Case-control study of breast cancer in south east England: nutritional factors

J Cade, E Thomas, A Vail

Abstract
Objectives—To explore dietary risk factors, in particular fat intake, for breast cancer, using an approach to reduce recall bias of subjects and so provide a more reliable estimate of dietary intake than previous similar studies.

Design—A case-control study of women aged 50–65 years attending the breast assessment clinics of the breast screening programme in Southampton and Portsmouth, southern England. Data were analysed for all women requiring further clinical procedures; all women recalled to have an early rescreen; and a random sample of women found to be normal and referred for a routine rescreening appointment (standard recall).

Measurements—An interview obtained information on various lifestyle characteristics including smoking and alcohol intake, weight, waist, and hip measurements were also taken at the clinic. Women were given a detailed questionnaire on food intake to complete at home and return by post.

Results—1577 women were included in the study: 220 with breast cancer (cases); 353 early rescreen and 825 given a standard recall with benign breast disease; 353 early study: 220 with breast cancer (cases); 179 of cases with that of each control group. Logistic regression analyses were carried out comparing the dietary intake of cases with that of each control group adjusting for important demographic and reproductive factors. Results for the case and standard recall comparison are presented. The only non-calorific nutrient to reach significance was iron, which was negatively associated with risk (p=0.03). For fat intake, the odds decreased with increasing polyunsaturated fat (p=0.15), showed no trend with monounsaturated fat (p=0.37) and increased (p=0.10) with increasing saturated fat. No pattern was clear for the other calorie providing nutrients.

Conclusions—In line with recent cohort studies, this study has shown no evidence to support the hypothesis that dietary fat is an important contributor to breast cancer rates. Biases should have been reduced by studying subjects from the screening programme who were at an early stage of disease.

The aetiology of breast cancer is uncertain. Various risk factors have been postulated. Diet has been prominent among the hypothesised environmental risk factors but few, if any, constituents of the diet can definitely be associated with the disease. Most risk factors are associated with only a modest increase in risk. The American Cancer Society has estimated that only about one quarter of breast cancer cases can be accounted for by known risk factors. It is important to discover if diet is involved in the aetiology of breast cancer, as it is more amenable to change than some other risk factors (for example, age at menarche).

Dietary fat has long been suspected of playing a part in the aetiology of breast cancer. Animal experiments in the early 1940s showed a positive relation between a high fat diet and risk of mammary gland cancer. A meta-analysis of animal experiments has shown that not only is total fat consumption a risk factor but also type of fat is important, linoleic acid (a polyunsaturated fat) and lard (a saturated fat) may increase risk, whereas fish oil (also a polyunsaturated fat) may be protective.

Ecological studies have shown a positive relationship between a high fat diet and risk of breast cancer mortality: both between countries and over time in the same country. However, the findings of these studies are limited. Average per capita consumption data take no account of individual differences in dietary practices and there may be other factors related to fat intake that are the real cause of the high correlations. Migrant studies have shown an increase in breast cancer rates with increasing fat intake.

Case-control studies relevant to fat and breast cancer have reported conflicting results. A review of 35 case-control studies showed increasing risk in association with high meat consumption in 12 studies. Fourteen of the studies presented odds ratios for total fat intake. Most of the studies suggested an increasing risk in association with high meat consumption in 12 studies. Fourteen of the studies presented odds ratios for total fat intake. Most of the studies suggested an increase in risk of breast cancer with higher fat consumption, but only two had confidence intervals excluding unity. One study suggested a protective effect of a higher fat diet. A meta-analysis of 12 case-control studies showed a statistically significant increased risk of breast cancer in women who consumed more fat, primarily because of intake of saturated and monounsaturated fats, rather than polyunsaturated fat. A disadvantage of case-control studies is that misclassification may arise because of bias in selection of patients and bias in recall of past diet. Recall of usual pre-disease intake is influenced by current, different
Table 1  Nutrient intake (median (2.5 and 97.5 percentiles) for cases and control in including the National Diet and Nutrition Survey (NDNS) results for comparison)

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Case median</th>
<th>Benign breast disease median</th>
<th>Early rescreen median</th>
<th>Standard recall median</th>
<th>NDNS median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total energy (kcal)</td>
<td>1642 (728, 3044)</td>
<td>1612 (774, 3126)</td>
<td>1645 (900, 3124)</td>
<td>1611 (790, 2987)</td>
<td>1620 (810, 2340)</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>75.4 (40.0, 131.0)</td>
<td>75.0 (50.0, 131.4)</td>
<td>75.0 (40.0, 131.6)</td>
<td>75.0 (37.0, 133.4)</td>
<td>75.0 (33.7, 81.8)</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>197 (73, 399)</td>
<td>207 (91, 443)</td>
<td>206 (91, 436)</td>
<td>198 (87, 390)</td>
<td>198 (84, 285)</td>
</tr>
<tr>
<td>Complex carbohydrate (g)</td>
<td>100 (29, 241)</td>
<td>106 (30, 233)</td>
<td>106 (44, 252)</td>
<td>104 (36, 252)</td>
<td></td>
</tr>
<tr>
<td>Sugars (g)</td>
<td>99 (28, 199)</td>
<td>90 (34, 213)</td>
<td>93 (39, 232)</td>
<td>91 (31, 187)</td>
<td></td>
</tr>
<tr>
<td>Total fat (g)</td>
<td>60.0 (23.0, 118.9)</td>
<td>59.0 (26.0, 129.0)</td>
<td>59.0 (29.0, 131.2)</td>
<td>59.0 (22.4, 122.7)</td>
<td>78 (25, 147)</td>
</tr>
<tr>
<td>Monounsaturated fat (g)</td>
<td>23.0 (8.0, 49.0)</td>
<td>23.0 (9.0, 51.5)</td>
<td>23.0 (10.0, 48.2)</td>
<td>24.0 (8.0, 49.4)</td>
<td>70.2 (32.3, 112.5)</td>
</tr>
<tr>
<td>Polyunsaturated fat (g)</td>
<td>23.0 (8.0, 50.4)</td>
<td>24.0 (8.5, 55.0)</td>
<td>23.0 (10.0, 56.2)</td>
<td>24.0 (8.1, 52.2)</td>
<td>20.5 (10.1, 35.3)</td>
</tr>
<tr>
<td>Saturated fat (g)</td>
<td>23.0 (8.0, 50.4)</td>
<td>24.0 (8.5, 55.0)</td>
<td>23.0 (10.0, 56.2)</td>
<td>24.0 (8.1, 52.2)</td>
<td>7.9 (2.9, 20.8)</td>
</tr>
<tr>
<td>Alcohol (g)</td>
<td>1.0 (0.3, 2.5)</td>
<td>1.0 (0.3, 2.5)</td>
<td>1.0 (0.3, 2.5)</td>
<td>1.0 (0.3, 2.5)</td>
<td>1.0 (0.3, 2.5)</td>
</tr>
<tr>
<td>Fibre (g)</td>
<td>24.0 (7.0, 49.4)</td>
<td>26.0 (9.0, 62.5)</td>
<td>26.0 (9.8, 61.0)</td>
<td>25.0 (9.0, 62.5)</td>
<td>18.6 (8.6, 32.9)</td>
</tr>
<tr>
<td>Cholesterol (mg)</td>
<td>260 (88, 590)</td>
<td>242 (83, 730)</td>
<td>259 (99, 569)</td>
<td>247 (77, 586)</td>
<td>288 (113, 515)</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>98.5 (16.5, 272.8)</td>
<td>103.0 (28.5, 276.7)</td>
<td>110.0 (23.0, 277.2)</td>
<td>107.0 (29.0, 235.0)</td>
<td>58.8 (17.9, 169)</td>
</tr>
<tr>
<td>β Carotene (mg)</td>
<td>390.0 (348, 496.0)</td>
<td>3272 (527, 9778)</td>
<td>3530 (415, 8804)</td>
<td>3457 (609, 8384)</td>
<td>1848 (214, 7121)</td>
</tr>
<tr>
<td>Retinol (µg)</td>
<td>705 (82, 1702)</td>
<td>690 (44, 3144)</td>
<td>585 (91, 2967)</td>
<td>672 (76, 2970)</td>
<td>5163 (192, 5487)</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>89.0 (304, 1702)</td>
<td>867 (313, 1819)</td>
<td>864 (303, 1648)</td>
<td>843 (346, 1741)</td>
<td>7312 (305, 1113)</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>12.0 (5.0, 29.0)</td>
<td>13.0 (6.7, 31.4)</td>
<td>13.0 (5.0, 30.2)</td>
<td>12.7 (6.0, 28.4)</td>
<td>10.12 (5.6, 12.1)</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>10.0 (4.5, 22.0)</td>
<td>10.0 (4.9, 21.5)</td>
<td>11.0 (5.0, 19.0)</td>
<td>10.0 (5.0, 20.0)</td>
<td>8.3 (4.5, 13.7)</td>
</tr>
</tbody>
</table>

* Information not available. † From n-6 polyunsaturated fatty acids only. ‡ From food sources only.
were compared with those with complete data using logistic regression to assess differences in disease status, demographic, and reproductive details. Completed values for each case and control group of subjects were compared with the UK National Diet and Nutrition Survey (NDNS).26

Unconditional logistic regression models were fitted to analyse the nutritional data for each control group. These were made up of three components: important demographic and reproductive factors determined from previous modelling22 (age group at screening, age at menarche, age at first birth, social class, body mass index, and smoking); components of caloric intake (alcohol, complex carbohydrates, protein, polyunsaturated fat, monounsaturated fat, saturated fat, cholesterol, and sugar); and non-calorific nutrients (β-carotene, calcium, fibre, iron, retinol, vitamin C, vitamin E, and zinc). Full models, including all three components, were fitted using quartiles of nutrient values. As this study was primarily concerned with dietary fat, non-calorific nutrients were eliminated from reported models unless at least one quartile attained a statistical significance of 10%. From the final model, an estimate of the odds ratio and 95% confidence intervals were calculated for each quartile of nutrients remaining in the model. Evidence of a linear trend in the odds ratios for quartiles of each nutrient was also tested.

To assess possible under-reporting, estimated basal metabolic rates were calculated from anthropometric data27 and compared with self reported dietary intakes of energy.

### Results

Interviews were carried out on 1947 women (98% response) in Southampton and 1280 women (96% response) in Portsmouth. From this number, 1813 subjects were chosen for further study.23 Of these, 87% (1577) returned the postal questionnaire with usable dietary information. We found no evidence that the 236 women with missing nutrient values had different distributions of the demographic and reproductive characteristics, nor of disease status, though they were more likely to have missing reproductive data.

The 1577 subjects consisted of 220 women with breast cancer (cases); 179 women with benign breast disease; 353 women called back for an early rescreen and 825 who were given a standard recall appointment.

Summary statistics for nutritional intake (table 1) are given in a format comparable to that given by the NDNS report.22 Few differences in nutritional intake were apparent between the four groups in this study. Although subjects had a similar intake of calories to the NDNS, a smaller percentage of total energy came from fat intake. Our subjects also had higher intakes of most vitamins and minerals measured.

Adjusted odds ratios and 95% confidence intervals are shown for the logistic regression model comparing dietary intake for cases with that of the standard recall controls only (table 2), as results from other comparison groups

#### Table 2 Results of logistic regression analysis comparing cases and standard recall groups

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Quartile</th>
<th>OR (95% CI)*</th>
<th>z test for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated fat</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>2.46 (1.43, 4.24)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1.79 (0.93, 3.46)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>2.35 (1.11, 4.95)</td>
<td></td>
</tr>
<tr>
<td>Monounsaturated fat</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.18 (0.68, 2.06)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.69 (0.36, 1.32)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.86 (0.41, 1.80)</td>
<td></td>
</tr>
<tr>
<td>Polyunsaturated fat</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.95 (0.55, 1.63)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.88 (0.47, 1.67)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.61 (0.30, 1.26)</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.18 (0.68, 2.04)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1.30 (0.69, 2.43)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>1.17 (0.56, 2.47)</td>
<td></td>
</tr>
<tr>
<td>Complex carbohydrate</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.17 (0.73, 1.88)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.84 (0.48, 1.45)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.77 (0.41, 1.42)</td>
<td></td>
</tr>
<tr>
<td>Sugar</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.74 (0.44, 1.22)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1.53 (0.93, 2.53)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>1.28 (0.73, 2.24)</td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.77 (0.49, 1.21)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.97 (0.63, 1.49)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.97 (0.61, 1.54)</td>
<td></td>
</tr>
<tr>
<td>Iron</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>0.82 (0.48, 1.40)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>0.51 (0.26, 0.99)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.49 (0.23, 1.01)</td>
<td></td>
</tr>
<tr>
<td>Vitamin E</td>
<td>1</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1.42 (0.79, 2.55)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1.36 (0.65, 2.86)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>2.05 (0.93, 4.56)</td>
<td></td>
</tr>
</tbody>
</table>

* OR adjusted for demographic and reproductive factors determined by previous modelling.

#### Table 3 Ratio of energy intake (MJ) to calculated basal metabolic rate for subjects

<table>
<thead>
<tr>
<th>Cases cumulative</th>
<th>Benign breast disease cumulative</th>
<th>Early rescreen cumulative</th>
<th>Standard recall cumulative</th>
<th>NDNS study cumulative</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.0</td>
<td>32</td>
<td>33</td>
<td>33</td>
<td>26</td>
</tr>
<tr>
<td>1.2</td>
<td>55</td>
<td>58</td>
<td>54</td>
<td>47</td>
</tr>
<tr>
<td>1.4</td>
<td>75</td>
<td>73</td>
<td>75</td>
<td>71</td>
</tr>
<tr>
<td>1.6</td>
<td>86</td>
<td>84</td>
<td>89</td>
<td>87</td>
</tr>
<tr>
<td>All</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Number</td>
<td>196</td>
<td>166</td>
<td>326</td>
<td>763</td>
</tr>
<tr>
<td>Mean</td>
<td>1.19</td>
<td>1.20</td>
<td>1.23</td>
<td>1.20</td>
</tr>
<tr>
<td>Median</td>
<td>1.15</td>
<td>1.13</td>
<td>1.17</td>
<td>1.16</td>
</tr>
<tr>
<td>5th percentile</td>
<td>0.61</td>
<td>0.64</td>
<td>0.70</td>
<td>0.63</td>
</tr>
<tr>
<td>95th percentile</td>
<td>1.88</td>
<td>1.93</td>
<td>2.06</td>
<td>1.93</td>
</tr>
</tbody>
</table>
were not materially different. The only non-
calorific nutrients remaining in the model after
exclusions were iron and vitamin E. Subjects
with iron intakes above the median value were
at half the odds of those with intakes below the
median. Subjects with vitamin E intake above
the reference category had increased odds with
some slight evidence of linear trend \((p=0.10)\).
For fat intake, the odds decreased with
increasing polyunsaturated fat \((p=0.15)\),
showed no trend with monounsaturated fat
\((p=0.37)\), and were increased with increased
saturated fat, although no trend was apparent
\((p=0.10)\). No pattern was clear for other calo-
rie providing nutrients. Comparisons were
made between the cases and the other control
groups and similar results were found.

The ratio of energy intake (MJ) to the cal-
lculated basal metabolic rate for those subjects
with recorded height and weight is shown for
the four groups separately (table 3). Little dif-
cference was apparent between the groups. A
higher proportion of subjects in this study than
in the NDNS had low ratios indicating possible
under-reporting.

Discussion
The response rate to this study was high. Of
those interviewed who were to be analysed,
87% returned the postal questionnaire with
usable dietary information.

The method chosen to assess dietary intake
was a food frequency questionnaire (FFQ).
This method asks subjects to describe their
usual intake of foods rather than measuring
actual intake. When carefully designed, FFQs
have been shown to provide valid and repeat-
able measures of nutrient intake.\(^{28}\) The FFQ
used in this study had been previously
validated in an Australian population,\(^{22,23}\) it
was then piloted and adapted for use in our study
population.

The FFQ is the most appropriate dietary
assessment method for large scale epidemi-
ological studies. It can be completed by the
study subjects and does not entail complicated
weighing or recording procedures, which can
lead to changes in diet or under-reporting of
actual foods consumed.\(^{29}\) Measures of diet
that do not rely on subjects recalling intake in
some way entail invasive blood tests. Reliable
biological markers for all nutrients consumed
are still not available. FFQs have been shown to
give results which correlate significantly with
some blood measures including carotene and α
tocopherol.\(^{30}\)

The FFQ relies on memory for recall of usual
consumption of food items. It is probable that
subject’s memories are equally unreliable across
all groups, resulting in a dilution of any effect of
different dietary intake between groups rather
than bias in one direction or another. Recall
bias can occur in case-control studies, because
recall of diet before the disease onset will be
biased towards current dietary intake, which
may be different, having changed as a result of
the disease.\(^{12,13}\) This study aimed to limit this
bias by using subjects from the screening
programme. This gave two main advantages
over other case-control studies: subjects would
be at an early stage of disease, possibly before
any dietary changes resulting from the disease;
and the interviews took place at a time before
the subjects knew their diagnosis.

The results showed no statistically significant
trends in nutrient intake between the cases and
the control groups for fat intake. This is in
agreement with most recently published results
from cohort studies.\(^{4,15,32}\) There was a
suggestion from our study that risk may
increase with increased saturated fat intake.
The only cohort study that has shown an effect
of a high fat diet was the NHANES study, which
suggested a protective effect of total fat
and saturated fat on breast cancer.\(^{33}\) However,
the authors acknowledge methodological prob-
lems with the dietary assessment and this study
scored poorly in a review of published studies.\(^{5}\)
The same review looked at 35 case-control
studies, of these 14 presented odds ratios for
estimates of total fat intake. Most of these
studies tended to suggest an increased risk of
breast cancer with higher fat intake, though
only two were statistically significant. One
study suggested a protective effect of a higher
fat diet.\(^{9}\) Another review of 12 case-control
studies that had carried out a combined analy-
sis, showed a statistically significant positive
association between breast cancer risk and
saturated fat intake in postmenopausal
women.\(^{34}\) We believe that the principal reason
our case-control study contradicted these is
that recall bias was reduced in our study. A
case-control study carried out in Sweden also
using subjects from a mammography screening
programme did not find any increased risk with
high fat intake.\(^{35}\) Polyunsaturated fatty acid
(PUFA) intake showed a non-significant pro-
tection effect for all three case-control compari-
sions. Most case-control studies have not shown
any significant effect of PUFA on breast cancer
risk\(^{11}\) although one study in Singapore has
shown a significant protective effect of PUFA.\(^{36}\)
Cohort studies have also not shown any effect
of PUFA on breast cancer risk. Polyunsatu-
rated fat has been suggested to be a promoter
of breast cancer, but in particular it is the
n-6 series of fats, which are derived from vegetable
oils that seems to have this effect. On the other
hand, PUFA from the n-3 series derived from
fish oils may inhibit tumour development.\(^{37}\)

The nutrient intakes for our sample show
differences to dietary intake from the National
Diet and Nutrition Survey (NDNS)\(^{38}\) (table 1).
NDNS used a seven day weighed intake to
assess dietary intake, recording actual rather

**KEY POINTS**
- This study design aimed to reduce recall bias associated with dietary recall in case-control studies.
- No statistically significant trends in fat intake were found between cases and controls.
- Iron intake was consistently protective across all three control groups compared with cases.
than usual intake. In the NDNS, median total energy intake for women aged 50–64 years was 1620 kcal, this result was similar to our survey. It is known that weighed intakes lead to under-reporting and the NDNS found that 47% of women surveyed had an energy intake to basal metabolic rate ratio of less than 1.2. Habitual intakes of this order are unlikely to meet requirements, suggesting that under-reporting in the national study had occurred. In our study about 55% of the women had energy intake to basal metabolic rate ratios of less than 1.2. However, our study had higher intakes of protein, CHO, sugars, fibre, vitamin C, carotenoids, calcium, iron, and zinc than the NDNS suggesting that under-reporting for all nutrients was not a problem. Reported fat intakes were lower in our study. In particular saturated fat intake was lower in our study. It is possible that the FFQ used here was underestimating fat, particularly saturated fat intake. As fat was the focus of the study, however, it would be more likely that the FFQ would overestimate intakes. It is also possible that the women in our study were eating a lower fat diet than was found by the NDNS. The fieldwork for the NDNS was carried out in 1986–87. Since then health promotion has been emphasising reducing fat in the diet and more low fat products are available. There is some evidence that absolute fat intakes have been decreasing over the past decade. Further work is needed to compare the food sources of nutrients in diets from our sample with NDNS results.

Vitamin C intakes were particularly high in our study. This may reflect the comparatively affluent nature of the population being studied because vitamin C intake and social class are positively correlated. Fibre intakes were also high. The subjects in our study may have been health conscious and eaten a healthier diet than the majority of the population. They had already indicated their interest in health issues by attending the screening clinic.

Iron intake showed a consistently protective effect across all three control groups when compared with cases. Only two epidemiological studies have reported iron intake in relation to breast cancer, although neither of these found statistically significant associations. Iron intakes in our study were 2–3 mg higher on average than those found for women of similar age in the NDNS. Iron in the diet comes mainly from cereals, meat, and vegetables. In an ecological study there was a highly negative correlation of cereal intake with breast cancer mortality in England and Wales over a 50 year period. Epidemiological studies have shown that people with high body stores of iron are at increased risk for cancer and animal studies have shown that tumour cells need iron to grow. The results of our study do not support these findings. This study only measured dietary intake of iron from food, however, and took no account of iron supplement use. Supplementary iron has been shown to provide 15% of women’s average intake from all sources. There was a suggestion from these data that vitamin E intake increased odds of breast cancer, although the test for trend was not significant. Vitamin E is generally thought to have a protective effect. It functions as an antioxidant, particularly in cell membranes where it plays a part in protecting the body from lipid-peroxide generated damage; it also inhibits the formation of carcinogenic nitrosamines and nitrosamides. A review of five case-control studies that looked at dietary intake of vitamin E and breast cancer found that three studies showed a protective effect for vitamin E while the other two did not.

In line with recent cohort studies, this study has shown no evidence to support the hypothesis that dietary fat is an important contributor to breast cancer rates. It is unlikely that dietary fat intake has an important influence on breast cancer risk, unless this influence occurs much earlier in life.

The authors would like to thank Pauline Herd, Cynmöne Imms, and Christine Candy for fieldwork and Kirsten Maters for data entry.

Funding: the research was supported by: Wessex Cancer Trust, Wessex Medical School Trust, Wessex Regional Health Authority, Parke-Davis Research Laboratories.

Conflicts of interest: none.