RESEARCH REPORT

Fruit, vegetables, and antioxidants in childhood and risk of adult cancer: the Boyd Orr cohort

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Study objective: To examine associations between food and nutrient intake, measured in childhood, and adult cancer in a cohort with over 60 years follow up.
Design and setting: The study is based on the Boyd Orr cohort. Intake of fruit and vegetables, energy, vitamins C and E, carotene, and retinol was assessed from seven day household food inventories carried out during a study of family diet and health in 16 rural and urban areas of England and Scotland in 1937–39.
Participants: 4999 men and women, from largely working class backgrounds, who had been children in the households participating in the pre-war survey. Analyses are based on 3878 traced subjects with full data on diet and social circumstances.
Main results: Over the follow up period there were 483 incident malignant neoplasms. Increased childhood fruit intake was associated with reduced risk of incident cancer. In fully adjusted logistic regression models, odds ratios (95% confidence intervals) with increasing quartiles of fruit consumption were 1.0 (reference), 0.66 (0.48 to 0.90), 0.70 (0.51 to 0.97), 0.62 (0.43 to 0.90); p value for linear trend=0.02. The association was weaker for cancer mortality. There was no clear pattern of association between the other dietary factors and total cancer risk.
Conclusions: Childhood fruit consumption may have a long term protective effect on cancer risk in adults. Further prospective studies, with individual measures of diet are required to further elucidate these relations.

Until recently, investigation into the causes of chronic disease, including cancer, has focused on adult behavioural or "lifestyle" factors. There is growing evidence, however, that exposures at different stages of the life course influence disease risk.[1] Diet is considered to be of particular importance in the aetiology of many adult cancers[2] and it is probable that diet acts cumulatively, throughout life, on a person's risk of neoplasia.

There are few opportunities, however, to directly examine the influence of childhood diet on cancer risk. Studies using anthropometric measures of childhood nutrition suggest that early diet may be important[3] but those using adult recall of childhood dietary intake have not been consistent.[4–11] The Boyd Orr cohort presents a unique opportunity to assess the long term influence of childhood diet on adult health.[12 13] Study members have a measure of family diet collected during their childhood, as well as a wealth of anthropometric, social, and economic data, and are at an age when cancer has become a significant burden in terms of mortality and morbidity. This paper describes the association of fruit, vegetable, vitamin C, E, and carotene intake with the incidence and mortality from cancer in adulthood. The association between retinol or preformed vitamin A intake (which does not have antioxidant properties) and these outcomes was also assessed. This was in order that any association between cancer risk and carotene could be interpreted in the light of its role as a dietary antioxidant and as a pre-cursor to vitamin A.

METHODS

The Boyd Orr cohort

The establishment of the cohort has been described in detail elsewhere.[12] In brief, the data forming the basis of these analyses were obtained from the original records of the Carnegie survey of diet and health in pre-war Britain.[14] The survey was carried out in 1937–9 among 1352 mainly working class families living in 16 rural and urban areas of England and Scotland. Detailed measurements were made of household diet (see below) and the health, growth, and living conditions of the children in the households. The name, age, and address of the children (mean age 8 years) of the families surveyed were obtained from the original records and used to trace them through the National Health Service (NHS) Central Register. Of the 4999 children identified 86.6% were successfully traced (n=4334). The representativeness of those traced has previously been described. Traced survey participants were almost one year younger than their non-traced counterparts ($p<0.0001$) but did not differ in terms of childhood energy intake, food expenditure, or social class.[15] The study team are notified of the death, cancer registrations, area of current residence, and emigration of those cohort members who had been successfully traced through the NHS central register. Cause of death is ascertained from death certificates and classified according to International Classification of Diseases, 9th edition (ICD-9). This analysis is based on traced cohort members who were resident in Britain on 1 January 1948 and deaths and cancer registrations occurring up to 31 July 2000. It is limited to the 3878 subjects for whom full data are available and is an update of the analyses previously presented in a published abstract.[16] The 456 excluded subjects were 262 emigrants, 10 people with no family diet data, 21 with missing Townsend score, and 43 with missing family food expenditure (see below), 112 subjects who died before 1948, and 10 with missing cause of death.

Dietary data

Dietary data in the original Carnegie survey were obtained using a seven day household inventory method. A weighed inventory of all foods on hand in the household was recorded in a diary at the beginning of the survey period. A weighed record of all subsequent food brought into the home was made, and finally a second inventory was carried out at the

Table 1 Distribution of cancer sites

Cancer (ICD-9 codes)	Women n=1959		Men n=1919	
	Incident	Deaths	Incident	Deaths
Cancers not related to smoking				
Salivary gland (142)	2	0	1	1
Stomach (151)	5	5	12	10
Small intestine (152)	2	1	0	0
Colorectal (153–154)	23	14	28	12
Liver (155)	4	3	1	1
Gall bladder (156)	2	2	1	0
Peritoneum/ other digestive tract (158)	1	0	0	0
Thymus/ heart (164)	0	0	2	2
Bone (170)	1	0	0	0
Breast (174)	82	36	–	–
Uterus and cervix (179–182)	14	2	–	–
Ovary (183)	15	12	–	–
Vulva (184)	1	1	–	–
Prostate (185)	–	–	21	15
Penis (187)	–	–	1	0
Eye (190)	0	0	1	0
Brain and central nervous system (191–192)	4	2	4	4
Thyroid (193)	2	0	2	1
Disseminated/ Unspecified (199)	21	13	20	17
Lymphatic and haematopoietic tissue (200–208)	16	9	14	8
Malignant melanomas (172)	10	1	4	0
Total cancers not related to smoking	*191**	*101*	*110**	*71*
Smoking related cancers				
Lip, oral cavity, larynx (140–149)	5	5	10	6
Oesophagus (150)	6	5	11	10
Pancreas (157)	7	7	6	5
Respiratory tract (160–163)	35	33	81	73
Urinary tract (188–89)	11	7	22	10
Total smoking related cancers	*60**	*57*	*122**	*104*
Total (all cancers)	*251**	*158*	*232**	*175*

*Total incident cancers represent the sum of all subjects with cancer in any site or sites. These figures are less than the sum of all individual incident cancers because 26 subjects had multiple site cancers.

end of the survey period. Data from the diaries were then transcribed onto separate summary sheets for each household. Re-analysis of the food records was necessary to include nutrients not measured in the original study and also to make use of advances in analytical techniques where food composition is unlikely to differ between the 1930s and today. Re-coding of the foods for this study was carried out using the DIDO (diet in data out) program[17] developed at the Medical Research Council Human Nutrition Research in Cambridge, UK. Total fruit and vegetable (excluding potato) consumption, and intake of vitamins C, E, carotene, and retinol were re-analysed, using programs based on *McCance and Widdowson's the composition of foods*[18] and supplements. The database was adapted where composition of 1930s foods are very different than they are today (such as meat and meat products), or where there was no modern day equivalent, using pre-war food tables.[19–24] Per capita food and nutrient intake was calculated, as in the original study, by dividing daily total intake by the total number of household members regardless of age, sex, or occupation, but taking into account meals missed by family members and meals consumed by visitors. In a sensitivity analysis, we assessed the effects on the strength of observed associations of using estimates of household per capita daily food and nutrient intake weighted according to age and sex of household members. The weights were based on "man values" where intake in a particular age/sex group is expressed in relation to intake of an adult male (which is taken as unity). The "man values" used were those relating to energy intake proposed by the 1933 BMA committee of nutrition.[25] These values suggest, for example, that a child aged 8–10 years consumes 70% of the energy of an adult man and an adult woman 0.83%. So, the denominator in our analysis for per capita intake for a three person household with an adult man, adult woman, and 9 year old child, would be 2.53.

Statistical analysis

The end points in this analysis are incidence and mortality from all cancers, cancers related to smoking, cancers not related to smoking, and mortality from all causes. Fatal cancers were a subset of incident cancers. They were examined separately because mortality from cancer is socially patterned and therefore findings could potentially differ in this subgroup. In addition we subdivided cancers into those related to smoking and others because smoking may confound the childhood diet and adult cancer association and information on smoking patterns is not available for all subjects. All malignant neoplasms were included in the analyses (ICD-9 codes 140–208); non-melanoma skin cancers and all other benign cancers were excluded. As in previous analysis of the cohort, cancers of the lip (ICD-9 140); tongue (141); mouth and pharynx (143–9); oesophagus (150); pancreas (157); respiratory tract (160–163); and urinary tract (188–189) were deemed cancers related to smoking. Cancers not related to smoking were all other malignant cancers (140–208) except those listed. Analyses were carried out in Stata (release 6.0).[26] Logistic regression analysis was used to compute odds ratios for associations between these outcomes and the dietary factors of interest. This approach was taken instead of Cox's proportional hazards ratios because we wanted to use the available incident cancer data, and date of cancer registration can often be unreliable. The per capita dietary variables were entered into the models as quartiles of intake. A test for linear trend was obtained by entering the quartiles as continuous terms. All p values were two sided. Deviation from linear trend was examined using likelihood ratio tests.

The association between diet and cancer was examined in men and women together controlling for sex, age, and energy intake. Subsequent fully adjusted models took account of the

Table 2 Mean dietary intake by quartiles of the distribution (n=3878)

Dietary factor	Mean (SD) intake per head per day by quartile of the distribution			
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Total fruit (g)	0.6 (1.3)	12.7 (4.7)	31.3 (6.5)	88.4 (51.6)
Total vegetables (g)	23.1 (10.4)	47.3 (5.7)	68.5 (7.0)	115.2 (35.4)
Vitamin C (mg)	14.3 (3.6)	23.6 (2.4)	33.4 (3.2)	57.2 (19.1)
Vitamin E (mg)	1.9 (0.44)	3.0 (0.3)	4.1 (0.4)	6.1 (1.5)
Carotene (μg)	135.5 (64.1)	403.4 (83.7)	767.0 (140.4)	1629.4 (681.2)
Retinol (μg)	174.0 (50.5)	311.8 (37.5)	499.9 (79.1)	1148.3 (765.4)
Energy (MJ)	6.6 (0.9)	8.4 (0.4)	9.8 (0.4)	12.3 (1.6)

following additional potential confounding factors: per capita weekly food expenditure, season of survey, and social class of head of household during childhood, and Townsend deprivation score of the subjects' health authority of residence at death or in 1997 as a measure of adult socioeconomic position.[27] Per capita food expenditure and Townsend score were entered as continuous terms. Social class was entered as a categorical variable: I and II; III (the distinction between manual and non-manual was not introduced until 1951); IV; V; unemployed; and unclassifiable. District of residence at the time of the original survey was entered as a categorical variable with the 16 categories corresponding to the 16 survey centres. Adjustment for the hierarchical nature of the data (that is, the dietary data were for households rather than individuals and the risk of cancer may also cluster within families) was carried out for all models by means of robust standard errors (using the xtlogistic commands in Stata).

RESULTS

The numbers of incident and fatal cancers (a subset of total incidence) are shown in table 1. The commonest cancer sites were respiratory tract cancers in men and breast cancer in women. In table 2 mean dietary intake within each quartile of the distribution are presented. Table 3 shows Spearman's rank correlation coefficients between the dietary factors. Fruit and vegetables were moderately correlated with vitamin C (r=0.52 and r=0.55 respectively). The strongest correlations were between vegetable intake and carotene (r=0.65). Associations between fruit and vegetables and other food groups (cereals, meat, fish, milk and milk products, fats and oils) were also examined: correlations ranged from 0.00 to 0.39.

Total cancer incidence and mortality and all cause mortality

Logistic regression analyses of dietary intake and cancer incidence and mortality, and mortality from all causes are shown in table 4. Increasing levels of fruit consumption were associated with reduced risk of cancer in the models controlling for age, energy intake, and sex (p=0.02). This association was not materially changed after controlling for measures of childhood and adult socioeconomic circumstances. There was no evidence of interaction between fruit intake and either sex or age in their relation to cancer risk. When the analyses were repeated with fruit consumption weighted by the age structure of each household the findings were very similar. In fully adjusted models, odds ratios (95% confidence intervals) for incident cancers with increasing consumption were 1.0 (reference), 0.71 (0.52 to 0.97), 0.71 (0.52 to 0.98), 0.57 (0.39 to 0.82), p value for linear trend=0.004. Increased intake of fruit was also associated with lower mortality from all causes, but effects were weaker than those for cancer. There was no clear pattern of risk between vegetable consumption, vitamins C, E, carotene and retinol intake and cancer incidence, mortality, or all cause mortality.

Cancers related to smoking and cancers not related to smoking

Table 5 shows associations between dietary factors and cancers subdivided into those related to smoking and those not related to smoking. The association of fruit intake with both categories of cancer risk was similar in magnitude and direction to total cancer risk (table 4). There was evidence of an increased risk of cancers related to smoking with higher vitamin E intake.

The single most common cancer site in this cohort was breast cancer among women with 82 incident cases and 36 deaths. There were no clear associations between fruit, vegetables, and vitamin C and breast cancer. Higher vitamin E intake was associated with decreased breast cancer mortality, while higher intake of retinol was associated with increased risk (see table 6).

Energy intake and cancer risk

Previous analyses of the Boyd Orr cohort show an increase in risk associated with increasing energy intake.[13] The relation is maintained in the present analyses and was strongest for total cancer mortality. Odds ratios (95% CI) for the association between energy intake and cancer mortality, controlling for

Table 3 Spearman's rank correlation coefficients between daily intake of fruit, vegetables, energy, and nutrients

	Spearman's rank correlation coefficients						
	Fruit (g)	Vegetables (g)	Energy (MJ)	Vitamin C (mg)	Vitamin E (mg)	Carotene (μg)	Retinol (μg)
Fruit (g)	1.0						
Vegetables (g)	0.30	1.0					
Energy (MJ)	0.37	0.25	1.0				
Vitamin C (mg)	0.52	0.55	0.45	1.0			
Vitamin E (mg)	0.25	0.38	0.44	0.40	1.0		
Carotene (μg)	0.20	0.65	0.14	0.46	0.16	1.0	
Retinol (μg)	0.32	0.29	0.38	0.35	0.32	0.23	1.0

Table 4 Odds ratios* for cancer incidence and mortality, and all-cause mortality risk in relation to diet (n=3878)

Quartiles of dietary intake	Total cancer incidence			Total cancer mortality			All cause mortality		
	Number of cases	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)	Number of cases	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)	Number of deaths	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)
Fruit									
1 (low)	141	1.0	1.0	89	1.0	1.0	266	1.0	1.0
2	110	0.71 (0.52 to 0.97)	0.66 (0.48 to 0.90)	83	0.86 (0.61 to 1.21)	0.84 (0.59 to 1.20)	255	0.90 (0.70 to 1.15)	0.91 (0.71 to 1.16)
3	120	0.75 (0.56 to 1.02)	0.70 (0.51 to 0.97)	86	0.86 (0.61 to 1.21)	0.85 (0.59 to 1.22)	247	0.83 (0.65 to 1.07)	0.87 (0.67 to 1.12)
4 (high)	112	0.66 (0.48 to 0.91)	0.62 (0.43 to 0.90)	75	0.72 (0.50 to 1.04)	0.73 (0.47 to 1.11)	232	0.75 (0.58 to 0.98)	0.83 (0.62 to 1.11)
p value for linear trend		0.02	0.02		0.09	0.17		0.03	0.18
Vegetables									
1 (low)	111	1.0	1.0	76	1.0	1.0	248	1.0	1.0
2	127	1.21 (0.89 to 1.66)	1.17 (0.84 to 1.64)	93	1.29 (0.92 to 1.83)	1.18 (0.81 to 1.71)	271	1.17 (0.92 to 1.50)	1.06 (0.82 to 1.36)
3	109	1.01 (0.73 to 1.38)	0.95 (0.66 to 1.34)	76	1.04 (0.73 to 1.49)	0.90 (0.61 to 1.34)	237	1.01 (0.79 to 1.29)	0.86 (0.66 to 1.12)
4 (high)	136	1.33 (0.97 to 1.82)	1.34 (0.93 to 1.93)	88	1.22 (0.85 to 1.74)	1.14 (0.75 to 1.72)	244	1.04 (0.81 to 1.34)	0.92 (0.69 to 1.22)
p value for linear trend		0.18	0.27		0.52	0.92		0.97	0.28
Vitamin C									
1 (low)	114	1.0	1.0	84	1.0	1.0	259	1.0	1.0
2	127	1.14 (0.84 to 1.57)	1.16 (0.84 to 1.61)	83	0.94 (0.67 to 1.34)	0.98 (0.68 to 1.41)	251	0.94 (0.73 to 1.21)	0.99 (0.77 to 1.27)
3	120	0.97 (0.70 to 1.36)	1.03 (0.72 to 1.48)	83	0.88 (0.61 to 1.26)	0.95 (0.64 to 1.42)	251	0.89 (0.69 to 1.15)	0.95 (0.72 to 1.25)
4 (high)	122	1.04 (0.74 to 1.45)	1.11 (0.74 to 1.65)	83	0.91 (0.63 to 1.32)	0.98 (0.63 to 1.54)	239	0.87 (0.67 to 1.14)	0.98 (0.72 to 1.34)
p value for linear trend		0.92	0.80		0.56	0.92		0.29	0.85
Vitamin E									
1 (low)	115	1.0	1.0	80	1.0	1.0	246	1.0	1.0
2	108	0.94 (0.69 to 1.29)	0.91 (0.65 to 1.29)	75	0.94 (0.66 to 1.33)	0.90 (0.61 to 1.33)	253	1.09 (0.86 to 1.40)	1.13 (0.87 to 1.47)
3	114	0.97 (0.70 to 1.33)	0.88 (0.61 to 1.28)	75	0.89 (0.62 to 1.28)	0.79 (0.52 to 1.20)	231	0.92 (0.71 to 1.19)	0.89 (0.67 to 1.19)
4 (high)	146	1.29 (0.93 to 1.78)	1.12 (0.75 to 1.66)	103	1.28 (0.89 to 1.83)	1.09 (0.70 to 1.70)	270	1.16 (0.90 to 1.51)	1.11 (0.81 to 1.52)
p value for linear trend		0.13	0.52		0.23	0.71		0.51	0.89
Carotene									
1 (low)	123	1.0	1.0	90	1.0	1.0	240	1.0	1.0
2	116	0.86 (0.63 to 1.17)	0.88 (0.64 to 1.21)	76	0.75 (0.53 to 1.07)	0.74 (0.52 to 1.06)	257	1.00 (0.78 to 1.28)	0.99 (0.78 to 1.27)
3	129	1.03 (0.76 to 1.40)	1.05 (0.76 to 1.45)	83	0.88 (0.62 to 1.23)	0.81 (0.57 to 1.16)	256	1.04 (0.82 to 1.34)	0.95 (0.74 to 1.23)
4 (high)	115	0.89 (0.76 to 1.40)	0.92 (0.65 to 1.32)	84	0.88 (0.63 to 1.24)	0.80 (0.54 to 1.18)	247	1.00 (0.78 to 1.27)	0.90 (0.68 to 1.18)
p value for linear trend		0.74	0.96		0.67	0.36		0.94	0.41
Retinol									
1 (low)	126	1.0	1.0	91	1.0	1.0	262	1.0	1.0
2	110	0.86 (0.63 to 1.17)	0.83 (0.60 to 1.14)	77	0.84 (0.59 to 1.18)	0.80 (0.56 to 1.14)	233	0.89 (0.70 to 1.14)	0.89 (0.69 to 1.13)
3	118	0.88 (0.64 to 1.20)	0.86 (0.62 to 1.19)	78	0.80 (0.56 to 1.14)	0.78 (0.54 to 1.13)	264	1.02 (0.80 to 1.31)	1.03 (0.80 to 1.32)
4 (high)	129	0.94 (0.69 to 1.29)	0.93 (0.65 to 1.32)	87	0.86 (0.61 to 1.23)	0.88 (0.59 to 1.31)	241	0.86 (0.67 to 1.11)	0.89 (0.67 to 1.17)
p value for linear trend		0.76	0.73		0.41	0.52		0.45	0.66

*All effect estimates and confidence intervals are adjusted for intra-family clustering. †Fully adjusted models include age, sex, energy intake, food expenditure, Townsend score, season, and district.

Table 5 Odds ratios* for incidence and mortality from cancers related to smoking and cancers not related to smoking (n=3878)

	Cancers related to smoking				Cancers not related to smoking			
	Incidence		Mortality		Incidence		Mortality	
Quartiles of dietary intake	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)	Age, energy, sex, adjusted OR (95% CI)	Fully adjusted† OR (95% CI)
Fruit								
1 (low)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2	0.95 (0.62 to 1.46)	0.88 (0.56 to 1.37)	1.03 (0.65 to 1.65)	0.96 (0.59 to 1.55)	0.61 (0.41 to 0.89)	0.57 (0.38 to 0.84)	0.73 (0.46 to 1.16)	0.77 (0.47 to 1.24)
3	0.84 (0.54 to 1.31)	0.81 (0.51 to 1.29)	0.96 (0.59 to 1.54)	0.94 (0.57 to 1.55)	0.73 (0.50 to 1.05)	0.67 (0.46 to 0.99)	0.78 (0.49 to 1.24)	0.79 (0.48 to 1.28)
4 (high)	0.60 (0.37 to 0.98)	0.63 (0.36 to 1.11)	0.63 (0.37 to 1.08)	0.69 (0.37 to 1.27)	0.73 (0.50 to 1.07)	0.66 (0.42 to 1.03)	0.83 (0.52 to 1.32)	0.81 (0.47 to 1.42)
p value for linear trend	0.04	0.12	0.10	0.29	0.18	0.08	0.49	0.47
Vegetable								
1 (low)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2	1.42 (0.89 to 2.26)	1.25 (0.76 to 2.06)	1.41 (0.88 to 2.25)	1.25 (0.74 to 2.13)	1.06 (0.73 to 1.55)	1.06 (0.71 to 1.59)	1.14 (0.73 to 1.78)	1.09 (0.68 to 1.77)
3	1.36 (0.85 to 2.18)	1.23 (0.73 to 2.07)	1.33 (0.83 to 2.15)	1.14 (0.65 to 1.98)	0.83 (0.56 to 1.22)	0.79 (0.51 to 1.21)	0.81 (0.50 to 1.31)	0.72 (0.42 to 1.21)
4 (high)	1.40 (0.87 to 2.25)	1.29 (0.75 to 2.23)	1.33 (0.82 to 2.15)	1.17 (0.65 to 2.11)	1.24 (0.85 to 1.80)	1.28 (0.83 to 1.99)	1.08 (0.68 to 1.71)	1.08 (0.63 to 1.85)
p value for linear trend	0.22	0.43	0.31	0.75	0.49	0.50	0.89	0.81
Vitamin C								
1 (low)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2	1.36 (0.84 to 2.18)	1.39 (0.85 to 2.27)	1.31 (0.79 to 2.19)	1.24 (0.73 to 2.11)	1.02 (0.69 to 1.48)	1.03 (0.69 to 1.52)	0.72 (0.46 to 1.13)	0.81 (0.51 to 1.30)
3	1.44 (0.88 to 2.34)	1.49 (0.88 to 2.54)	1.49 (0.88 to 2.50)	1.44 (0.82 to 2.54)	0.76 (0.50 to 1.15)	0.81 (0.52 to 1.25)	0.55 (0.34 to 0.89)	0.65 (0.38 to 1.11)
4 (high)	1.17 (0.70 to 1.96)	1.32 (0.72 to 2.41)	1.24 (0.71 to 2.14)	1.25 (0.66 to 2.38)	0.96 (0.64 to 1.44)	0.97 (0.60 to 1.58)	0.71 (0.44 to 1.14)	0.82 (0.45 to 1.47)
p value for linear trend	0.59	0.39	0.44	0.46	0.61	0.70	0.12	0.38
Vitamin E								
1 (low)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2	0.81 (0.50 to 1.32)	0.84 (0.49 to 1.43)	0.95 (0.56 to 1.61)	0.92 (0.52 to 1.64)	1.02 (0.69 to 1.49)	0.95 (0.63 to 1.44)	0.92 (0.59 to 1.45)	0.90 (0.54 to 1.47)
3	1.20 (0.76 to 1.90)	1.27 (0.74 to 2.18)	1.39 (0.84 to 2.28)	1.35 (0.75 to 2.42)	0.84 (0.56 to 1.26)	0.71 (0.45 to 1.12)	0.58 (0.35 to 0.96)	0.48 (0.27 to 0.86)
4 (high)	1.42 (0.89 to 2.26)	1.43 (0.80 to 2.56)	1.59 (0.95 to 2.66)	1.46 (0.77 to 2.76)	1.19 (0.80 to 1.76)	0.94 (0.58 to 1.53)	1.03 (0.65 to 1.64)	0.85 (0.48 to 1.51)
p value for linear trend	0.07	0.09	0.04	0.13	0.59	0.64	0.70	0.35
Carotene								
1 (low)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2	0.85 (0.58 to 1.24)	0.89 (0.61 to 1.30)	0.67 (0.42 to 1.07)	0.70 (0.43 to 1.12)	0.89 (0.56 to 1.41)	0.85 (0.53 to 1.37)	0.87 (0.54 to 1.42)	0.81 (0.49 to 1.33)
3	1.01 (0.70 to 1.46)	1.08 (0.73 to 1.59)	0.88 (0.56 to 1.37)	0.83 (0.52 to 1.32)	1.07 (0.69 to 1.68)	0.98 (0.61 to 1.58)	0.90 (0.56 to 1.45)	0.82 (0.49 to 1.36)
4 (high)	0.84 (0.58 to 1.23)	0.95 (0.62 to 1.46)	0.87 (0.55 to 1.35)	0.84 (0.51 to 1.41)	0.99 (0.63 to 1.57)	0.90 (0.53 to 1.50)	0.91 (0.56 to 1.47)	0.78 (0.45 to 1.35)
p value for linear trend	0.58	0.93	0.80	0.66	0.81	0.85	0.74	0.41
Retinol								
1 (low)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
2	0.75 (0.51 to 1.10)	0.72 (0.50 to 1.04)	0.56 (0.35 to 0.89)	0.53 (0.33 to 0.85)	1.07 (0.68 to 1.69)	1.08 (0.68 to 1.72)	1.31 (0.80 to 2.12)	1.31 (0.79 to 2.15)
3	0.88 (0.60 to 1.28)	0.84 (0.58 to 1.21)	0.68 (0.43 to 1.06)	0.64 (0.40 to 1.03)	0.88 (0.55 to 1.42)	0.94 (0.57 to 1.54)	1.02 (0.61 to 1.71)	1.03 (0.60 to 1.76)
4 (high)	0.83 (0.56 to 1.22)	0.77 (0.51 to 1.15)	0.67 (0.42 to 1.05)	0.67 (0.40 to 1.12)	1.16 (0.73 to 1.84)	1.29 (0.77 to 2.16)	1.20 (0.72 to 1.99)	1.21 (0.69 to 2.12)
p value for linear trend	0.50	0.33	0.14	0.19	0.69	0.44	0.71	0.74

*All effect estimates and confidence intervals are adjusted for intra-family clustering. †Fully adjusted models include age, sex, energy intake, food expenditure, Townsend score, season, and district.

Table 6 Odds ratios* for incidence and mortality from breast cancer in relation to diet (n=1959)

Quartiles of intake	Breast cancer incidence		Breast cancer mortality	
	Age, energy, adjusted	Fully adjusted†	Age, energy, adjusted	Fully adjusted†
Fruit				
1 (low)	1.0	1.0	1.0	1.0
2	0.66 (0.33 to 1.33)	0.66 (0.33 to 1.34)	1.23 (0.45 to 3.35)	1.26 (0.45 to 3.47)
3	1.04 (0.55 to 1.96)	1.10 (0.58 to 2.10)	1.10 (0.40 to 3.06)	1.19 (0.42 to 3.35)
4 (high)	1.04 (0.54 to 2.01)	1.08 (0.52 to 2.25)	1.28 (0.46 to 3.52)	1.25 (0.40 to 3.92)
p value for linear trend	0.65	0.61	0.70	0.73
Vegetables				
1 (low)	1.0	1.0	1.0	1.0
2	1.36 (0.72 to 2.57)	1.37 (0.72 to 2.63)	1.53 (0.65 to 3.63)	1.47 (0.61 to 3.57)
3	0.85 (0.42 to 1.70)	0.86 (0.42 to 1.76)	0.41 (0.12 to 1.34)	0.38 (0.11 to 1.30)
4 (high)	1.44 (0.75 to 2.75)	1.43 (0.70 to 2.92)	1.03 (0.40 to 2.62)	0.86 (0.30 to 2.47)
p value for linear trend	0.55	0.59	0.51	0.35
Vitamin C				
1 (low)	1.0	1.0	1.0	1.0
2	1.46 (0.77 to 2.79)	1.50 (0.78 to 2.88)	1.13 (0.46 to 2.82)	1.17 (0.46 to 2.94)
3	1.13 (0.56 to 2.30)	1.10 (0.54 to 2.26)	0.68 (0.24 to 1.90)	0.67 (0.23 to 1.91)
4 (high)	1.10 (0.53 to 2.26)	0.99 (0.45 to 2.15)	0.68 (0.24 to 1.92)	0.58 (0.19 to 1.84)
p value for linear trend	0.99	0.78	0.32	0.24
Vitamin E				
1 (low)	1.0	1.0	1.0	1.0
2	1.27 (0.69 to 2.33)	1.18 (0.63 to 2.19)	1.15 (0.52 to 2.54)	1.00 (0.44 to 2.28)
3	0.78 (0.39 to 1.57)	0.70 (0.34 to 1.44)	0.26 (0.08 to 0.83)	0.19 (0.06 to 0.65)
4 (high)	0.88 (0.44 to 1.76)	0.77 (0.37 to 1.64)	0.34 (0.12 to 0.96)	0.25 (0.08 to 0.80)
p value for linear trend	0.48	0.30	0.007	0.002
Carotene				
1 (low)	1.0	1.0	1.0	1.0
2	1.38 (0.73 to 2.59)	1.36 (0.71 to 2.59)	0.60 (0.23 to 1.62)	0.58 (0.21 to 1.56)
3	1.32 (0.69 to 2.55)	1.32 (0.67 to 2.58)	1.05 (0.43 to 2.57)	0.96 (0.38 to 2.42)
4 (high)	0.99 (0.50 to 1.97)	0.93 (0.44 to 1.96)	0.86 (0.34 to 2.16)	0.68 (0.25 to 1.91)
p value for linear trend	0.94	0.84	0.96	0.72
Retinol				
1 (low)	1.0	1.0	1.0	1.0
2	0.96 (0.46 to 2.00)	1.00 (0.48 to 2.10)	0.91 (0.29 to 2.86)	0.95 (0.30 to 3.01)
3	1.91 (0.98 to 3.74)	1.95 (0.98 to 3.87)	2.08 (0.76 to 5.67)	2.19 (0.78 to 6.18)
4 (high)	1.68 (0.83 to 3.38)	1.75 (0.84 to 3.66)	1.66 (0.58 to 4.72)	1.94 (0.64 to 5.92)
p value for linear trend	0.05	0.05	0.18	0.12

*All effect estimates and confidence intervals are adjusted for intra-family clustering. †Fully adjusted models include age, energy intake, food expenditure, Townsend score, and season.

fruit consumption and fully adjusted for other potential confounding factors were 1.0 (reference) 1.49 (1.02 to 2.17), 1.51 (1.01 to 2.27), 1.72 (1.04 to 2.83); p value for linear trend=0.04. Results for total cancer incidence, cancers related to smoking, and cancers not related to smoking were similar but weaker. There was no evidence that the association between fruit intake and cancer incidence differed at different levels of energy intake (p interaction =0.84).

DISCUSSION

Main findings

The main finding in this analysis was that increased fruit consumption in childhood was inversely associated with cancer incidence in adulthood. Associations with cancer mortality were weaker. No clear association between vegetable intake and cancer risk was apparent.

Previous studies

No previous longitudinal studies using diet measured during childhood have examined the association of early fruit, vegetable, and antioxidant vitamin intake with later risk of cancer. In the most closely comparable previous study of childhood fruit intake, Potischman and colleagues[4] report no association between recalled adolescent (combined) fruit and vegetable intake and breast cancer incidence. Our null findings in relation to vegetable consumption are consistent with those from previous research of recalled childhood food habits and adult cancer risk,[4 5 11] although non-significant decreases in risk of breast[8] and prostate cancer[9] associated with a vegetarian diet before the age of 15 years have been reported. The only previous report of childhood vitamin E intake and cancer showed no association between recalled intake and gastric cancer risk.[6]

The findings for fruit intake in this study are consistent with studies on adult fruit intake and cancer risk.[2] A number of observational studies of adult diet report a reduced risk of a range of cancers in relation to higher levels of vegetable consumption.[2 28] However, in a cohort study of UK men, fruit was more strongly inversely associated with cancer risk than vegetables.[29]

The lack of association between fruit and vegetable intake in childhood and breast cancer risk supports the result of a recent meta-analysis of cohort studies of adult diet and breast cancer risk.[30] Epidemiological evidence of a protective role of vitamin E intake against breast cancer in adult studies is weak,[2 31] although lower prostate cancer risk associated with vitamin E supplementation has been reported from a large randomised controlled trial.[32] The higher risk of breast cancer associated with increasing intake of retinol is surprising and in contrast with the weak but consistent observational evidence that higher intake of vitamin A, including preformed retinol, is associated with lower risk of breast cancer.[28]

Mechanisms

DNA damage is implicated in the initiation of cancer. It has been suggested that antioxidant constituents in fruit and vegetables protect against free radical mediated damage to DNA and this may underlie any protective effect of early diet on adult cancer risk. Alternatively, the many other constituents in fruit and vegetables, as well as antioxidants, such as fibre, isoflavones, coumarins, and glucosinolates, may be important.[29]

Key points

- Observational studies in adulthood suggest that higher levels of fruit and vegetable consumption are associated with decreased risk of some cancers.
- Antioxidants have been suggested as possibly mediating this protective effect through the prevention of oxidative damage to DNA.
- This study is the first to examine associations between fruit, vegetables, and antioxidants measured during childhood and subsequent cancer risk.
- Childhood fruit consumption seems to be protective against later development of cancer but associations with individual antioxidants were weaker or non-existent for cancers as a whole.

This is supported by the finding in this study that early consumption of fruit is associated with decreased cancer risk, but that individual antioxidants (vitamin C, E, and carotene) had a weaker effect. However, vegetables are also a rich source of potential anticarcinogenic compounds but we found no association between total vegetable consumption and cancer risk. It is possible that the convention for prolonged cooking of vegetables at the time of the original survey (many of the protective substances in vegetables are heat labile) may have contributed to this finding. Values for the vitamin content of vegetables in food tables published in the 1930s are based on analysis of foodstuffs "allowed to boil briskly" for up to 60 minutes.[21] In modern food tables[18] the maximum boiling time for most vegetables is 20 minutes (5–10 minutes for many items) reflecting the changes in cooking practices over the past 60 years.

The strong reduction in risk of breast cancer associated with higher vitamin E intake should be interpreted with caution in view of the absence of any association with other cancers and its weaker association with breast cancer incidence. In vitro studies show vitamin E to be a potent antioxidant and animal studies have shown a reduction in mammary gland tumours associated with increased vitamin E intake.[31] However, information on the blend of oils and fats used in margarine in the 1930s, and the fats used in prepared foods such as biscuits and cakes, potentially major sources of vitamin E in this study, is limited. The extent to which these factors vary, for example, by social class is therefore unknown. Such uncertainty means that the findings for vitamin E and breast cancer must be viewed with caution as socially patterned misclassification could confound observed associations.

Study strengths and limitations

This study has three main strengths. Firstly, diet was measured in childhood long before the occurrence of disease thus avoiding the problem with recall bias encountered in case-control studies based on recalled childhood diet. Secondly, all foods consumed in the home were assessed, facilitating analysis at the food and nutrient level, but also consideration of the potential for other dietary factors—such as energy intake—to confound the relation between the constituents of interest and cancer. Thirdly, dietary habits and cancer risk are known to be socially patterned.[28] A number of indicators of socioeconomic position were collected in the original survey, allowing us to assess possible confounding by these factors.

There are a number of possible methodological limitations to these analyses. Firstly, the measure of childhood diet is derived from a study of household diets. This is therefore not a direct measure of individual diet. In dividing diet by total number of household members, two main assumptions are made. The first is that diets of children and adults are qualitatively similar. In support of this assumption there were few foods recorded by the Boyd Orr families aimed specifically at children in the way that they are today. There is some anecdotal evidence that "food fads" among children may be less of a phenomenon in this cohort, where most of the families were poor, than it might be today or in a wealthier cohort.[33] The second assumption is that foods and nutrients are distributed evenly within families, however, there is evidence historically and cross culturally that food is not always distributed within families according to need.[34] Nutrient distribution within households does not accord with dietary reference values nor do the reference values give any indication of the distribution of foods within households.[35 36] If the measurement error inherent in these limitations were random then this is likely to lead to underestimation of any diet and disease associations. If, however, there is systematic misclassification of dietary intake then any diet-cancer associations may be biased.[37] It is difficult to establish the extent to which this may be the case. If, for example, division of foods within families varied with social class such that intake was overestimated in wealthier families and underestimated in poorer families this would inflate diet-cancer associations for cancers that follow this same social patterning. A second limitation is that, in total, 50 diet-disease associations were examined in the fully adjusted models of which two were statistically significant at the 5% level. Therefore, as an explanation of the findings the role of chance cannot be excluded. A crude means of controlling for the effects of multiple hypothesis testing is the Bonferroni adjustment,[38] however, this may be an over-conservative measure as a number of the associations are correlated. For example, associations between total cancer incidence and mortality are likely to be similar. Thirdly, adult risk factors such as adult diet and smoking could not be controlled for in the analysis. Adult diet is associated with cancer risk in a number of studies among which the evidence for fruit and vegetables in a protective role is the most convincing.[2] Although there are many influences on diet throughout life, adult food habits may, in part, be established in childhood. To assess the possible confounding effect of smoking on the observed patterns of risk we assessed diet-cancer associations separately for smoking and non-smoking related cancers (table 5). There was no strong evidence that fruit-cancer associations differed greatly for smoking compared with non-smoking related cancers. This contrasts with a previous analysis of the cohort[13] in which associations with childhood energy intake were strongest in relation non-smoking related cancers. The findings in the present analysis are consistent with the possibility that childhood fruit intake may be protective against a wide range of cancers, both related and unrelated to smoking, through some common pathway.

Current policy context

It is recommended that adults eat at least five portions of fruit and vegetables per day (about 400 g) in order to promote good health and to prevent cardiovascular disease and cancer. Children over 5 years are recommended to follow a diet "consistent with the recommendations for adults".[28 39] In the recent UK National Diet and Nutrition Survey (NDNS) of 4–18 year olds,[40] median consumption of fruit and vegetables was 125 g and 112 g per day respectively. While these values are considerably higher than average values for the Boyd Orr children (table 2), only just over 25% of children in the NDNS study had consumed any citrus fruit during the survey period and almost a quarter of vegetable consumption was of baked beans. This study provides some support for dietary guidelines focusing on fruit consumption rather than on the intake of particular micronutrients.

Conclusion

This study shows that childhood fruit consumption may have a long term protective effect on cancer risk in adulthood. Prospective studies with individual measures of diet are required to further investigate these associations.

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PostScript

BOOK REVIEWS

Children of the 21st century. From birth to 9 months

Edited by Shirley Dex, Heather Joshi. Published by Policy Press, Bristol, 2005, 282 pp, £24.99 (softcover). ISBN 1-86134-6881-3

This book offers the first report on data obtained from the Millennium Cohort Study (MCS), a cohort of 18 819 UK babies born in the 21st century. The book takes an original approach to the study of child health and development by considering social and health conditions as sources of capital. For instance, family resources (father, mother and relatives) and neighbourhood are studied as sources of social capital, ethnicity, religion and language as sources of cultural capital, and parents' health as a source of human capital. Doing so, the authors open a general concern that inequalities are not only rooted in material goods, but also in social (neighbourhood) and cultural (ethnicity) domains, as is shown throughout the book.

The first chapter describes how MCS was planned, what objectives it pursued and what were the policy context and the methods used. Then a series of chapters present the distribution of variables within the sample in terms of: household structure, parents language, neighbourhood conditions (chapter 2), socioeconomic conditions (chapter 3), prenatal care (chapter 4), children's health (chapter 5), and parenthood and parenting (chapter 7). Chapters 6 and 8 bring more analytically elaborated answers, which we expect to see in a book of this character, describing how socioeconomic and physical environments affect child development (gross and fine motor coordination, and communicative gestures), and how family characteristics and employment affect mothers' mental health (satisfaction in life and depression). The final chapter presents the conclusions, a summary of the information and answers for the questions asked in the introduction of the book.

Most chapters are accompanied by a considerable number of graphics and tables that are easy to interpret. Although the book is the result of a multidisciplinary effort, chapters show coherence and cohesion. An introductory discussion at the beginning of each chapter summarises the state of knowledge and helps the reader to be situated within the context to be developed. Subsequently, a plan of the chapter clarifies the measures and analysis used. All chapters deal with issues of class (ie, education and occupation), ethnicity and neighbourhood inequalities and the manner in which they are related to other variables such as prenatal care, poverty, child's health, etc. Although this sometimes adds complexity to the discussion, it considerably enriches its content. Methodological topics are appropriately presented. For instance, comparison of different definitions of poverty (chapter 3), the use of positive child health indicators such as adequate environment (being breastfed, being completely immunised and living with a non-smoking mother during infancy; chapter 5), and especially measurement of the father's involvement in the child's care and parenting attitudes (chapters 7 and 8) are assets of this publication. The discussions in the text and the concluding chapter are clear and focus on the research questions, always leading to policy recommendations.

This book is an excellent introduction to the dynamics of family structure, ethnicity and social position and its effect on child health and development. Researchers on child health in developing countries will find in it helpful information about methods, definition of variables, presentation and policy implications of results. Concepts of human, social and cultural capital overlap with the concept of CARE used by many researchers in international nutrition. Thus, the book will also be a useful reference for those working in that field. Likewise, this work should be of interest to policy makers and government agencies around the world.

Beatriz E Alvarado

Mortality, Biochemistry, Diet and Lifestyle in Rural China. Geographical Study of the characteristics of 69 Counties in mainland China and 16 Areas in Taiwan

Edited by J Chen, R Peto, W Pan, B Liu, T C Campbell, J Boreham, B Parpia, P Cassano, Z Chen, Z Feng, H Gelband, J Li, H Pan, M Root, Y Wu, L Youngman. Oxford University Press, Oxford, 2006, $295, pp 840. ISBN 0-19-856933-5

China has the large population of people who have different diets, habits and lifestyles from Western people. What's more, enormous difference also exist within the Chinese as a result of geographical differences. This monograph offers a great mass of first-hand Chinese data involving diets, habits and lifestyles, and the corresponding differences in anthropometry and blood biochemistry. A total of 69 rural counties in the mainland and 16 cites in Taiwan were chosen. These 69 rural counties are distributed throughout mainland China, in terms of mass, which well reasonably represents the whole rural area in mainland China. In Taiwan, these 16 survey cities were chosen, including different types from typical urban to rural.

The monograph comprises four sections, study description and methods, summary statistics for all 639 variables, and detailed displays for 333 of 639 variables, with a foreword and an appendix. The results mainly contain: (1) death rates and their specific causes at the county level during 1986–8 (all 69 counties, obtained by retrospective sampling survey) and 1973–5 (65 of 69 counties, referred from a previous nationwide study); (2) analysis of blood and urine biochemistry, dietary, lifestyle, anthropometric and socioeconomic characteristics in 1989 (all 69 counties) and 1983 (65 of 69 counties); and (3) retrospective study on mortality in 16 cities in Taiwan in (1986–8) and a similar survey on biochemistry, diet and lifestyle in mainland China (1989).

This herculean task was finished owing to the contribution of thousands of people, which is also a successful example of epidemiological collaboration across the Taiwan Strait. The original data, and the findings of the geographical associations between the death rates and the multiple local characteristics (such as biochemical, dietary and behavioural factors), provide clues for further aetiological studies in the field of epidemiological research. However, the study is an ecological study in the combined use of sampling technique, retrospective analysis, questionnaire survey and laboratory detection. The 69 rural counties were distributed throughout mainland China, but mainly in the southeast coastal region, and the observational unit comprised all the research sites but not individual sites. The bias, mainly "ecological fallacy", information bias and selection bias, is unavoidable. Hence, not all results with statistical significance in this study have practical significance or will be found to be true in the future. The readers must selectively refer to the observations and findings in consideration of correlative knowledge.

Kun Chen, Mingjuan Jin

CORRECTIONS

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D F Salerno. Fiorella Belpoggi, MD: per aspera ad astra (*J Epidemiol Community Health* 2006; **60**:1019). In this article Fiorella Belpoggi appears as an MD; she actually holds a PhD.

M Maynard, D Gunnell, P Emmett, *et al*. Fruit, vegetables, and antioxidants in childhood and risk of adult cancer: the Boyd Orr cohort (*J Epidemiol Community Health* 2003;**57**:218–25).

Since the publication of the above paper, as a result of further analyses on the cohort, the authors have identified an error in the coding of the milk and egg consumption for some families. This occurred because the Boyd Orr childhood dietary data record *family* consumption in *weekly* amounts and the database used in recoding dietary intake for this paper was a modified version of one designed for coding *daily* intakes of *individuals*. Extremely large quantities of milk and eggs were not accepted by the package resulting in an error term. These error terms were detected and corrected (Maynard M. Diet in childhood and risk of cancer in adulthood. PhD thesis, University of Bristol, 2000). It was not noted, however, that the programme was interpreting some of the moderate amounts as data entry error and inserting a decimal place into the value (for example an intake of 2000 g became coded as 2.000 g. Of particular relevance to the article these underestimates resulted in errors in our calculation of total energy and vitamin A intake for some families. The effect on odds ratios of diet-cancer associations was to slightly weaken some and to strengthen others.

The main association highlighted in the article and abstract was that between fruit consumption and cancer incidence. In fully adjusted logistic regression models, odds ratios with increasing quartiles of fruit consumption in the original findings were: 1.0 (reference), 0.66 (0.48 to 0.90), 0.70 (0.51 to 0.97), 0.62 (0.43 to 0.90); p value for linear trend = 0.02. These findings are now 1.0 (reference), 0.67 (0.49 to 091), 0.74 (0.54 to 1.01), 0.62 (0.43 to 0.91); p = 0.03. The interpretation of the data therefore remains the same. The other associations altered in a re-analysis using the corrected data are (i) the apparent adverse effect of vitamin E on total cancer incidence/mortality and smoking-related cancer incidence/mortality—these adverse effects are now slightly stronger (p = 0.02 to p = 0.04 respectively) and (ii) the association between higher energy intake and cancer risk, which is weaker: the OR for cancer mortality across quartiles of increasing energy intake were 1.0 (reference), 1.54 (1.07 to 2.22), 1.12 (0.74 to 1.70), 1.62 (0.96 to 2.74) (p = 0.23 in fully adjusted models). A previous analyses of the energy intake-cancer incidence in this cohort (Frankel *et al*. *BMJ* 1998;**316**:499–504) is not affected as this used cohort members' energy intakes as estimated at the time of the original survey.