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Nutritional Factors and Cancers of the Breast, Endometrium and Ovary

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Abstract—*From an overview of epidemiological evidence on nutrition, diet and cancers of the breast, endometrium and ovary, the following indications can be drawn:*

— *Overweight and obesity are causally related to endometrial and post-menopausal breast cancer, and may account for as much as one third of the cases of endometrial and one tenth of breast cancer in Europe. It is not known whether obesity or overweight early in life has any role on breast cancer risk, nor whether obesity influences ovarian carcinogenesis. Overweight tends to be associated with an unfavourable prognosis for breast cancer.*

— *Despite extensive research, the available knowledge on diet and breast cancer is largely inconsistent, and the results from ecological and individual-based studies are contradictory in relation to fat, proteins, total energy, alcohol, etc.*

— *There are only scanty data on diet and endometrial or ovarian cancer, which tend to suggest role for fat (or animal fat) in the risk of these neoplasms.*

— *The evidence on diet and breast, ovarian and endometrial carcinogenesis is still too scanty or inconsistent to be of any practical preventive value. Thus, the only clear indication for prevention is that a reduction of overweight would avoid a substantial number of cases of endometrial and post-menopausal breast cancer.*

THERE ARE important reasons to support a role for nutrition in breast, ovarian and endometrial carcinogenesis. First, studies on rodents dating back to 1940 demonstrated an effect of dietary deficiencies and excesses on breast cancer incidence [1]. Secondly, international differences in incidence and mortality from these neoplasms are substantial [2], and can only partly be explained in terms of recognized risk factors for these neoplasms, i.e. reproductive and hormonal factors. Further, it is known that nutrition and diet are related to sex hormones and their binding proteins, and this provides a plausible biological explanation for an association between diet and female-hormone related neoplasms [3–5].

Despite extensive research, however, the relation between nutrition, diet and cancers of the breast and female genital tract is only partly understood. This article will therefore review some of the open questions and discuss possible lines for further research.

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MEASURES OF WEIGHT AND HEIGHT

Obesity is a recognized risk factor for endometrial cancer [6, 7]. Since the association is observed both in pre- and post-menopausal women, it is likely that the underlying biological mechanism is different at various ages, i.e. that increased aromatization of androgens to oestrogens and reduced sex-hormone binding globulin are the relevant factors post-menopause, while the influence of obesity on ovulation is important pre-menopause [8]. Whatever the biological mechanism(s), overweight may explain one fourth of cases of the disease in North America [9] and as many as one third in Europe, where oestrogen replacement treatment (i.e. the other major recognized cause of the disease) is less widespread.

At least two studies of endometrial cancer, moreover, have found that not only body weight, but also height [10, 11] is directly associated with risk. This is consistent with findings on other sex-hormone related neoplasms (breast [12, 13] and prostate [14]) and, in aetiological terms, could be related to a potential role of nutritional factors in childhood and adolescence on the subsequent risk of these neoplasms.

Overweight in post-, but not in pre-menopause, is related to the risk of breast cancer too, although the

association is approximately an order of magnitude less strong than for endometrial cancer (i.e. the relative risk is for obese women about 1.5–1.8 rather than 5–8, Table 1) [8–12, 15, 16].

One important open question is whether body weight early in life influences the later risk of breast and endometrial cancer. There are some indications that this may be the case, from studies on migrants and cohorts of young athletes [17] or ballet dancers [18], but the American Nurses Prospective Study [19] and a cohort investigation of Hawaiian women [20] on whom information on weight and height was directly available showed an *inverse* association between adolescent body mass and pre-menopausal breast cancer (but no relation with early age weight and height). The question has important theoretical implications in order to understand the role of overweight (and its biological correlates) in relation to the process of breast carcinogenesis, i.e. whether it has essentially a late stage effect (as that of exogenous oestrogens in endometrial cancer) or an early stage effect, too [21].

Another unsettled issue is whether obesity has any influence on the risk of epithelial ovarian cancer. The American Cancer Society One Million Study [7] suggested that this may be the case, since the point estimate was 1.6 in severely obese women (Table 2), but the evidence from case-control studies [22–25] is largely negative, possibly on account of loss of weight induced by the neoplastic process. Further, any aetio-pathological inference is hampered by the unsettled state of knowledge on the role of steroid hormones (or their correlates) on ovarian carcinogenesis.

In relation to height, the original prospective study by de Waard and Baanders-van Halewijn [12] showed an independent effect of this factor on post-menopausal breast cancer: the relative risk rose to 3.6 for the tallest/heaviest women compared to the lowest height/weight category. Subsequent studies [13, 18, 19] produced inconsistent results, but, at least from an aetiological viewpoint, it is clearly important that the two factors (height and weight) are examined separately as well as in terms of combined indices [21].

DIET AND BREAST CANCER

Much of the initial evidence linking breast cancer risk to diet came from correlational studies. On an international scale, strong direct correlations were observed with total calories, fats, protein, as well as with selected foods such as milk, eggs, or meat [26, 27]. Several correlations were also observed on a national level, and persisted after allowance for reproductive variables. Table 3 gives, as an example, the crude and partial (after allowance for parity) correlation coefficients between selected food items and breast cancer risk in various regions of Italy, a country with marked differences in dietary habits and cancer rates [28].

Unfortunately, evidence from analytical studies is much less convincing. Case-control studies have reported associations with total energy, fats, proteins, fried foods, dairy products, beef and other red meats, pork and sweet desserts [23, 29–36]. The associations observed, however, were weak and inconsistent in various studies (see Table 4 for a summary review of published data on estimates of

*Table 1. Relative risk of endometrial and breast cancer according to body mass index**

Body mass index (kg/m^2)	Endometrial cancer (283 cases, 566 controls)		Breast cancer (2884 cases, 2907 controls)	
	Pre-menopause	Post-menopause	Pre-menopause	Post-menopause
<20	1†	1†	1†	1†
20–24	1.5	1.6	1.3	1.2
25–29	3.9	3.3	1.0	1.3
≥30	20.3	7.6	1.0	1.6

*Data from Italian studies (La Vecchia *et al.* [8]; Negri *et al.* [16]).
†Reference category.

*Table 2. Mortality ratios for selected sites of cancer in females**

Site	Weight index						
	<80	80–89	90–109	110–119	120–129	130–139	≥140
Breast	82	86	100	119	116	122	153
Endometrium	89	104	100	136	185	230†	542
Ovary	86	98	100	115	99	88	163

*Derived from the American Cancer Society One Million Study (Lew and Garfinkel [7]).
†Estimates based on less than five deaths.

*Table 3. Correlation between breast cancer mortality and selected dietary variables in various Italian regions**

Food items	Correlation coefficients	
	Crude	Adjusted for age at first birth
Milk	0.81	0.52
Cheese	0.74	0.52
Meat	0.39	−0.25
Sugar	0.66	0.15
Wine	0.37	−0.45
Pasta	−0.78	−0.31

*From La Vecchia and Pampallona [28].

fat intake) [23, 29–36]. The most plausible conclusion from case-control studies is that there is indeed some association between fats and breast cancer risk (and probably for total calories as well), which however is limited (and hence extremely difficult to prove in epidemiological terms) in studies that collect information on individuals.

To further complicate the issue, the prospective American Nurses Health Study [37] reported no evidence of any association with total fat, saturated fat, linoleic acid or cholesterol, although the cut-off points for extreme quintiles of total fat were 32% and 44% of total energy intake, conceivably too narrow an interval to permit any biological effect to be detected.

Paradoxically, stronger analytical epidemiological evidence has emerged in relation to two dietary items for which no convincing biological interpretation is available: green vegetables and alcohol. A strong independent protection has been reported for green/salad-type vegetables from two studies in Mediterranean countries [32, 33], with relative risks between 0.2 and 0.4 for the highest level of consumption, which were not explained by allowance for the major identified breast cancer risk factors or other dietary items. Preliminary data from a case-control study conducted in Argentina tended to confirm this protection [38].

In relation to alcohol, most of the over 20 studies published to date found significantly elevated risks among drinkers. Only six out of 19 studies conducted in a formal meta-analysis of the topic published in 1987 [39] did not find a positive connection. As an example, in Table 5 the updated relative risks from a large case-control study conducted in Northern Italy [32] are presented. In this population, where wine drinking by women is widespread (and socially accepted), the relative risk rose up to threefold among drinkers of four or more drinks per day. In some of the studies (including the American Nurses cohort) [40] the relative risk was elevated even among extremely light drinkers, and only part of the studies found a dose–risk relationship. This indicates that the association, if real, may not be causal, and possibly reflects a number of as yet undefined correlates of alcohol drinking.

Nonetheless, alcohol must at present be considered the sole single item for which consistent evidence of an association has emerged from analytical epidemiological studies. Ironically, and further confirming the currently undefined state of knowledge on diet and breast cancer, correlational studies on populations provide no support for this association [41] (see Table 3 as an example), although it is not known how consumption in women is reflected in overall alcohol disappearance statistics.

IMPLICATIONS OF NUTRITION AND DIET IN BREAST CANCER PROGNOSIS

The observation that survival of breast cancer is higher in low incidence areas, such as in Japan compared with the U.S.A., has led to the suggestion that the same factors that are related to breast cancer development may interfere with its prognosis [42–44].

Direct examinations of breast cancer survival in clinical series indicated that obese women had lower survival rates than non-obese [45] and, after multi-

Table 4. Association between fat intake and breast cancer risk in selected case-control studies*

Study, year	Fat, level of intake				
	1	2	3	4	5
Phillips, 1975 [29]	1†	÷2	—	—	—
Miller *et al.*, 1978 [30]	1†	1.7	1.2	1.8	—
Lubin *et al.*, 1981 [31]	1†	1.6	1.5	1.8	—
Graham *et al.*, 1982 [23]	1†	1.1	1.2	0.9	—
La Vecchia *et al.*, 1987 [32]	1†	1.3	1.3	—	—
Katsouyanni *et al.*, 1988‡ [34]	1†	1.4	—	—	
Rohan *et al.*, 1988 [35]	1†	0.9	1.1	1.1	0.9

*Total fat, whenever available.
†Reference category.
‡90th vs. 10th centile.

Table 5. Distribution of 1926 breast cancer cases and 1866 controls according to total alcoholic beverage consumption. Milan, Italy, 1983–1987

Average total alcohol consumption (drinks per day)	Breast cancer	Controls	Relative risk (95% CI)*
0	513	668	1†
<2	565	552	1.35 (1.25–1.60)
2 < 3	601	512	1.53 (1.30–1.81)
3 < 4	128	83	2.01 (1.50–2.70)
≥4	119	51	3.02 (2.15–4.23)
χ^2_1			60.29 ($P < 0.001$)

*Mantel–Haenszel estimates adjusted for age.
†Reference category.

variate analysis, that body weight was the only risk factor associated with prognosis [46, 47]. For instance, follow-up of breast cancer cases participating in a case-control study in Canada indicated that body weight, but not dietary fat intake, had an unfavourable impact on survival [48].

Although there is evidence that these influences are independent, and not explained through an association with advanced previous disease [49], there are, nonetheless, problems in the definition of stage, lymph node involvement and other prognostic factors, and of competing cause(s) of death in obese as compared with non-obese women, which make any comparison difficult.

In relation to diet, a study based on 666 women with newly diagnosed infiltrating breast cancer suggested that saturated fat intake was related to unfavourable prognostic indicators, i.e. a higher frequency of node involvement at diagnosis [50]. Likewise, breast cancer survival was more favourable among 282 Seventh-day Adventists compared with other white females: the difference, however, was no longer evident when stage at diagnosis was allowed for in the analysis [51].

DIET AND ENDOMETRIAL CANCER

Strong positive correlation coefficients were observed between meat, eggs, milk, fats and oils, total protein fats and calorie intake and endometrial cancer incidence [27]. The issue is clearly complicated by the fact that proteins and fats are correlated with total energy intake which, in turn, is the major determinant of obesity, i.e. a consistently recognized risk factor for endometrial cancer.

Few analytical studies have directly discussed the role of diet in the aetiology of endometrial cancer. Preliminary results from a case-control study based on 24-h dietary recall found that the carbohydrate and total caloric (but not protein or fat) intake was higher in cases than in controls [52].

A case-control study from Italy based on 206 age-matched pairs [53] reported strong direct associations with subjective intake scores for fats and oils, and significant protection by green vegetables and fresh fruit (Table 6). However, information on only a small number of food items was collected in that study, and it was not possible to estimate total calorie intake.

There is, therefore, ample scope for further research on the question of diet and endometrial cancer, particularly in consideration of the fact that, although the risk factors for cancer of the corpus uteri are better defined than for breast or ovarian neoplasms, they cannot by themselves explain the 30-fold difference in incidence observed between various cancer registration areas [2].

*Table 6. Diet and endometrial cancer: major findings from a case-control study in Northern Italy**

Food item	Multivariate relative risk (95% CI)	
Total fat (subjective score)		
1 (low)	1†	
2 (intermediate)	3.0	(1.7–5.1)
3 (high)	5.0	(2.9–8.4)
Green vegetables (frequency)		
1 (low)	1†	
2 (high)	0.2	(0.1–0.5)
Fresh fruit (frequency)		
1 (low)	1†	
2 (high)	0.6	(0.3–1.0)

*Data derived from La Vecchia *et al.* [53].
†Reference category.

Further, the well-known association between levels of oestrogens and endometrial cancer risk may help identify low-risk dietary patterns: for instance, it has been shown that the levels of oestriol, total oestrogens and prolactin were lower and those of sex-hormone binding globulin higher in post-menopausal vegetarian women, and the differences were not explainable in terms of different body weight [52, 54].

DIET AND OVARIAN CANCER

As for endometrial cancer, most of the links between ovarian cancer and diet are indirect, and based on international differences or correlational studies. Correlations with fats, proteins and calories are in the same direction as those of endometrial cancer [55], although somewhat less strong in relation to incidence [27].

Information from follow-up studies in selected populations is scanty and contradictory: Seventh-day Adventists, many of whom are lacto-vegetarians, showed lower ovarian cancer mortality (SMR = 0.6) than the population of California [56], but Mormons in Utah had a standardized incidence ratio of 1.7 in relation to the general U.S. population [57]. In a cohort of British nuns with low intake of meat and fats, no reduction in risk was apparent [58].

The results of case-control studies are somewhat more consistent. Among four studies [22, 24, 25, 59] which considered various measures of fat intake (Table 7), three [24, 25, 59] found significant direct associations. In one of these [25], based on a Chinese population with wide ranges of consumption of various nutrients and which could provide estimates of intake of several nutrients, the effect of fat persisted after adjustment for total calories whereas, after allowance for animal fat intake, no significant association persisted with calories or proteins.

Along this line, a follow-up study of a cohort of Seventh-day Adventists reported that women who more frequently ate eggs or fried foods had an approximately three-fold elevated risk of fatal ovarian cancer [60], and a similar risk emerged for daily meat consumption from a case-control study of 56 cases aged over 50 conducted in Japan [61].

Studies in Italy [24] and North America [59], found reduced risk with more frequent consumption of green vegetables, carrots and greater vitamin A intake. Vitamin C and total vegetables were somewhat protective in the Chinese study as well [25], although the mechanism of action of these nutrients is far from being understood.

CONCLUSIONS

Nutrition and diet are certainly among the priorities for epidemiological research on breast, endometrial and ovarian cancer, not only from an aetiological viewpoint, but also from a preventive one. In fact, in contrast with reproductive factors, action on these factors is conceivable.

However, at present, the only practical preventive measure is avoiding obesity in order to reduce endometrial and post-menopausal breast cancer risk; this could lower the overall endometrial cancer incidence by one third, and that of post-menopausal breast cancer by one tenth in European countries.

In relation to diet, despite extensive research on breast cancer, available knowledge is largely inconsistent, with largely contradictory results from ecological and individual-based studies. Hence, no sound conclusion of practical preventive relevance can be drawn.

There are only scanty data on diet and endometrial or ovarian cancer, which suggest a role of fat (or animal fat) on the risk of these neoplasms. Needless to repeat, however, further research is needed before any indications of practical preventive value can be drawn.

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Table 7. Fats and ovarian cancer: summary results from case-control studies

Study	Level of fat intake 1	2	3	4
Byers *et al.*, 1983 [22]	1*	1.3	1.2	—
Cramer *et al.*, 1984 [59]	1*	1.4	1.9	1.8
La Vecchia *et al.*, 1987 [24]	1*	1.2	2.1	—
Shu *et al.*, 1989 [25]	1*	1.1	1.8	1.9

*Reference category.

REFERENCES

1. Tannenbaum A. Relationship of body weight to cancer incidence. *Arch Pathol* 1940, **30**, 509–517.
2. Muir C, Waterhouse J, Mack T, Powell J, Whelan S, eds. *Cancer in Five Continents*. Lyon, IARC, 1987 (IARC Scientific Publication 88), Vol. 7.
3. Siiteri PK, Murai JT, Hammond GL, Nisker JA, Raymoure WJ, Kuhn RW. The serum transport of steroid hormones. *Rec Prog Horm Res* 1982, **38**, 457–510.
4. Ingram DM, Bennett FC, Willcox D, de Klerk N. Effect of low-fat diet on female sex hormone levels. *JNCI* 1987, **79**, 1225–1229.
5. Fentiman IS, Caleffi DY, Wang SJ *et al.* The binding of blood-borne estrogens in normal vegetarian and omnivorous women and the risk of breast cancer. *Nutr Cancer* 1988, **11**, 101–106.
6. Elwood JM, Cole P, Rothman KJ, Kaplan SD. Epidemiology of endometrial cancer. *JNCI* 1977, **59**, 1055–1060.
7. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. *J Chronic Dis* 1979, **32**, 563–576.
8. La Vecchia C, Franceschi S, Decarli A, Gallus G, Tognoni G. Risk factors for endometrial cancer at different ages. *JNCI* 1984, **73**, 667–671.
9. McDonald TW, Annegers JF, O'Fallon WM *et al.* Exogenous estrogen and endometrial carcinoma: case-control and incidence study. *Am J Obstet Gynecol* 1977, **127**, 572–580.
10. Davis J, Rosenheim NB, Antunes C, Stolley P. A review of the risk factors for endometrial carcinoma. *Obstet Gynecol* 1981, **36**, 107–116.
11. Wynder E, Escher G, Mantel N. An epidemiological investigation of cancer of the endometrium. *Cancer* 1966, **19**, 489–520.
12. De Waard F, Baanders-van Halewijn EA. A prospective study in general practice on breast cancer risk in postmenopausal women. *Int J Cancer* 1974, **14**, 153–160.
13. Swanson CA, Jones DY, Schatzkin A, Brinton LA, Ziegler RG. Breast cancer risk assessed by anthropometry in the NHANES I. Epidemiological follow-up study. *Cancer Res* 1988, **48**, 5363–5367.
14. Severson RK, Grove JS, Nomura AMY, Stemmermann OR. Body mass and prostatic cancer: a prospective study. *Br Med J* 1988, **297**, 713–715.
15. Boyle P. Epidemiology of breast cancer. *Baillière Clin Oncol* 1988, **2**, 1–59.
16. Negri E, La Vecchia C, Bruzzi P *et al.* Risk factors for breast cancer: pooled results from three Italian case-control studies. *Am J Epidemiol* 1988, **128**, 1207–1215.
17. Frisch RE, Wyshak G, Albright NL *et al.* Lower lifetime occurrence of breast cancer and cancer of the reproductive system among former college athletes. *Am J Clin Nutr* 1987, **45**, 328–335.
18. Warren MP. The effects of exercise on pubertal progression and reproduction on girls. *J Clin Endocr Metab* 1980, **51**, 1150–1157.
19. Willett WC, Browne ML, Bain C *et al.* Relative weight and risk of breast cancer among premenopausal women. *Am J Epidemiol* 1985, **122**, 731–740.
20. Le Marchand L, Kolonel LM, Earle ME, Ming-Pi Mi. Body size at different periods of life and breast cancer risk. *Am J Epidemiol* 1988, **128**, 137–152.
21. De Waard F, Trichopoulos D. A unifying concept of the aetiology of breast cancer. *Int J Cancer* 1988, **41**, 666–669.
22. Byers T, Marshall J, Graham S, Mettlin C, Swanson M. A case-control study of dietary and nondietary factors in ovarian cancer. *JNCI* 1983, **71**, 681–686.
23. Graham S, Marshall J, Mettlin C *et al.* Diet in the epidemiology of breast cancer. *Am J Epidemiol* 1982, **116**, 68–75.
24. La Vecchia C, Decarli A, Negri E *et al.* Dietary factors and the risk of epithelial ovarian cancer. *JNCI* 1987, **79**, 663–669.
25. Shu XO, Gao YT, Yuan JM, Ziegler RG, Brinton LA. Dietary factors and epithelial ovarian cancer. *Br J Cancer* 1989, **59**, 92–96.
26. Lea AJ, Birm MB. Dietary factors associated with death rates from certain neoplasms in man. *Lancet* 1966, **ii**, 332–333.
27. Armstrong BK, Doll R. Environmental factors and cancer incidence and mortality in different countries, with special reference to dietary practice. *Int J Cancer* 1975, **15**, 617–631.
28. La Vecchia C, Pampallona S. Age at first birth, dietary practices and breast cancer mortality in various Italian regions. *Oncology* 1986, **43**, 1–6.
29. Phillips RL. Role of life-style and dietary habits in risk of cancer among Seventh-day Adventists. *Cancer Res* 1975, **35**, 3513–3522.
30. Miller AB, Kelly A, Choi NW *et al.* A study of diet and breast cancer. *Am J Epidemiol* 1978, **107**, 499–509.
31. Lubin JH, Burns PE, Blot WJ, Ziegler RG, Lees AW, Fraumeni JF Jr. Dietary factors and breast cancer risk. *Int J Cancer* 1981, **28**, 685–689.
32. La Vecchia C, Decarli A, Franceschi S *et al.* Dietary factors and the risk of breast cancer. *Nutr Cancer* 1987, **10**, 205–214.
33. Katsouyanni K, Trichopoulos D, Boyle P *et al.* Diet and breast cancer: a case-control study in Greece. *Int J Cancer* 1986, **38**, 815–820.

34. Katsouyanni K, Willet WC, Trichopoulos D *et al.* Risk of breast cancer among Greek women in relation to nutrient intake. *Cancer* 1988, **61**, 181–185.
35. Rohan TE, McMichael AJ, Baghurst PA. A population-based case-control study of diet and breast cancer in Australia. *Am J Epidemiol* 1988, **128**, 478–489.
36. Howe GR. The use of polytomous dual response data to increase power in case-control studies: an application to the association between dietary fat and breast cancer. *J Chron Dis* 1985, **38**, 663–670.
37. Willett WC, Stampfer MJ, Colditz GA, Rosner BA, Hennekens CH, Speizer FE. Dietary fat and the risk of breast cancer. *N Engl J Med* 1987, **316**, 22–28.
38. Kaldor J, Shiboski S, Arslan A. Breast cancer in Argentina. In: *IARC Biennial Report 1988.* Lyon, IARC, 1988, 120.
39. Longnecker MP, Berlin JA, Orza MJ, Chalmers TC. A meta-analysis of alcohol consumption in relation to risk of breast cancer. *JAMA* 1988, **260**, 652–656.
40. Willett WC, Stampfer MJ, Colditz GA, Rosner BA, Hennekens CH, Speizer FE. Moderate alcohol consumption and risk of breast cancer. *N Engl J Med* 1987, **316**, 1174–1180.
41. La Vecchia C, Franceschi S, Cuzick J. Alcohol and breast cancer. *Lancet* 1982, **i**, 621.
42. Wynder EL, Kajitani T, Kuno J, Lucas JC, DePalo A, Farrow J. A comparison of survival rates between American and Japanese patients with breast cancer. *Surg Gynec Obstet* 1963, **117**, 196–200.
43. Morrison AS, Black MM, Lowe CR, MacMahon B, Yuasa S. Some international differences in histology and survival in breast cancer. *Int J Cancer* 1973, **11**, 261–267.
44. Morrison AS, Lowe CR, MacMahon B, Ravnihar B, Yuasa S. Some international differences in treatment and survival in breast cancer. *Int J Cancer* 1976, **18**, 269–273.
45. Abe R, Kumagai N, Kimagai M. Biological characteristics of breast cancer in obesity. *Tohoku J Exp Med* 1976, **120**, 351–359.
46. Boyd NE, Campbell JE, Germanson T, Thomson DB, Sutherland DJ, Meakin JW. Body weight and prognosis on breast cancer. *JNCI* 1981, **67**, 785–789.
47. Hebert JR, Augustine A, Barone J *et al.* Weight, height and body mass index in the prognosis of breast cancer: early results of a prospective study. *Int J Cancer* 1988, **42**, 315–318.
48. Newmann SC, Miller AB, Howe GR. A study of the effect of weight and dietary fat on breast cancer survival time. *Am J Epidemiol* 1986, **123**, 767–774.
49. Howson CP, Kinne D, Wynder EL. Body weight, serum cholesterol, and stage of primary breast cancer. *Cancer* 1986, **58**, 2372–2381.
50. Verrault R, Brisson J, Deschênes L, Naud F, Meyer F, Bèlanger L. Dietary fat in relation to prognostic indicators in breast cancer. *JNCI* 1988, **80**, 819–821.
51. Zollinger TW, Phillips RL, Muzma JW. Breast cancer survival rates among Seventh-day Adventists and non-Seventh-day Adventists. *Am J Epidemiol* 1984, **119**, 503–509.
52. Armstrong BK. Diet and hormones in the epidemiology of breast and endometrial cancer. *Nutr Cancer* 1979, **1**, 90–95.
53. La Vecchia C, Decarli A, Fasoli M, Gentile A. Nutrition and diet in the etiology of endometrial cancer. *Cancer* 1986, **57**, 1248–1253.
54. Armstrong BK, Brown JB, Clarke HT *et al.* Diet and reproductive hormones: a study of vegetarian and nonvegetarian postmenopausal women. *JNCI* 1981, **67**, 761–767.
55. Rose DP, Boyar AP, Wynder EL. International comparisons of mortality rates for cancer of the breast, ovary, prostate and colon and per capita food consumption. *Cancer* 1986, **58**, 2363–2371.
56. Phillips RL, Garfinkel L, Kuzma JW, Beeson WL, Lotz T, Brin B. Mortality among California Seventh-day Adventists for selected cancer sites. *JNCI* 1980, **65**, 1097–1107.
57. Lyon JL, Gardner JW, West DW. Cancer risk and life-style: cancer among Mormons from 1967–1975. In: Cairns A, Lyon FL, Stolnick M, eds. *Cancer Incidence in Defined Populations.* Cold Spring Harbor, Cold Spring Harbor Laboratory, 1980, 93–98 (Banbury Report No. 4).
58. Kinlen LJ. Meat and fat consumption and cancer mortality: a study of strict religious orders in Britain. *Lancet* 1982, **i**, 946–949.
59. Cramer DW, Welch WR, Hutchinson GB, Willett WC, Scully RE. Dietary animal fat in relation to ovarian cancer risk. *Obstet Gynecol* 1984, **83**, 833–838.
60. Snowdon DA. Diet and ovarian cancer. *JAMA* 1985, **254**, 356–357.
61. Mori M, Miyake H. Dietary and other risk factors of ovarian cancer among elderly women. *Jpn J Cancer Res (Gann)* 1988, **79**, 997–1004.