covariance model would have detected any significant differences. The sole instance where the recall group was a significant factor was for fathers in the hyperlipidemic recall group, whose saturated fat intake was related to that of their children, as noted above (Table 2). Otherwise, recall group location was not relevant to parent-child nutrient interrelationships.

The proportion of variation of the children's nutrient intake accounted for by parental nutrient intake varied from a low of 23% for parent-child cholesterol intake relationships to a high of 97% for carbohydrate intake in black fathers over age 40 and their children (Table 2). The multiple R^2 statistics for black parents-black children nutrient intakes were higher than those for white parents-white children for carbohydrate, saturated fat, and calories (Table 2).

Discussion

It is a biological truism that both genetics and environment play a major role in parent-child lipid and lipoprotein association (1–8). Since children's nutrient intakes have small but highlly significant associations with their plasma lipids and lipoproteins (10, 11), an examination of parent-child nutrient interrelationships can provide some information pertinent to shared parent-child environmental influences that may, in aggregate, contribute, albeit indirectly, to parent-child clustering of coronary heart disease risk factors (1–5).

In studies of Danish children during the first 4 years of life, Anderson et al. (23) speculated that shared nutritional patterns common to parents and children contributed to significant parent-child plasma cholesterol correlations. It is known that family structure, and parent-child social and emotional interactions contribute to shared eating patterns (24–26). Garn and Clark (27), in commenting on the Ten-State Nutrition Survey (27), suggested that significant husband-wife correlations in relative ponderosity either resulted from communal living or reflected the tendency of like to mate with like. Garn and Clark (27) speculated that fatness in family lines appears primarily because of family line similarities in caloric intake, caloric expenditure, and attitudes toward food and eating. Within this frame of reference (23–27), the significant correlations observed in this study between parent and child nutrient intakes were not unexpected. When considering relationships between all parents and all children by simple correlations, the parent-child nutrient intakes were significantly and positively related for total calories, saturated fat, polyunsaturated fat, and carbohydrate intakes, but not for cholesterol. However, using covariance analysis with adjustment for potential confounding explanatory variables, the cholesterol intakes of all parents and all children were significantly associated. Moreover, our findings of parent-child nutrient interrelationships by covariance analysis were congruent with those observed using simple correlations. We do not know why, overall, parent-child nutrient interrelationships and the portion of children's nutrient intake variation explained by independent variables was greater for black than for white parent-child dyads. We are not aware of comparable black/white data for comparison.

The close associations of nutrient intake of parents and children, although teleologically accepted, have not been recently documented in cohort studies. The substantial parent-child nutrient intake relationships are very much consistent with the conclusions of Brunner et al. (8), that a considerable portion of lipid and lipoprotein variability may be nutritionally and environmentally determined.

In the covariance model used in this study, from 23% to over 90% of the variance of children's nutrient intakes could be explained by those of their parents with other explanatory variables such as age, race, and recall group. The observed parent-child nutrient interrelationships may be underestimates of the actual relationships, since 66% of the dietary data for parents and children was collected on different days (within the same week), which inevitably would increase overall variance (17, 18). The findings in this study of close parent-child nutrient interrelationships provide a firm data base to the postulations that broad scale approaches to alter dietary habits of population groups must focus upon the family unit (12). Although much a conclusion is surely not revelatory (24–27), it seems possible that public health education directed at schoolchildren's dietary choices might be "exportable" for family units (12).

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