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UNIVERSITY OF ALBERTA

TASTE PERCEPTION, DIET AND CANCER:  
COMPARISON OF WOMEN WITH AND WITHOUT BREAST CANCER

BY

HOLLY G. AMES

A thesis submitted to the Faculty of Graduate Studies and Research in  
partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

IN

FOODS AND NUTRITION

DEPARTMENT OF FOODS AND NUTRITION

EDMONTON, ALBERTA

SPRING 1992



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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "Taste perception, diet and cancer: comparison of women with and without breast cancer" submitted by Holly G. Ames in partial fulfillment of the requirements for the degree of Master of Science in Foods and Nutrition.

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## ABSTRACT

Suprathreshold taste perception and nutrient intake were assessed for two groups of women aged 44-56 years: 24 mastectomized breast cancer outpatients and 24 matched controls. Salty and sweet taste intensity and pleasantness in aqueous and food systems were evaluated by unstructured category line scaling. Dietary intakes were assessed quantitatively by combined dietary recall (one day) and food record (three days) methods.

Suprathreshold taste intensity and pleasantness of salt and sucrose in aqueous and food systems did not differ between the cancer and control groups. The slopes for saltiness intensity for the cancer and control groups, respectively were: 10.3 and 9.8 for the aqueous system; 8.5 and 8.3, respectively for food. For sweetness, the slopes for sucrose intensity for the cancer and control groups, respectively were: 9.9 and 9.0 for the aqueous system; 6.0 and 5.0, respectively for food. For both groups, slopes for saltiness ( $p \leq 0.01$ ) and sweetness ( $p \leq 0.001$ ) intensity were flatter in food than in the aqueous system. For both groups, pleasantness responses for saltiness and sweetness significantly differed between the aqueous and food systems.

The cancer group consumed less ( $p \leq 0.05$ ) energy (1501 kcal) than the control group (1763 kcal). The index of overall nutritional risk for the cancer group (14.6%) was higher ( $p \leq 0.05$ ) than that for the control group (7.0%). The nutrients at greatest risk of deficiency for the cancer group were: calcium, folacin, zinc, vitamin B12, vitamin A, ascorbic acid and iron. Compared to the control group, the cancer group was at greater risk of calcium ( $p \leq 0.01$ ) and iron ( $p \leq 0.05$ ) deficiency.

For a breast cancer subgroup ( $n=7$ ) with low energy intake ( $\leq 1300$  kcal) and high overall nutritional risk (25.6%), significant relationships

between taste perception and diet were found although taste data did not differ from that of the controls. Vitamin B12 and folacin intake; percent risk of vitamin B12, thiamin, folacin, iron and riboflavin deficiency; and iron, ascorbic acid, folacin and phosphorus density were important predictors of the variance in taste intensity slopes for the cancer subgroup. Findings suggest that for some breast cancer patients, suprathreshold taste intensity data may be useful to indicate nutritional problems.

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## 1 INTRODUCTION

Cancer of the breast is the most commonly diagnosed cancer and is the leading cause of cancer mortality for Canadian women (National Cancer Institute, 1990). It is estimated that one in 10 Canadian women will develop breast cancer during their lifetime (National Cancer Institute, 1990). The magnitude of the breast cancer incidence and mortality rates in Canada suggests that research concerning the nutritional health of women with breast cancer would be prudent.

Nutrition influences both the health and the quality of life of cancer patients. However, nutritional information regarding cancer patients is limited. Available dietary data for cancer patients relates predominately to the pre- rather than the post-diagnosis state. Taste perception plays a critical role in the nutritional health of cancer patients. Many cancer patients experience alterations or diminutions of taste. Taste disturbances may alter food selection by the cancer patient such that the nutritional status of the individual is compromised. Studies that have examined taste perception and nutrition in cancer patients are lacking. To ensure that cancer patients consume nutritionally adequate diets, investigations of the relationships between taste perception and diet in cancer patients must be conducted.

The objectives of the present study were to assess suprathreshold taste perception and dietary intake in breast cancer outpatients and control women in order to:

1. compare suprathreshold taste perception between women with and without breast cancer.

2. compare suprathreshold taste perception between aqueous and food systems.
3. compare dietary intake data between women with and without breast cancer.
4. examine relationships between suprathreshold taste perception and dietary intake data for women with and without breast cancer.

## 2 LITERATURE REVIEW

### Breast Cancer

The etiology of breast cancer is multifactorial and not well understood (Boyle and Leake, 1988). It is generally accepted that breast cancer is a hormone-mediated disease (Howe et al., 1990) and that there is an inherited predisposition to breast cancer (Skolnick et al., 1990). However, international comparisons and migrational studies of breast cancer incidence and mortality (Trichopoulos et al., 1984; Rose et al., 1986; Miller, 1986) suggest that breast cancer risk is also influenced by environmental factors such as diet.

Strong epidemiological associations between breast cancer incidence and/or mortality rates and per capita intake of dietary fat have been observed (Rose et al., 1986; Goodwin and Boyd, 1987; Boyle and Leake, 1988). However, direct study of breast cancer cases has yielded conflicting results. In case-control studies, positive associations between breast cancer risk and intake of high fat foods such as dairy products and fried foods (Phillips, 1975), red meat, pork and sweet desserts (Lubin et al., 1981) and the intake of meat fat (Hislop et al., 1988) have been found. Yet, only weak (Miller et al., 1978; Knekt et al., 1990) or no (Graham et al., 1982; Katsouyanni et al., 1986; Willet et al., 1987a; Hirohata et al., 1987; Rohan et al., 1988; Iscovich et al., 1989; Zaridze et al., 1991) associations between breast cancer risk and total dietary fat intake have been documented in case-control reports. In contrast, an evaluation of the combined data from 12 case-control studies by Howe et al. (1990) revealed a significant dose-responsive relationship

between dietary fat intake and breast cancer risk, but for postmenopausal women only.

Dietary fat intake is very strongly correlated with energy intake (Mettlin, 1986). Positive correlations between breast cancer risk and total energy intake have been found by some researchers (Iscovich et al., 1989; Howe et al., 1990) but not by others (Miller et al., 1978; Willet et al., 1987a). In a prospective study, Knekt et al. (1990) observed a significant negative correlation between energy intake and breast cancer risk. However, obesity (from chronic excess energy intake) is a generally recognized risk factor for breast cancer (Rose, 1986; de Waard, 1986; Miller, 1990) and has been implicated in breast cancer recurrence (Donegan et al., 1978; Tarter et al., 1981; Boyd et al., 1981; Eiberlein et al., 1985; de Waard et al., 1985; Lees et al., 1991).

Associations between micronutrients and breast cancer risk have been inconsistent. Inverse correlations have been observed between breast cancer risk and the intake of beta-carotene rich foods such as green (Iscovich et al., 1989) or salad-type (Katsouyanni et al., 1986) vegetables. Breast cancer risk has also been negatively correlated with intakes of vitamin A (Katsouyanni et al., 1988) and beta-carotene (Rohan et al., 1988; Howe et al., 1990). Conversely, compared to controls, breast cancer cases have been reported to consume more (Iscovich et al., 1989) or similar amounts (Marubini et al., 1988; Gerber et al., 1988) of vitamin A and similar amounts of beta-carotene (Marubini et al., 1988; Gerber et al., 1988). Both inverse (Zaridze et al., 1991) and no (Graham et al., 1982) associations between ascorbic acid and breast cancer risk have been reported for case-control studies. However, when data from 12

case-control studies were combined, ascorbic acid was negatively correlated to breast cancer risk (Howe et al., 1990).

Investigations of the effect of diet on breast cancer risk have recently focused on dietary fiber and alcohol intake. In case-control studies, some inverse relationships between breast cancer risk and dietary fiber intake have been found (Iscovich et al., 1989; Howe et al., 1990; Zaridze et al., 1991) or at least implicated (Phillips, 1975; Lubin et al., 1986; Katsouyanni et al., 1986; Rohan et al., 1988). Moderate alcohol consumption has been found to be a relatively consistent risk factor for breast cancer (Graham, 1987; Howe et al., 1991). However, there is little evidence of increased risk of breast cancer for drinkers vs non-drinkers (Graham, 1987; Willet et al., 1987b; Willet et al., 1989; Wynder and Harris, 1989) and a dose-response relationship between alcohol consumption and breast cancer risk has not been defined (Willet et al., 1989; Wynder and Harris, 1989).

The lack of definitive relationships between breast cancer risk and dietary factors arises partly from the interrelated nature of the human diet (Mettlin, 1986). Exposure to one nutrient is not without exposure to others. Separating the effects of one dietary constituent on breast cancer risk from those of others is extremely difficult (Mettlin, 1986; Butrum et al., 1988). For example, Lubin et al. (1986) reported that although dietary fat influenced breast cancer risk, the highest risks were observed for women with a high fat, high animal protein, low dietary fiber intake.

Inconsistent associations between dietary factors and breast cancer risk could also result from methodological issues. Epidemiological tools

(food frequency questionnaires) have been extensively used to evaluate the effect of diet on breast cancer risk. Food frequency methodology is practical for field use (Block, 1982) but lacks the precision required to detect individual variations in dietary intake because subjects cannot accurately judge the frequency or the portion sizes of foods consumed (Gibson, 1987; Dwyer, 1988). Correlation of dietary and biochemical data requires precise quantification of the individuals' usual dietary intakes (Beaton et al., 1979; Beaton et al., 1983; Gibson, 1987). Imprecise dietary data lead to false negative results by reducing the strength of correlations between dietary and biochemical parameters (Beaton et al., 1979; Beaton et al., 1983; Gibson, 1987).

Investigation of the effect of diet on breast cancer risk has received much attention in recent years. However, the bulk of available dietary data is epidemiological in nature; quantitative dietary data for breast cancer patients are lacking. Direct quantitative assessment of the diets of breast cancer patients is required to ensure that true correlations between dietary and biochemical data are revealed by statistical analysis.

### Taste Perception and Cancer

Cancer patients often report subjective changes in taste perception (DeWys and Walters, 1975; Bolze et al., 1982). Most investigations concerning taste perception in cancer patients have revealed abnormalities in taste thresholds for one or more of the four basic taste modalities: salty, sweet, sour and bitter (Trant et al., 1982). A taste threshold is the lowest concentration at which an individual can either detect

(detection threshold) or recognize the taste quality of (recognition threshold) a tastant in solution (Bartoshuk, 1978). Taste thresholds are inversely related to taste sensitivity. DeWys and Walters (1975) evaluated taste thresholds in 50 cancer patients of mixed etiology and in 23 controls. Subpopulations of cancer patients had significantly elevated sucrose and lowered bitter recognition thresholds compared to the controls (DeWys and Walters, 1975). Salty and sour recognition thresholds in the cancer patients were unaffected (DeWys and Walters, 1975). Studies by Gorshein (1977) (comparing five cancer patients of mixed etiology and five healthy controls) and Gallagher and Tweedle (1983) (50 cancer patients of mixed etiology and 50 age and sex matched controls) also showed elevated sweet and lowered bitter recognition thresholds in cancer patients, while salty and sour taste thresholds remained unchanged compared to the controls. However, Hall et al. (1980) reported lowered bitter recognition thresholds only in 30 gastro-intestinal cancer patients compared to 30 healthy controls. Sweet, salty and sour thresholds were not significantly different between the cancer and control groups (Hall et al., 1980). In contrast, Williams and Cohen (1978) determined that the sour recognition thresholds in 30 lung cancer patients were significantly lower than those of 30 age and smoking matched controls. Bitter, sweet and salty thresholds were similar for the cancer and control groups (Williams and Cohen, 1978). Carson and Gormican (1977) however, noted increased salty recognition thresholds but no difference in bitter, sweet or sour thresholds in 29 breast cancer and 19 colon cancer patients compared to 28 age and sex matched controls. All taste thresholds were elevated in seven (Henkin, 1977) and in 35 (Bolze et al., 1982) cancer

patients of mixed etiology when compared to controls. In contrast, Ovesen et al. (1991) found no significant differences in taste thresholds for 27 lung cancer patients compared to 22 weight-matched controls for any taste modality.

Threshold analysis however, documents only the lowest extreme of the perceptual range. Subjective changes in taste perception usually reflect taste deficits in the suprathreshold, not the threshold range (Schiffman, 1983b). Suprathreshold taste analysis measures perception of a range of tastant concentrations above threshold, including those normally found in foods (Schiffman, 1983b). Alterations in the ability to discriminate suprathreshold tastes can exist without changes in taste thresholds (Schiffman, 1979). Therefore, suprathreshold taste perception measurements better reflect the ordinary experience of subjects and provide more information about the nature of taste abnormalities than do threshold determinations (Bartoshuk, 1978). Measurement of suprathreshold taste perception involves direct scaling of the intensity and pleasantness of tastant concentrations ranging from threshold to very strong. Functional equations that relate perceived tastant intensity or pleasantness to measurable tastant concentration are developed from the scaling data (Bartoshuk, 1978).

To date only two publications have investigated suprathreshold taste perception in cancer patients. Trant et al. (1982) evaluated suprathreshold intensity and pleasantness for saltiness, sourness, sweetness and bitterness of food systems in 24 lung cancer and 38 upper gastro-intestinal cancer patients. No differences in taste intensity or pleasantness responses were noted between the two cancer groups (Trant et

al., 1982). Settle et al. (1979) recorded pleasantness responses to suprathreshold saltiness, sweetness, sourness and bitterness of aqueous solutions in 72 cancer patients (mixed etiology) and 22 control patients. There were no significant differences in solution preferences between the cancer and control groups for any taste modality (Settle et al., 1979).

Previous research concerning taste alterations in cancer patients has been confounded by factors that influence taste perception. Settle et al. (1979) reported that taste threshold data were dependent on cancer site, histology and stage. Compared to controls, breast cancer patients had reduced sour recognition thresholds, while lung cancer patients had increased bitter recognition thresholds (Settle et al., 1979). Squamous cell carcinoma patients had increased sour recognition thresholds while cancer patients with metastasis had elevated bitter recognition thresholds compared to controls (Settle et al., 1979). Correlations between taste threshold alterations in cancer patients and the degree of malignancy (DeWys and Walters, 1975) and the presence of active tumors (Carson and Gormican, 1977) have been documented. Treatment regime has also been shown to affect taste acuity in cancer patients. Chemotherapy has caused taste threshold abnormalities (Reyes et al., 1973; Guthrie and Way, 1974; Tomita and Osaki, 1990) and changes in ratings for suprathreshold intensity (Mulder et al., 1983) and preference (Trant et al., 1982) in cancer patients. Radiation-induced taste threshold anomalies in cancer patients have also been reported (Mossman and Henkin, 1978; Tomita and Osaki, 1990).

The common practice of using hospitalized patients as controls in studies of taste perception in cancer patients poses specific problems.

A variety of nervous, endocrine, infectious, nutritional and local diseases have been reported to affect taste (Carson and Gormican, 1976; Schiffman, 1983a). Certain medications can also cause taste aberrations (Carson and Gormican, 1976; Schiffman, 1983a). Anti-rheumatic, anti-inflammatory, anti-hypertensive, anti-depressant and anti-proliferative agents are common offenders (Schiffman, 1983a).

Gender can also influence taste performance. Elevated thresholds for men compared to women for salty (Glanville et al., 1964; Weiffenbach et al., 1982) and sour (Greger and Geissler, 1978) tastes have been reported. However, other researchers (Cooper et al., 1959; Grzegorzczuk et al., 1979; Murphy, 1979) reported no differences in any taste threshold for men compared to women. Similar discrepancies were found for suprathreshold taste perception. Several authors (Hyde and Feller, 1981; Little and Brinner, 1984; Zallen et al., 1990) noted no effect of gender on suprathreshold taste intensity or pleasantness measurements. In contrast, Enns et al. (1979) observed that women rated high concentrations of sucrose as significantly more pleasant than did men. Chauhan (1989a) found that young women preferred higher and elderly women preferred lower concentrations of salt in soup compared to similarly-aged men. In the past, the effect of gender on taste perception may have been partly explained by a difference in smoking patterns between men and women (Schiffman, 1983b). Subjective losses in taste function are common for smokers (Peterson et al., 1968). Elevated thresholds for salty (Baker et al., 1983) and bitter (Krut et al., 1961; Kaplan et al., 1965; Peterson et al., 1968) tastes have been documented. Kaplan et al. (1965) noted that the effects of smoking on salty taste thresholds were stronger for men

than for women. Conversely, Grzegorzczak et al. (1979) found that smoking had no effect on salty taste thresholds in adults. Perkins et al. (1990) reported that suprathreshold pleasantness but not intensity ratings of sweet tastes were depressed in smokers compared to nonsmokers. Ko (1988) noted that the previous smoking habits of young and elderly men significantly contributed to the variance in the slopes of suprathreshold sour taste intensity functions. In contrast, Redington (1984) found no differences in suprathreshold intensity and pleasantness responses for any taste modality attributable to smoking. Zallen et al. (1990) observed that salty intensity and pleasantness responses were similar for smokers and nonsmokers.

In general, data suggest that taste function declines with increasing age (Schiffman, 1983b). In a prospective study, Harris and Kalmus (1949) and later Kalmus and Trotter (1962) found that bitter taste thresholds increased with age. Elevated thresholds for one or more of the four taste modalities for elderly compared to young adults have been reported by the majority of authors (Grzegorzczak et al., 1979; Hyde et al., 1981; Moore et al., 1982; Bartoshuk et al., 1986). Significantly flatter slopes for suprathreshold taste intensity functions for elderly as opposed to young adults have been observed (Coward, 1981; Weiffenbach et al., 1986; Bartoshuk et al., 1986; Gee et al., 1988; Ko, 1988). Chauhan (1989b) evaluated the intensity and pleasantness responses of three groups of adults (aged 20-29 years, 70-79 years and 80-99 years) to suprathreshold salty and sour tastes in aqueous and food systems. Multiple regression analysis revealed that gender, age and smoking significantly contributed to the slopes of the intensity functions for

salty and sour tastes (Chauhan, 1989b). Age also significantly influenced salty and sour taste pleasantness responses (Chauhan and Hawrysh, 1988; Chauhan, 1989a). Enns et al. (1979) however, noted no significant effect of age on suprathreshold sucrose intensity or pleasantness. Some of the effects of age on taste have been attributed to the wearing of dentures. Elevated (Henkin and Christiansen, 1967; Hermel et al., 1970), lowered (Bartoshuk et al., 1986) or unaffected (Grzegorzczak et al., 1979) taste thresholds associated with the wearing of dentures have been reported in the literature. Ko (1988) found that dentures significantly contributed to the slope for suprathreshold salty taste intensity for elderly men. However, for men and women combined, the wearing of dentures was not significantly related to suprathreshold salty taste intensity (Chauhan, 1989b; Zallen et al., 1990), salty pleasantness (Zallen et al., 1990) or to sour taste intensity (Chauhan, 1989b).

Obesity can influence taste preferences. Rodin et al. (1976) observed that glucose pleasantness responses were increased in obese compared to normal weight control subjects. Weight loss reduced the glucose pleasantness ratings of the obese subjects to that of the controls (Rodin et al., 1976). Conversely, Enns et al. (1979) reported that sucrose preference scores were significantly inversely correlated with body fat. Drewnowski (1987) evaluated the suprathreshold intensity and pleasantness data of underweight, normal weight and overweight subjects for sweet (sucrose) and fat tastes. Perceived intensity of sucrose and fat did not differ among groups (Drewnowski, 1987). However, preference scores for sucrose relative to fat was inversely correlated with body mass index (Drewnowski, 1987).

The importance of the selection of an appropriate control group in studies of taste perception in cancer patients was aptly demonstrated by Kamath et al. (1983). No significant differences in the detection or recognition thresholds for any of the four taste modalities were recorded between 12 esophageal cancer patients and 14 age, smoking, and alcohol consumption matched hospitalized controls (Kamath et al., 1983). However, when the 12 esophageal cancer patients and eight young, healthy controls were compared, the cancer patients had significantly elevated detection thresholds for sour and bitter tastes and elevated recognition thresholds for salty, sour and sweet tastes (Kamath et al., 1983).

Meaningful information regarding the subjective taste changes in cancer patients is limited. However, the frequency with which subjective taste alterations occur demands that taste perception in cancer patients be further investigated. Therefore, controlled clinical measurement of suprathreshold taste perception in cancer patients is required.

#### Taste Perception, Diet and Cancer

Taste is of clinical importance because it may affect food intake. Flavour perceptions (including taste) are strongly correlated with the use of foods (Mela and Mattes, 1988). Henkin et al. (1971) and others (Markley et al., 1983; Mattes-Kulig and Henkin, 1985; Mattes et al., 1990) found that patients with dysgeusia (distorted taste) changed their normal dietary habits to avoid certain foods. Anorexia and/or food aversions often accompany subjective changes in taste perception in cancer patients (DeWys and Walters, 1975; Carson and Gormican, 1977). DeWys and Walters (1975) found that in cancer patients of mixed etiology, lowered bitter

recognition thresholds were correlated with meat aversions. Carson and Gormican (1977) observed an inverse relationship between increased sucrose recognition thresholds and subjective reports of appetite in cancer patients of mixed etiology. However, they (Carson and Gormican, 1977) and others (Settle et al., 1979) did not note any significant correlation between cancer patient taste thresholds and food selection. DeWys (1977) reported that caloric intake was reduced in cancer patients (mixed etiology) with abnormal taste thresholds compared to those with normal taste thresholds. Bolze et al. (1982) noted that both taste threshold and subjective taste changes were significantly correlated with weight loss in cancer patients undergoing radiation therapy. Grosvenor et al. (1989) surveyed 254 patients with advanced cancer (mixed etiology) and found a highly significant relationship between taste perception and weight loss. Symptoms of taste alterations occurred significantly more frequently among cancer patients with weight loss than among those with stable weight (Grosvenor et al., 1989). Conversely, Bruera et al. (1984) reported that for 36 cancer patients of mixed etiology, glucose recognition thresholds were not significantly correlated with either energy or protein intake.

Taste thresholds however, may not be accurate predictors of dietary intake. Normal taste stimuli comprise foods with tastants at concentrations well above threshold. Mattes (1985) compared taste thresholds and suprathreshold taste perception of bitter and sweet qualities in aqueous and food systems with the 7-day dietary records of 35 healthy, non-smoking adults. For sweetness in both systems, suprathreshold data accounted for a larger proportion of the variance in the intake of sweet foods than did threshold data (Mattes, 1985). For

bitterness in both systems, suprathreshold intensity functions accounted for a larger proportion of the variance in bitter food intake than did the threshold or preference responses (Mattes, 1985).

To date, only Trant et al. (1982) have investigated the relationships between suprathreshold taste perception and dietary intake in cancer patients. Suprathreshold taste perception responses of 24 lung cancer and 38 upper gastrointestinal cancer patients were compared with intakes of energy, protein, fat and carbohydrate (Trant et al., 1982). No significant correlations between any gustatory parameter and nutrient intake were noted (Trant et al., 1982).

Taste receptors are modified epithelial cells grouped in barrel-shaped aggregates (taste buds) beneath a pore in the oral epithelial sheet (Beidler, 1970; Murray and Murray, 1970). Microvilli, on the apical ends of the receptor cells, extend into the pore sensing the fluid chemistry of the oral environment (Oakley, 1986). The primary process of taste is the weak, reversible adsorption of electrolytes and non-electrolytes to hypothetical receptor proteins present in the taste cell microvilli (Kamath, 1982). The receptor cells are in a constant state of renewal ensuring viable receptors in spite of repeated mechanical, thermal and chemical damage (Oakley, 1986). The average life span of a receptor cell is about 10 days (Beidler, 1970). Taste cells are in anatomical contact with nerve fibers (Murray and Murray, 1970); synaptic connections degenerate and reform as taste cells die and are replaced (Kamath, 1982). Taste receptor or nerve damage and/or altered taste receptor renewal may affect taste function. In cancer patients, direct taste receptor and nerve damage can result from radiation therapy (Conger and Wells, 1969;

Mossman and Henkin, 1978). Reduced taste receptor turnover may occur as a systemic effect of malignancy (DeWys, 1972), anti-proliferative drugs (Schiffman, 1983a), and/or as an effect of malnutrition (Schiffman, 1983a). A deficiency of any nutrient of sufficient magnitude could be expected to alter chemosensory function by impairing cellular processes (Mattes and Mela, 1988). Because of their rapid turnover rate, taste receptors would be particularly susceptible to nutrient deficiencies.

Cancer patients are often malnourished (Dreizen et al., 1990). Weight loss and protein energy malnutrition are accepted systemic effects of cancer and have a significant negative influence on cancer survival (DeWys et al., 1980). Abnormal indices of vitamin and mineral nutriture are also common in cancer patients. Reduced plasma levels of zinc in some cancer patients (Davies, 1968; Henkin, 1977; Bolze et al., 1982; Mellow et al., 1983) and increased tissue levels of zinc content of breast (Mulay et al., 1971; Schwartz et al., 1974) and other tumors (Mulay et al., 1971) compared to control values have been reported. Basu et al. (1989) determined that serum levels of vitamin E, vitamin A and beta-carotene were slightly reduced in advanced breast cancer patients when compared to controls. Rigby (1991) observed that serum vitamin A levels of post-operative disease-free breast cancer patients were significantly less than those of control subjects. Low serum levels of vitamin A in untreated non-breast cancer patients compared to controls have also been reported (Basu et al., 1976; Mellow et al., 1983; Basu et al., 1987). Low serum levels of thiamin, riboflavin and vitamin B6 have been observed for untreated breast cancer patients compared to controls (Potera et al., 1977; Leklem et al., 1979; Ladner and Salkeld, 1987). In addition,

vitamin B6-dependant metabolism of tryptophan was altered in mastectomized breast cancer patients (Rose, 1967) and in advanced cancer patients of mixed etiology (Basu et al., 1973). Rao et al. (1965) noted low serum folate levels in cancer patients (mixed etiology) compared to controls. Reduced leucocyte ascorbic acid levels have also been seen in advanced breast cancer patients (various treatments) (Basu et al., 1974) and other cancer patients (Krasner and Dymock, 1974) compared to controls.

It is not clear whether the abnormal biochemical indices of nutritional status in cancer patients were caused by the direct effect of disease or by inadequate dietary intake. Abnormal nutrient indices in cancer patients have varied with histology (Davies et al., 1968; Mulay et al., 1971; Basu et al., 1976) and stage of malignancy (Basu et al., 1974; Potera et al., 1977; Ladner and Salkeld, 1987). Serum vitamin A levels were similar for patients with either benign or malignant colorectal disease (Basu et al., 1987). Serum vitamin A (Basu et al., 1989; Rigby, 1991) and beta-carotene (Basu et al., 1989) levels did not differ between patients with benign or malignant breast disease. Advanced breast cancer patients displayed low leucocyte levels of vitamin C despite vitamin supplementation (Basu et al., 1974). However, inadequate intake was cited as a cofactor in the low serum levels of vitamin B6 observed in breast cancer patients (Potera et al., 1977; Leklem et al., 1979) and in the low levels of leucocyte ascorbic acid (Krasner and Dymock, 1974) and serum folate (Rao et al., 1965) noted in non-breast cancer patients. The reduced serum levels of vitamin A and zinc seen in esophageal cancer patients were also partly attributed to reduced nutrient intake (Mellow et al., 1983). However, only Leklem et al. (1979) directly assessed the

diets of their cancer subjects. No correlations between dietary parameters and serum vitamin B6 levels for breast cancer patients were observed (Leklem et al., 1979).

Protein energy malnutrition could be expected to affect taste by inhibiting receptor cell turnover in a manner similar to that known to occur in the gastrointestinal mucosa (Schiffman, 1983a). Russ and DeWys (1978) suggested that overt malnutrition was responsible for the elevated sucrose recognition threshold observed in an anorexic patient with bladder cancer. Intravenous hyperalimentation, accompanied by a positive nitrogen balance and weight gain, reduced the sucrose threshold to normal and alleviated the anorexia (Russ and DeWys, 1978). Bolze et al. (1982) observed that in cancer patients of mixed etiology undergoing radiation therapy, weight loss was more closely correlated to alterations in taste thresholds than was radiation.

Most research examining the interactions of nutrients and taste perception has focused on zinc and vitamin A. Limited study of the relationships between taste perception and other nutrients has been conducted. To date, data relating nutritional status to taste perception in cancer has been confined to indices of zinc nutriture (Bolze et al., 1982; Trant et al., 1982).

Zinc is a cofactor of many metalloenzymes (Parisi and Vallee, 1969) and is required for nucleic acid and protein synthesis (Prasad, 1967; Mills et al., 1969). Taste receptors would be especially sensitive to zinc deficiency. Zinc is also part of a metalloprotein (gustin) found in the saliva surrounding taste buds (Henkin et al., 1975). Decreased saliva concentrations of gustin have been determined in patients with hypogeusia

(loss of taste) (Shatzman and Henkin, 1981). Zinc is also found in taste bud receptors (Law and Henkin, 1983) and in peripheral and cranial nerve tissue (Henkin et al., 1979), suggesting a role of zinc in the transmission of taste information. In man, experimental zinc deficiency produced reversible changes in taste acuity (Wright et al., 1981; Prasad, 1985). In subjects with taste aberrations, zinc supplements were reported to normalize taste acuity (Henkin and Bradley, 1970; Henkin et al., 1971; Hambidge et al., 1972; Schecter et al., 1972; Atkin-Thor et al., 1978; Shatzman and Henkin, 1981). Henkin (1977) was able to reverse hypogeusia accompanied by low serum levels of zinc in seven cancer patients (mixed etiology) with zinc supplementation. However, more recent attempts to correlate indices of zinc nutriture and taste perception in cancer patients have been unsuccessful. Bolze et al. (1982) found no significant correlations between plasma zinc values and taste thresholds for cancer patients of mixed etiology. A study by Trant et al. (1982) revealed no significant correlations between hair zinc and suprathreshold taste intensity or pleasantness responses for any taste modality in lung and upper gastro-intestinal cancer patients.

Vitamin A is important for maintenance of healthy epithelial tissue (Wolbach and Howe, 1925). Keratinization of the tongue, including the taste bud pore has been observed in vitamin A deficient rats (Bernard et al., 1961; Bernard and Halpern, 1968). Reversible abnormal taste responses to NaCl and quinine solutions were produced in rats fed a vitamin A deficient diet (Bernard et al., 1961; Bernard and Halpern, 1968). Experimental vitamin A deficiency in man was accompanied by reports of taste alterations (Sauberlich et al., 1974; Hodges and Hodges,

1980). Vitamin A metabolism is dependent on the availability of zinc-containing enzymes and a deficiency of zinc will result in a secondary deficiency of vitamin A (Solomons and Russell, 1980).

Experimental deficiencies of riboflavin, niacin, vitamin B6 and folate in animals have resulted in glossitis and atrophy of the lingual papillae and epithelium (Afonsky, 1960). Green (1971) reported a case of reversible subclinical pellagra in conjunction with hypogeusia in man. In rats, vitamin B6 deficiency caused altered intakes and preferences for salty, sweet, and bitter taste solutions (Grewack et al., 1977; Chan and Kare, 1979; Greeley and Gniecko, 1986). Patients with toxicity of 5-thiopyridoxine (an antirheumatic drug and vitamin B6 antagonist) have reported subjective losses in taste (Huskisson et al., 1980).

Taste perception is a strong motivator of food selection. For cancer patients, abnormalities in taste perception may lead to alterations in dietary intake, compromise in nutritional status and further changes in taste perception. Investigation of the effect of taste on diet in cancer patients is extremely limited. Therefore, examination of the relationships between taste perception and dietary intake in cancer patients is warranted. A project, undertaken to quantitate the suprathreshold taste perception and dietary intake of breast cancer patients and matched healthy controls is relevant. Such research would permit the investigation of relationships among dietary and taste perception parameters for breast cancer patients.

### 3 METHODOLOGY

Data collection took place from March to October, 1989. Each subject participated in 3 test sessions of approximately 2 hours duration. Subjects were interviewed individually either at the Department of Foods and Nutrition, or at the subjects' homes. The general protocol for data collection is presented in Table 1.

#### Subject Characteristics

##### Subject Selection

Two groups of women aged 44-56 years, participated in the present study: 24 breast cancer outpatients and 24 control women. Breast cancer subjects were obtained through the Northern Alberta Breast Cancer Registry, Edmonton, Alberta. All breast cancer subjects had Stage I breast malignancy: primary mammary gland T1 or T2 tumors ( $\leq 2$  cm or  $> 2$  cm but  $< 5$  cm in the greatest dimension, respectively) and negative lymph node biopsy. Surgery was the only mode of treatment and all cancer subjects were free of recurrence. Approval to approach the subject was obtained from the patient's personal physician by telephone and a letter of confirmation (Appendix 1). One hundred and four (104) breast cancer patients were contacted: 11 (11%) were unwilling to participate, 69 (66%) were disqualified according to study criteria, and 24 (23%) agreed to participate. Volunteer control subjects matched to the breast cancer subjects by age and relative body weight, were recruited from the local community.

Suitable subjects met the following criteria:

- 1) No known condition requiring undue diet modification.

Table 1. Data collection protocol.

Appointment	Sensory Evaluation	Dietary Evaluation
1	Sign informed consent. Complete questionnaire. Complete Anthropometric measurements. Orient subject to the sensory procedure.	
	Taste quality I	24-hour recall; instruct on food record completion (one day).
	Taste quality II	
2	Taste quality I	Review food record (one day);instruct on food record completion (two day).
	Taste quality II	
3	Taste quality I	Review food record (two day).
	Taste quality II	
	Present gift of appreciation.	

- 2) No nasal obstruction.
- 3) No prior neurosurgery or recent head injury.
- 4) No facial hypoplasia.
- 5) No depression or nervous system disorders.
- 6) No current smoking habit.
- 7) No artificial dentition.

In addition, all subjects had resided in North Central Alberta for at least six months; were able to communicate in English; and were willing and able to participate. Subject eligibility was further determined on the basis of responses to the first page of a subject profile questionnaire (Appendix 2, Part I). Each eligible subject was then fully informed about the study and signed a consent form (Appendix 3, Parts I and II) before participating in the study.

#### Anthropometric Data

Each subject was measured for height, weight, triceps skinfold thickness (TSF) and mid-arm circumference (MAC). Measurements were made while the subject wore light indoor clothing without shoes.

Height was measured with a steel measuring tape from the floor to a point where a horizontal, flat metal plate met the subject's crown while the subject stood with her heels, buttocks, shoulders and head against a vertical surface. Weight was obtained using a portable spring scale (Precision Scale Co. Ltd.). Relative body weight (RBW) was determined using the height and weight measurements of each subject and desirable body weight tables (Metropolitan Life Insurance Co., 1959).

Upper mid-arm circumference was measured with a flexible, non-stretchable tape measure, at a point half-way between the tip of the elbow and the acromial process of the scapula with the unclothed arm hanging relaxed at the side. Mid-arm triceps skinfold thickness was measured along the posterior mid-line of the same arm. The skinfold over the triceps muscle was grasped at a point 1 cm above the midpoint and measured with a Lange skinfold calliper. Measurements were made of the control subjects' non-dominant arm and the breast cancer subjects' non-mastectomy arm. In the event of double mastectomy, the patient's non-dominant arm was chosen. Mid-arm muscle circumference (MAMC) was determined using a standard equation:

$$\text{MAMC(cm)} = \text{MAC(cm)} - (0.314 \times \text{TSF(mm)})$$

MAMC, MAC, and TSF were also expressed as % standard, using sex and age specific standards [50th percentile for the Canadian population (Jette, 1983)].

The theoretical basal energy requirement (BER) for each subject was calculated using the Harris-Benedict equation for women (Harris and Benedict, 1919):

$$\text{BER (kcal)} = 655.10 + 9.56(W) + 1.85(H) - 4.68(A)$$

where W = desirable body weight (kg) for height (Metropolitan Life Insurance Co., 1959), H = height (cm), and A = age (years).

#### Subject Profile Data

Subject profile information including: medication use, alcohol consumption, past smoking habits, salt use, and demographic data such as

education, income and marital status were obtained from replies to the subject profile questionnaire (Appendix 2, part II).

### Taste Perception

#### **Tastants**

To avoid gustatory fatigue, only two taste modalities: salty and sweet, were evaluated. Aqueous and food systems, containing sodium chloride (salty) and sucrose (sweet) at six suprathreshold concentrations were prepared. Each concentration differed from the next by a one quarter log molar step. The lowest concentration of each taste modality was higher than the reported salty and sweet detection thresholds for healthy adults of comparable age to the study participants (Weiffenbach et al., 1982; Grzegorzczuk et al., 1979). Aqueous solutions and simple food systems (strained peas or applesauce) contained 40, 72, 130, 233, 420, and 756 mM concentrations of sodium chloride (NaCl) or sucrose, respectively. Fresh batches of each modality and system (salty/sweet in water/food) were prepared weekly according to standardized techniques. All sample preparation was completed by the author.

Appropriate amounts of commercial table salt [Windsor<sup>R</sup> Salt (Appendix 4)] were added to double distilled deionized water and strained peas (courtesy of H.J. Heinz Co. Ltd.). Commercial sucrose (Alberta Sugar Co.) was added to double distilled deionized water and junior applesauce (courtesy of H.J. Heinz Co. Ltd.). Food grade guar gum (8/22 mesh, courtesy of TIC Gums Inc.) was added to the applesauce to ensure similar viscosity across concentrations of sucrose. Guar was added to the sucrose/applesauce mixtures in amounts of 0.01, 0.03, 0.07, 0.10, 0.19 and

0.25% (w/w) corresponding to the ascending concentrations of sucrose. Viscosity measurements (Brookfield Digital Viscometer) of the salt/peas and sucrose/guar/applesauce mixtures are shown in Table 2. No differences in viscosity were noted among concentrations of salt in peas or among concentrations of sucrose/guar in applesauce.

Samples were dispensed (in seven mL portions) into disposable plastic Souffle<sup>R</sup> cups (29.6 mL, Solo Cup Co.) and refrigerated at  $2\pm 1^{\circ}\text{C}$  until required. Samples were coded with random three-digit numbers. Aqueous solutions were tasted directly from the Souffle<sup>R</sup> cups; foods were sampled with the aid of five-inch plastic spoons (Listo Products Ltd.). Aqueous solutions were evaluated at room temperature ( $21\pm 2^{\circ}\text{C}$ ); foods at temperatures appropriate to each food type. The salt/peas mixtures were brought to and maintained at  $48\pm 2^{\circ}\text{C}$  in a closed water-bath double-boiler system using two Corningware<sup>R</sup> casserole dishes fitted together on a heated Salton<sup>R</sup> Hotray. Sucrose/guar/applesauce mixtures were maintained at  $11\pm 1^{\circ}\text{C}$  by placing the samples in one cm of water in an aluminum tray, in which a reusable Ice-Pak<sup>R</sup> (Stanbel Ltd.) was immersed.

#### Tastant Evaluation

Subjects were asked to refrain from eating or drinking anything except water for at least one hour before testing. Prior to beginning the taste sessions, the salivary pH of each subject was measured using short range pH paper [pH 6.0 to 8.5 (Fisher Scientific Co.)]. Salivary pH measurements for all subjects were within normal range.

Samples were evaluated using a standardized method of "sip and spit". Subjects were instructed to taste but not swallow the samples.

Table 2. Apparent viscosities of salt in peas and sucrose/guar in applesauce.

Product	Additive	Concentration (mM)	Apparent Viscosity (CPS) <sup>1,2</sup>
Strained Peas	Salt (NaCl)	40	8440.0(158.7) <sup>3</sup>
		72	8540.0( 81.2)
		130	8793.3( 89.7)
		233	8433.3( /1.1)
		420	8953.3(283.9)
		756	8413.3( 96.7)
Applesauce	Sucrose/Guar <sup>4</sup>	40/0.01 <sup>5</sup>	15473.3(136.0)
		72/0.03	15286.7(123.2)
		130/0.07	15546.7(243.9)
		233/0.10	15320.0(147.9)
		420/0.19	15580.0(169.0)
		756/0.25	15653.3(133.7)

<sup>1</sup>Centipoises.

<sup>2</sup>Mean of 6 determinations.

<sup>3</sup>Mean(standard error of the mean).

<sup>4</sup>TIC Gums Inc.

<sup>5</sup>Weight percent guar.

Each subject was asked to place and hold the contents of the sample cup in her mouth for about five seconds. All samples were expectorated into a styrofoam cup. The subject then rinsed her mouth with double distilled deionized water ad libitum, took a bite of unsalted cracker [Premium Plus<sup>R</sup> Crackers (Unsalted Tops), Christie Brown and Co.] and rinsed her mouth again. Rinse water was also expectorated. The rinsing procedure was performed prior to and between all sample evaluations. A minimum of 20 seconds passed between the final water rinse and tasting the next sample.

Suprathreshold taste perception of intensity and pleasantness was assessed using unstructured category line scaling (Giovanni and Pangborn, 1983). For perceived taste intensity, a 15 cm horizontal line, anchored at either end from least salty (sweet) to most salty (sweet) was employed. For perceived pleasantness, a similar line anchored at either end from least to most pleasant was used (Appendix 5). Prior to evaluating the test samples, subjects were familiarized with the line scaling procedure by tasting two samples, marked "L" and "M" corresponding to the least and most intense taste modality (salty or sweet), respectively. Anchors were presented in a randomized, balanced manner. Then a reference sample marked "R" of a specified intensity (Appendix 5) was tasted. Subjects were then presented with six test samples (including a hidden reference) in a partially randomized order, using a modification of the sequence order of Hyde et al. (1981). No two consecutive test samples differed in concentration by greater than four-fold. The order of anchor, reference and test sample presentation was indicated on the scoresheets for each taste modality and system (Appendix 5), and were identical for each breast cancer subject and her matched control.

For each test sample, the subject made a vertical mark on the intensity line according to her perceived intensity of the sample in relation to the anchors and the reference. The subject then marked the pleasantness line according to her perception of the pleasantness of the test sample. For each sample, numerical scores were assigned to the intensity and pleasantness responses by measuring the distance (in cm) from the origin (least intense/pleasant) to the mark made by the subject.

Suprathreshold taste perception measurements (intensity and pleasantness) were replicated three times. For each taste modality, replicates took place on separate days (Table 1). At each taste session, two series of the same taste modality (salt or sucrose) were presented: first the aqueous then the food system. A break of five or more minutes between systems (aqueous and food) within a taste series was required. After a rest period of at least 20 minutes, subjects were given the two series of the second taste modality (sucrose or salt). Taste modality presentation was randomized, balanced and identical for each breast cancer subject and her matched control.

### Dietary Data

A combination of 24-hour recall and food records was used to collect quantitative dietary data for four complete days (three weekdays and one weekend day) for each subject. The dietary assessment schedule was coordinated with taste perception testing (Table 1). Dietary data were collected by two researchers trained in the Nutrition Canada techniques of quantitative dietary assessment (Health and Welfare Canada, 1973). The

author supervised the collection of all dietary data and personally interviewed approximately two thirds of the subjects.

Initially, a 24-hour recall of food intake was obtained. Each subject was asked to recall in chronological order, all foods and beverages consumed over the prior 24-hour period, starting when the subject awakened. The recall was itemized on a dietary intake form (Appendix 6) and was reviewed with the subject to ensure that no item was overlooked. Detailed descriptions (eg. brand, preparation method, etc.) of each food item were obtained. Portion sizes were estimated by the subject with the aid of food models (constructed according to Nutrition Canada specifications) and by observations by the author of the dishes and utensils used in the home. Skilled probing on the part of the researchers ensured accuracy and completeness of dietary data collection. Each subject was then given a one-day food record form (Appendix 7) to be completed for the 24 hours immediately preceding the next tasting session. The researchers explained food record-keeping to the subject and included a sample food record and written instructions with the record for use as a guide (Appendix 7). At the next visit, the completed one-day food record was reviewed with the subject. A two-day food record form was then provided for the subject to complete for the 48 hours immediately preceding the last taste session. The two-day record was also reviewed with the subject. During each dietary interview, details about all vitamin or mineral supplements taken by the subject were recorded, including type, brand, amount taken, and frequency of supplement use.

For four breast cancer subjects, five days of dietary intake data were collected; four days were not sufficient to describe their usual food intake.

All recorded food items were coded by the researchers according to coding procedures standardized at the Department of Foods and Nutrition. All coded food records were reviewed and verified by the author. The coded data were then entered onto the mainframe computer for nutrient analysis.

The nutrient data base used consisted of The Canadian Nutrient File (Health and Welfare Canada, 1985) based on the United States Department of Agriculture Handbook #456 (Adams, 1975) to which Canadian food composition data were added. The nutrient data base also contained food compositional values for zinc, dietary fiber and cholesterol from other sources (Feeley et al., 1972; Murphy et al., 1975; Freeland and Cousins, 1976; Paul and Southgate, 1978; Freeland-Graves et al., 1980; Lawler and Klevay, 1980; McNeill et al., 1985).

Nutrient intakes per day were calculated for the following 25 nutrients: kilocalories (kcal), protein, fat, saturated fat, monounsaturated fat, polyunsaturated fat, cholesterol, carbohydrate, sugar, starch, dietary fiber, calcium, phosphorus, iron, sodium, potassium, zinc, vitamin A, thiamin, riboflavin, niacin, vitamins B6 and B12, folacin, and ascorbic acid. Percent risk of nutrient deficiency was calculated by a probability approach using a software package designed for an Apple IIe microcomputer (Beaton, 1984). This approach recognizes that the Recommended Nutrient Intakes (Health and Welfare Canada, 1990) exceed the actual requirements of almost all individuals. The lower an

individual's nutrient intake compared to the recommended level, the greater the probability of not meeting the individual's actual requirement for that nutrient (Anderson et al., 1982). For each subject's intake, the percent risk of nutrient deficiency for protein, thiamin, riboflavin, vitamins B6 and B12, folacin, vitamin A, ascorbic acid, calcium, iron, and zinc were determined. An index of overall nutritional risk was calculated as an average of the 11 nutrient risks.

Daily intakes of food items were classified according to the following food groups (Davenport, 1964): dairy products; meat, poultry, fish and eggs; cereal products; fruit and fruit products; vegetables; fats and oils; nuts and legumes; foods primarily sugar; and miscellaneous items (including food group combinations, soups, condiments, and items not classified elsewhere).

### Data Analysis

Data analysis was carried out using the Statistical Package for the Social Sciences (SPSS-X, 1988).

### **Subject Characteristic and Profile Data**

Group mean anthropometric and subject characteristic values were compared using analysis of variance (ANOVA). The frequency distributions of relative body weights for the cancer and control groups were compared using Kolmogorov-Smirnov analysis. Individual category responses to the subject profile questionnaire were compared between study groups using Chi-Square analysis (Steel and Torrie, 1980).

### Taste Perception Data

For each subject, average replicate intensity estimates for each sample were calculated and used for the estimation of group mean values for statistical analysis. For each tastant and system, mean intensity estimates for each of the six concentrations were compared between the cancer and control groups using ANOVA. For each group and tastant, mean intensity estimates for each of the six concentrations were compared between the aqueous and food systems by ANOVA. For each tastant, interactions between concentration and group or system effects were determined by three-way ANOVA.

Indices of suprathreshold taste intensity perception were computed by linear regression analysis. Mean taste intensity estimates were regressed on the logs of the six tastant concentrations. Linear regression coefficients (slopes) were computed for each subject for each tastant and system. For each tastant and system, mean slope values were compared between groups using ANOVA. For each group and tastant, mean slope values were compared between systems by ANOVA.

Excluding the regression analyses, the statistical analysis of the taste pleasantness data was the same as that for the taste intensity data. Regression analyses were not performed on the taste pleasantness data.

### Dietary Data

For each subject, the dietary intakes for 25 nutrients were determined for each recalled or recorded day. Average daily intakes of each nutrient with and without vitamin/mineral supplementation were calculated for each subject. ANOVA was employed to compare group mean

daily nutrient intakes and percent risks of nutrient deficiency with and without supplementation. Group mean nutrient density values (daily nutrient intakes per 1000 kcal) for the non-supplemented dietary data were also compared by ANOVA. The frequency distributions of the average daily energy intake values of the cancer and control subjects were compared by Kolmogorov-Smirnov analysis.

#### Relationships Between Taste Perception and Dietary Data

Pearson correlation coefficients were determined for slopes for taste intensity vs the non-supplemented dietary data for: 1) nutrient intake; 2) percent risk of nutrient deficiency; and 3) nutrient density.

Relationships between taste intensity and non-supplemented dietary data were evaluated by linear stepwise multiple regression analysis. Stepwise regression equations were calculated for each taste modality and system. Dependent variables were the slopes for salt and sucrose intensity in aqueous and food systems. Independent variables were the non-supplemented dietary intake data for: 1) nutrient intake; 2) percent risk of nutrient deficiency; and 3) nutrient density. Independent variables were entered into the equation in the order of highest partial correlation coefficient between that variable and the dependent variable. The percent of total variance in the dependent variable accounted for by an independent variable is additional to that provided by the preceding independent variables entered into the equation. The critical F value for entry into the regression equation was at  $p \leq 0.10$ , therefore, all variables entered into the equations were significant to  $p \leq 0.10$  but not all were significant at  $p \leq 0.05$ .

#### 4 RESULTS

##### Subject Characteristics

The characteristics of the cancer and control groups are presented in Table 3. Each group comprised 24 women. The mean age of the two groups did not differ; the mean ages were 50.8 years for the cancer subjects and 49.2 years for the controls. Anthropometric measurements did not differ between the study groups. Average relative body weight, based on desirable body weight (Metropolitan Life Insurance Co., 1959), was 113.7% for the cancer group and 113.0% for the control group. Mean body mass indexes for the cancer and controls groups were 24.8 and 24.7, respectively. Mean mid-arm muscle circumference and triceps skinfold thickness measurements for both groups were similar to the Canadian reference standards (Jette, 1983).

Subject profile information, socioeconomic factors and variables that affect taste perception or dietary intake are shown in Table 4. Seven of 24 (29%) cancer subjects compared to 18/24 (75%) control subjects were of British or Western European origin ( $p \leq 0.01$ ). All subjects had received secondary education. Compared to control subjects, significantly ( $p \leq 0.01$ ) more cancer subjects had career training while significantly ( $p \leq 0.01$ ) fewer cancer subjects had post-secondary education. Most participants had company at meals; eating alone was less prevalent ( $p \leq 0.05$ ) in the cancer than in the control group. Use of the salt shaker was similar for the two groups; none of the subjects used the salt shaker on a regular basis. Significantly ( $p \leq 0.01$ ) fewer cancer subjects than controls consumed alcohol. Of those who drank, 89% of the cancer subjects and 92% of the controls were occasional ( $\leq 1$  drink/day) drinkers. Smokers

Table 3. Study group characteristics.

Characteristic	Cancer	Control
Number of subjects	24	24
Age (years)	50.8(0.6) <sup>1</sup>	49.2(0.7)
Height (cm)	163.5(1.4)	162.6(0.8)
Weight (kg)	66.5(1.9)	65.3(1.8)
(RBW <sup>2</sup> (%))	113.7(2.9)	113.0(2.6)
BMI <sup>3</sup> (kg/m <sup>2</sup> )	24.8(0.6)	24.7(0.6)
MAC <sup>4</sup> (cm)	29.1(0.6)	29.8(0.6)
MAMC <sup>5</sup> (cm)	21.9(0.5)	22.8(0.4)
(% of standard MAMC)	98.1(2.1)	101.9(2.0)
TSF <sup>6</sup> (mm)	22.7(1.2)	22.4(1.0)
(% of standard TSF)	101.3(5.6)	102.5(4.6)

<sup>1</sup>Mean(standard error of the mean).<sup>2</sup>Relative body weight.<sup>3</sup>Body mass index.<sup>4</sup>Mid-arm circumference.<sup>5</sup>Mid-arm muscle circumference.<sup>6</sup>Triceps skinfold thickness.

Table 4. Subject profile data.

Profile	Cancer <sup>1</sup>	Control <sup>1</sup>
Ethnic Origin		
Britain and Western Europe	7 <sup>2</sup>	18**
Germany	9	5
Eastern Europe	5	1
Other	3	0
Education		
High School	9	5
Career Preparation	12	3**
Post Secondary	3	16**
Annual Income (self and spouse)		
Less than \$20,000	1	2
\$20,000-\$29,999	4	2
\$30,000-\$39,999	5	3
\$40,000-\$49,999	4	3
\$50,000-\$59,999	2	7
Greater than \$59,999	7	7
Not released	1	0
Company at Meals		
None	1	7*
One other person or more	23	17
Salt Added at the Table		
Never	10	13
Occasionally	14	11
Usually	0	0
Alcohol Consumption		
Total Drinkers	18	24**
Never	6	0
Occasional (less than 1 drink/day)	16	22
Regular (more than 5 drinks/week)	2	2
Previous Smoking Habit		
Never	15	13
Less than 20 cigarettes/day	6	6
More than 20 cigarettes/day	3	5
Length of time since quitting		
Less than 10 years	2	2
More than 10 years	7	9

<sup>1</sup>n=24.<sup>2</sup>Number of subjects.\*, \*\* Significant at  $p \leq 0.05$ ,  $p \leq 0.01$ , respectively.

were excluded from this study. Fifteen (63%) cancer subjects and 13 (54%) control subjects had never smoked. Of those who had previously smoked, over 50% in each group had stopped smoking more than ten years ago.

Information on vitamin/mineral supplement and medication use is given in Table 5. Seventeen (71%) cancer and 16 (67%) control subjects used supplements. Single nutrient supplementation was most common: calcium for the cancer group (9/17 subjects) and ascorbic acid for the controls (6/16 subjects). Of participants who used more than one supplement daily, four cancer subjects and three controls took more than four supplements each day. One half of the women in each group were taking medications. The most common medications were sedatives for the cancer group (5/12) and estrogen for the controls (7/12). None of the cancer subjects took estrogen. The number of medications taken per user ranged from one (10/12 and 8/12 cancer and control subjects, respectively) to three (taken by one person within each group).

### Taste Perception

#### Taste Intensity

##### Comparison of Cancer and Control Groups

The average slopes and intercepts of the taste intensity functions for saltiness and sweetness for the cancer and control groups are presented in Table 6. For saltiness and sweetness in both aqueous and food systems, taste intensity slopes and intercepts did not differ between groups.

Mean intensity estimates of saltiness for the cancer and control subjects are given in Table 7. For saltiness in both aqueous and food

Table 5. Use of vitamin/mineral supplements and medications.

Supplement/Medication	Cancer <sup>1</sup>	Control <sup>1</sup>
Vitamin/Mineral Supplement Use		
Never	7 <sup>2</sup>	8
Irregular	4	3
Regular	13	13
Vitamin/Mineral Product		
Single vitamin and/or mineral	13	10
Multivitamin $\pm$ minerals	10	12
Fiber supplement	1	2
Number of Vitamin/Mineral Supplements Taken		
One	11	9
Two or more	6	7
Medications		
Subjects using medications	12	12
Estrogen	0	7
Thyroid hormone	2	3
Anti-inflammatories	4	3
Antihypertensives	1	2
Sedatives	5	1
H <sub>2</sub> receptor antagonists	2	1

<sup>1</sup>n=24.<sup>2</sup>Number of subjects.

Table 6. Mean slope (cm/log M) and intercept (cm) values for salty and sweet taste qualities for the cancer and control groups.

Taste Quality	System		Cancer <sup>1</sup>	Control <sup>1</sup>
Saltiness	Aqueous	Slope	10.3 $\pm$ 0.4 <sup>2</sup>	9.8 $\pm$ 0.3
		Intercept	15.1 $\pm$ 0.4	14.4 $\pm$ 0.3
	Food <sup>3</sup>	Slope	8.5 $\pm$ 0.5	8.3 $\pm$ 0.4
		Intercept	12.5 $\pm$ 0.5	12.5 $\pm$ 0.5
Sweetness	Aqueous	Slope	9.9 $\pm$ 0.5	9.0 $\pm$ 0.4
		Intercept	14.3 $\pm$ 0.5	13.2 $\pm$ 0.5
	Food <sup>4</sup>	Slope	6.0 $\pm$ 0.5	5.0 $\pm$ 0.5
		Intercept	11.5 $\pm$ 0.5	10.8 $\pm$ 0.5

<sup>1</sup>n=24.

<sup>2</sup>Mean  $\pm$  standard error of the mean.

<sup>3</sup>Strained peas.

<sup>4</sup>Applesauce.

Table 7. Mean intensity estimates (cm)<sup>1</sup> of concentrations of sodium chloride (NaCl) in aqueous and food systems for the cancer and control groups.

NaCl (mM)	Aqueous		Food <sup>2</sup>	
	Cancer <sup>3</sup>	Control <sup>3</sup>	Cancer <sup>3</sup>	Control <sup>3</sup>
40	1.2 (0.2) <sup>4</sup>	1.4 (0.2)	2.0 (0.4)	1.8 (0.3)
72	3.0 (0.3)	2.6 (0.3)	2.9 (0.4)	3.0 (0.2)
130	5.9 (0.6)	5.0 (0.3)	3.5 (0.4)	3.9 (0.3)
233	8.3 (0.6)	8.0 (0.5)	5.9 (0.5)	6.7 (0.5)
420	11.7 (0.4)	11.1 (0.3)	9.7 (0.4)	9.4 (0.4)
756	13.8 (0.3)	13.3 (0.2)	12.6 (0.4)	12.2 (0.4)

<sup>1</sup>Maximum estimate=15 cm.

<sup>2</sup>Strained peas.

<sup>3</sup>n=24.

<sup>4</sup>Mean (standard error of the mean).

systems, no differences in intensity ratings of the cancer and control groups were noted. Mean taste intensity functions for salt in aqueous and food systems for both groups are plotted in Figure 1. Group coefficients of determination for salt intensity functions in the aqueous system were similar:  $r^2=0.92$  and  $r^2=0.94$  for the cancer and control groups, respectively. For salt intensity functions in food, cancer and control group coefficients of determination did not differ; they were  $r^2=0.86$  and  $r^2=0.90$ , respectively. ANOVA of the salt intensity estimates did not reveal any significant group-related main effects or group x concentration interaction effects for either system.

Mean intensity estimates of sweetness for the cancer and control groups are shown in Table 8. Cancer subjects rated the 756 mM sucrose concentration in the aqueous system as significantly ( $p \leq 0.05$ ) more sweet than did the control subjects. For sucrose in food, no differences in intensity estimates of the cancer and control subjects were observed. Sucrose intensity functions for aqueous and food systems for the cancer and control groups are shown in Figure 2. For the cancer and control groups, coefficients of determination for sucrose intensity functions in both systems were similar:  $r^2=0.92$  and  $r^2=0.94$ , respectively, for the aqueous system;  $r^2=0.73$  and  $r^2=0.72$ , respectively, for the food system. ANOVA of the sucrose intensity estimates revealed no significant group-related or group x concentration interaction effects for either system.

#### Comparison of Aqueous and Food Systems

Mean slopes and intercepts of salty and sweet taste intensity functions for aqueous and food systems are shown in Table 9. For both study groups, slopes for saltiness in food were significantly ( $p \leq 0.01$ )

Figure 1. Intensity estimates of sodium chloride in aqueous and food systems

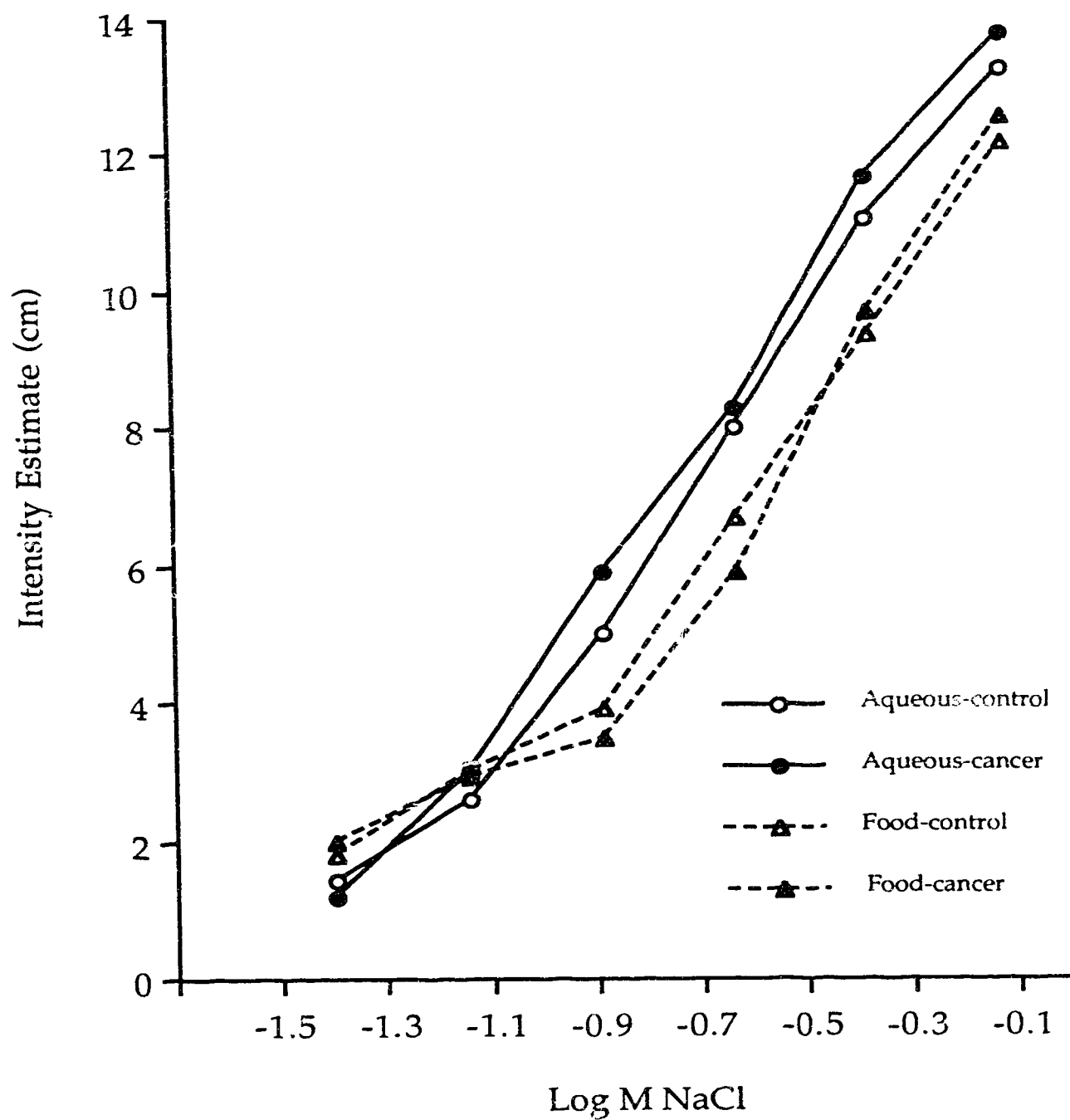


Table 8. Mean intensity estimates (cm)<sup>1</sup> of concentrations of sucrose in aqueous and food systems for the cancer and control groups.

Sucrose (mM)	Aqueous		Food <sup>2</sup>	
	Cancer <sup>3</sup>	Control <sup>3</sup>	Cancer <sup>3</sup>	Control <sup>3</sup>
40	1.3 (0.2) <sup>4</sup>	1.2 (0.2)	3.7 (0.6)	4.2 (0.5)
72	2.5 (0.2)	2.5 (0.2)	4.6 (0.5)	4.8 (0.5)
130	4.8 (0.4)	4.3 (0.3)	5.7 (0.6)	6.7 (0.5)
233	7.9 (0.5)	7.2 (0.4)	7.4 (0.5)	7.1 (0.5)
420	11.0 (0.5)	10.3 (0.4)	9.6 (0.5)	9.2 (0.4)
756	13.2 (0.3)	12.1 (0.4)*	11.0 (0.5)	10.4 (0.5)

<sup>1</sup>Maximum estimate=15 cm

<sup>2</sup>Applesauce.

<sup>3</sup>n=24.

<sup>4</sup>Mean (standard error of the mean).

\* Significant at  $p \leq 0.05$ .

Figure 2. Intensity estimates of sucrose in aqueous and food systems

45

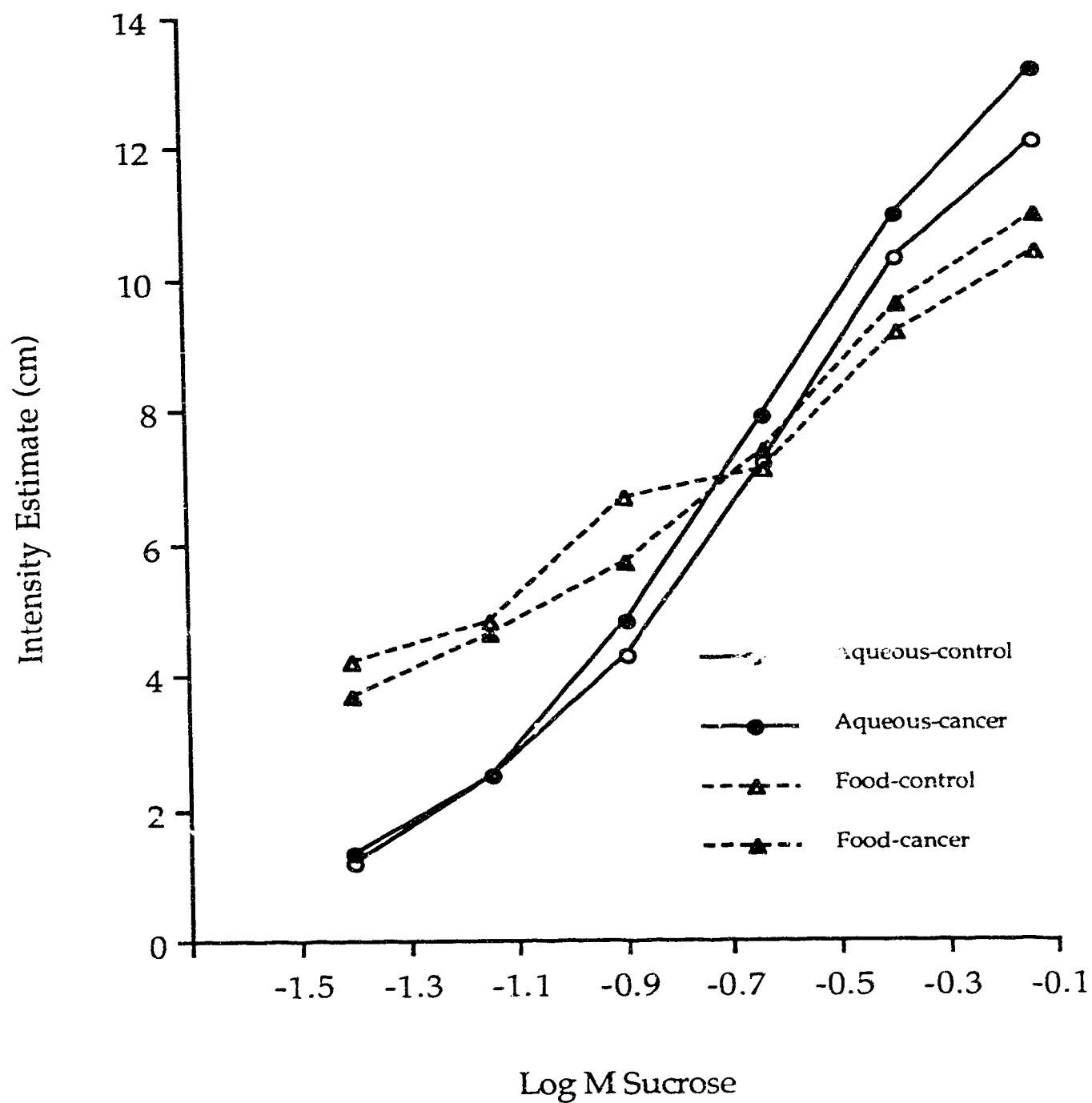


Table 9. Mean slope (cm/log M) and intercept (cm) values for salty and sweet taste qualities in aqueous and food systems.

Taste Quality	Group		Aqueous System	Food System <sup>1</sup>
Saltiness	Cancer <sup>2</sup>	Slope	$10.3 \pm 0.4^3$	$8.5 \pm 0.5^{**}$
		Intercept	$15.1 \pm 0.4$	$12.5 \pm 0.5^{***}$
	Control <sup>2</sup>	Slope	$9.8 \pm 0.3$	$8.3 \pm 0.4^{**}$
		Intercept	$14.4 \pm 0.3$	$12.5 \pm 0.5^{**}$
Sweetness	Cancer <sup>2</sup>	Slope	$9.9 \pm 0.5$	$6.0 \pm 0.5^{***}$
		Intercept	$14.3 \pm 0.5$	$11.5 \pm 0.5^{***}$
	Control <sup>2</sup>	Slope	$9.0 \pm 0.4$	$5.0 \pm 0.5^{***}$
		Intercept	$13.2 \pm 0.5$	$10.8 \pm 0.5^{**}$

<sup>1</sup>Strained peas for saltiness; applesauce for sweetness.

<sup>2</sup>n=24.

<sup>3</sup>Mean  $\pm$  standard error of the mean.

<sup>\*\*</sup>, <sup>\*\*\*</sup> Significant at  $p \leq 0.01$ ,  $p \leq 0.001$ , respectively.

flatter than those for the aqueous system. Comparison of the salt intensity estimates for the aqueous and food systems revealed significant differences for both groups (Table 10). ANOVA of the saltiness estimates showed significant system-related ( $p \leq 0.05$ ) and system x concentration interaction ( $p \leq 0.001$ ) effects for both the cancer and control groups (Figure 1). For both groups, the 40 mM and 72 mM concentrations of NaCl were perceived as equally salty in aqueous compared to food systems. Cancer subjects rated the 130 mM to 420 mM ( $p \leq 0.01$ ) and the 756 mM ( $p \leq 0.05$ ) NaCl concentrations in peas as significantly less salty than those in the aqueous system. For the controls, the 130 mM ( $p \leq 0.05$ ), 420 mM ( $p \leq 0.01$ ) and 756 mM ( $p \leq 0.05$ ) NaCl concentrations were judged as significantly less salty in food compared to the aqueous system.

Slopes for sucrose intensity functions in food systems were significantly ( $p \leq 0.001$ ) flatter than those for aqueous systems for both the cancer and control groups (Table 9). System comparisons of sucrose intensity estimates for the cancer subjects and the controls are displayed in Table 11. ANOVA of sweetness estimates revealed no significant system effects for either group. However, significant ( $p \leq 0.001$ ) system x concentration interaction effects on sweetness intensity estimates were observed for both groups (Figure 2). For the cancer group, the 40 mM and 72 mM sucrose concentrations were judged as significantly ( $p \leq 0.001$ ) more sweet in applesauce than in the aqueous system. For the cancer group, the 420 mM ( $p \leq 0.05$ ) and 756 mM ( $p \leq 0.001$ ) sucrose concentrations were perceived as significantly less sweet in applesauce compared to the aqueous system. For the control subjects, the 40 mM to 130 mM sucrose concentrations were perceived as significantly ( $p \leq 0.001$ ) more sweet while the 756 mM sucrose

Table 10. Mean intensity estimates (cm)<sup>1</sup> of concentrations of sodium chloride (NaCl) in aqueous and food systems.

NaCl (mM)	Cancer <sup>2</sup>		Control <sup>2</sup>	
	Aqueous	Food <sup>3</sup>	Aqueous	Food <sup>3</sup>
40	1.2(0.2) <sup>4</sup>	2.0(0.4)	1.4(0.2)	1.8(0.3)
72	3.0(0.3)	2.9(0.4)	2.6(0.3)	3.0(0.2)
130	5.9(0.6)	3.5(0.4)**	5.0(0.3)	3.9(0.3)*
233	8.3(0.6)	5.9(0.5)**	8.0(0.5)	6.7(0.5)
420	11.7(0.4)	9.7(0.4)**	11.1(0.3)	9.4(0.4)**
756	13.8(0.3)	12.6(0.4)*	13.3(0.2)	12.2(0.4)*

<sup>1</sup>Maximum estimate=15 cm.

<sup>2</sup>n=24.

<sup>3</sup>Strained peas.

<sup>4</sup>Mean(standard error of the mean).

\*. \*\* Significant at  $p \leq 0.05$ ,  $p \leq 0.01$ , respectively.

Table 11. Mean intensity estimates (cm)<sup>1</sup> of concentrations of sucrose in aqueous and food systems.

Sucrose (mM)	Cancer <sup>2</sup>		Control <sup>2</sup>	
	Aqueous	Food <sup>3</sup>	Aqueous	Food <sup>3</sup>
40	1.3(0.2) <sup>4</sup>	3.7(0.6)***	1.2(0.2)	4.2(0.5)***
72	2.5(0.2)	4.6(0.5)***	2.5(0.2)	4.8(0.5)***
130	4.8(0.4)	5.7(0.6)	4.3(0.3)	6.7(0.5)***
233	7.9(0.5)	7.4(0.5)	7.2(0.4)	7.1(0.5)
420	11.0(0.5)	9.6(0.5)*	10.3(0.4)	9.2(0.4)
756	13.2(0.3)	11.0(0.5)***	12.1(0.4)	10.4(0.5)*

<sup>1</sup>Maximum estimate=15 cm.

<sup>2</sup>n=24.

<sup>3</sup>Applesauce.

<sup>4</sup>Mean(standard error of the mean).

\*, \*\*\* Significant at  $p \leq 0.05$ ,  $p \leq 0.001$ , respectively.

concentration was judged as significantly ( $p \leq 0.05$ ) less sweet in applesauce compared to the aqueous system (Table 8).

## Taste Pleasantness

### Comparison of Cancer and Control Groups

Average pleasantness ratings of salt in aqueous and food systems for the cancer and control groups are given in Table 12. For saltiness in both systems, no significant group differences in pleasantness ratings were observed. Figure 3 displays the mean pleasantness functions for saltiness in each system for both groups. The pleasantness curve for the salt aqueous system for the cancer group showed a peak at 72 mM (-1.14 log M) NaCl, while that for the control group decreased from 40 mM (-1.39 log M) NaCl. For salt in peas, the pleasantness curves for both groups were relatively flat from 40 mM (-1.39 log M) NaCl to 130 mM (-0.89 log M) NaCl and then declined steeply. For both systems, ANOVA of the saltiness pleasantness ratings did not show any significant group effects or group x concentration interaction effects.

For sucrose in aqueous and food systems, group pleasantness ratings did not differ (Table 13). Pleasantness functions for sucrose for the cancer and control groups are shown in Figure 4. For sucrose in the aqueous system, the shape of the pleasantness curve for both groups showed an increase to 130 mM (-0.89 log M) sucrose, followed by a decrease. For sucrose in applesauce, the pleasantness curve for the cancer group exhibited a shallow parabolic shape with the 72 mM (-1.14 log M) and 130 mM (-0.89 log M) sucrose concentrations rated as most pleasant. The pleasantness function for sucrose in food for the control subjects peaked

Table 12. Mean pleasantness ratings (cm)<sup>1</sup> of concentrations of sodium chloride (NaCl) in aqueous and food systems for the cancer and control groups.

NaCl (mM)	Aqueous		Food <sup>2</sup>	
	Cancer <sup>3</sup>	Control <sup>3</sup>	Cancer <sup>3</sup>	Control <sup>3</sup>
40	9.3 (0.8) <sup>4</sup>	9.0 (0.9)	10.0 (0.7)	9.8 (0.7)
72	9.4 (0.7)	8.7 (0.8)	10.4 (0.7)	10.1 (0.6)
130	8.0 (0.6)	7.9 (0.6)	10.3 (0.6)	10.0 (0.6)
233	6.2 (0.6)	5.8 (0.6)	8.9 (0.6)	7.6 (0.7)
420	3.1 (0.5)	3.3 (0.5)	5.0 (0.5)	5.3 (0.6)
756	1.4 (0.3)	1.8 (0.4)	2.4 (0.4)	2.8 (0.5)

<sup>1</sup>Maximum rating=15 cm.

<sup>2</sup>Strained peas.

<sup>3</sup>n=24.

<sup>4</sup>Mean (standard error of the mean).

Figure 3. Pleasantness ratings of sodium chloride in aqueous and food systems

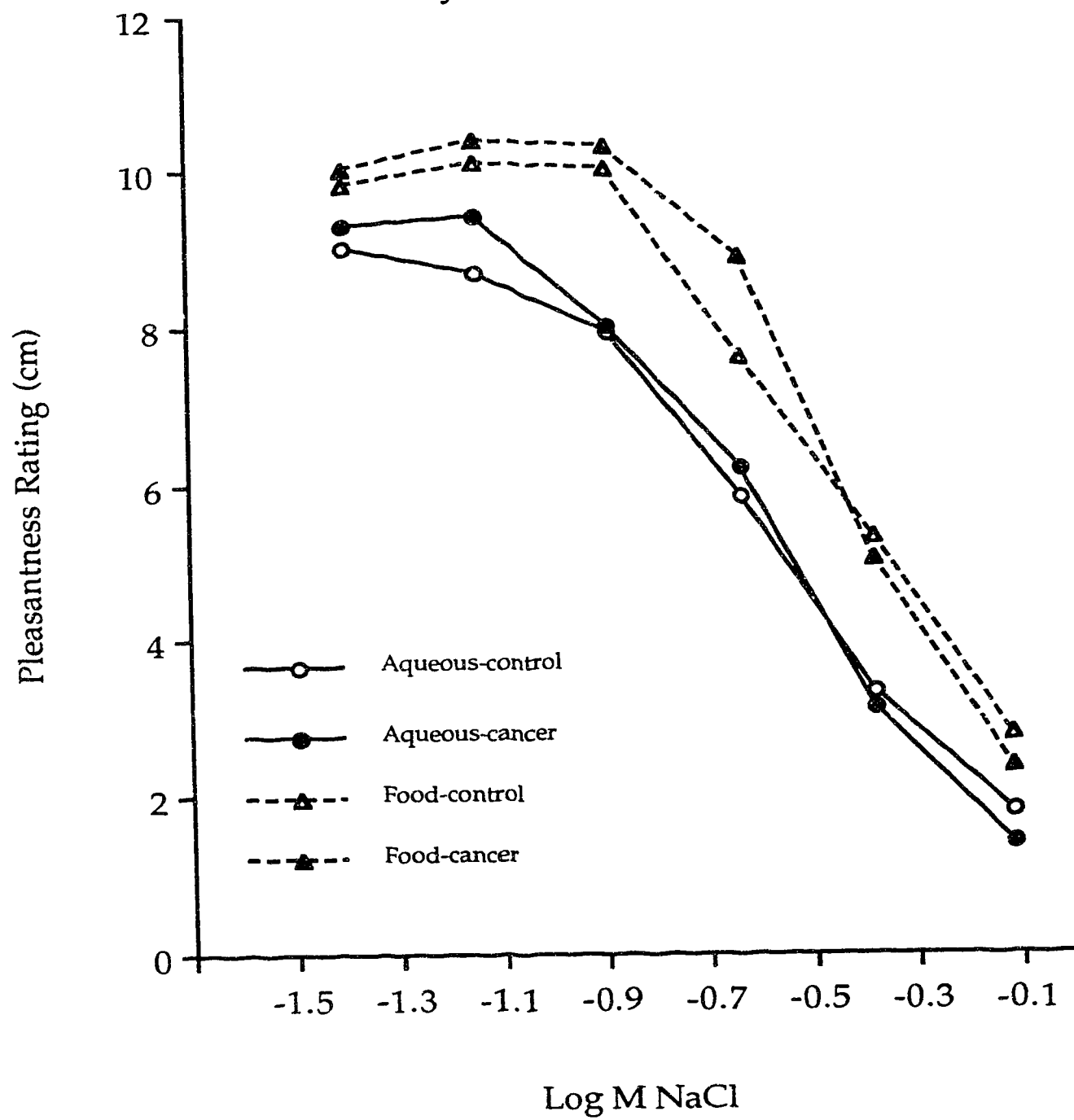


Figure 3. Pleasantness ratings of sodium chloride in aqueous and food systems

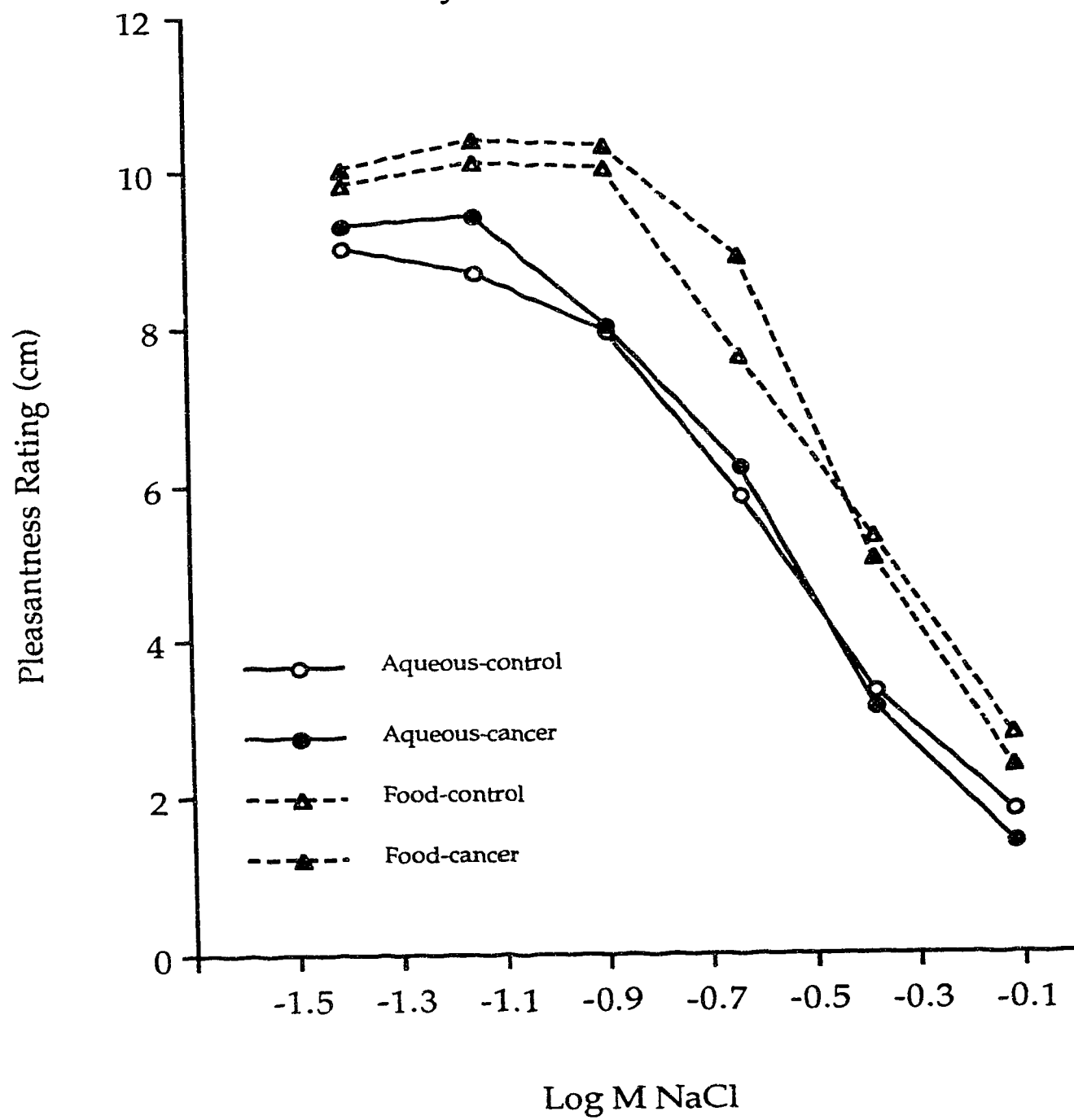
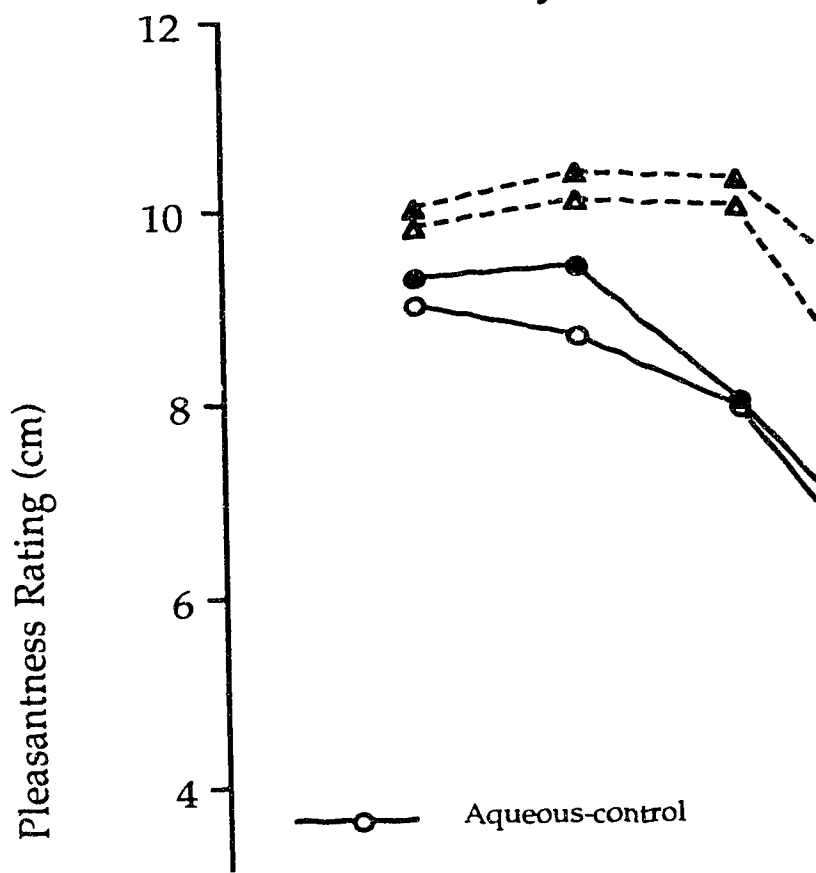


Figure 3. Pleasantness rating of  
chloride in aqueous  
systems



at 233 mM (-0.63 log M) sucrose. For both systems, ANOVA of the sweetness pleasantness ratings did not show any significant group effects or group x concentration interaction effects.

#### Comparison of Aqueous and Food Systems

Pleasantness ratings of salt in aqueous compared to food systems for the cancer and control groups are shown in Table 14. Significant system effects on pleasantness ratings for saltiness for both the cancer ( $p \leq 0.01$ ) and control groups ( $p \leq 0.05$ ) were determined by ANOVA. For both groups, pleasantness ratings for salt concentrations of 130 mM to 420 mM NaCl were significantly higher in peas than in aqueous solution (Table 14).

Table 15 presents the group pleasantness ratings for sweetness of aqueous and food systems. For the cancer group, the pleasantness rating for the 756 mM sucrose concentration in applesauce was significantly ( $p \leq 0.01$ ) higher than that in the aqueous system. The control group judged the 233 mM to 756 mM sucrose concentrations in applesauce as significantly ( $p \leq 0.05$ ) more pleasant than those in aqueous solutions. For sucrose pleasantness ratings, significant ( $p \leq 0.05$ ) system effects were determined by ANOVA for the cancer group only; no significant system x concentration interaction effects were revealed for either study group.

#### Dietary Intake

Table 16 presents the average daily energy intake and calculated basal energy requirement values for the study groups. Mean energy intake of the cancer group (1501 kcal) was significantly ( $p \leq 0.05$ ) lower than that of the controls (1763 kcal). Energy intake as kilocalories per kilogram of desirable body weight was 26 for the cancer group, significantly

Table 14. Mean pleasantness ratings (cm)<sup>1</sup> of concentrations of sodium chloride (NaCl) in aqueous and food systems.

NaCl (mM)	Cancer <sup>2</sup>		Control <sup>2</sup>	
	Aqueous	Food <sup>3</sup>	Aqueous	Food <sup>3</sup>
40	9.3(0.8) <sup>4</sup>	10.0(0.7)	9.0(0.9)	9.8(0.7)
72	9.4(0.7)	10.4(0.7)	8.7(0.8)	10.1(0.6)
130	8.0(0.6)	10.3(0.6)*	7.9(0.6)	10.0(0.6)*
233	6.2(0.6)	8.9(0.6)**	5.8(0.6)	7.6(0.7)*
420	3.1(0.5)	5.0(0.5)**	3.3(0.5)	5.3(0.6)**
756	1.4(0.3)	2.4(0.4)	1.8(0.4)	2.8(0.5)

<sup>1</sup>Maximum rating=15 cm.

<sup>2</sup>n=24.

<sup>3</sup>Strained peas.

<sup>4</sup>Mean(standard error of the mean).

\*, \*\* Significant at  $p \leq 0.05$ ,  $p \leq 0.01$ , respectively.

Table 15. Mean pleasantness ratings (cm)<sup>1</sup> of concentrations of sucrose in aqueous and food systems.

Sucrose (mM)	Cancer <sup>2</sup>		Control <sup>2</sup>	
	Aqueous	Food <sup>3</sup>	Aqueous	Food <sup>3</sup>
40	7.6(0.8) <sup>4</sup>	8.0(0.7)	8.2(1.0)	8.7(0.6)
72	7.8(0.7)	8.5(0.6)	8.6(0.8)	8.7(0.6)
130	8.5(0.5)	8.5(0.7)	9.0(0.6)	8.3(0.5)
233	7.2(0.5)	8.4(0.5)	7.7(0.5)	9.1(0.4)*
420	6.1(0.6)	7.4(0.5)	5.5(0.6)	7.1(0.5)*
756	4.1(0.6)	6.8(0.6)**	4.5(0.7)	6.5(0.6)*

<sup>1</sup>Maximum rating=15 cm.

<sup>2</sup>n=24.

<sup>3</sup>Applesauce.

<sup>4</sup>Mean(standard error of the mean).

\*, \*\* Significant at  $p \leq 0.05$ ,  $p \leq 0.01$ , respectively.

Table 16. Mean energy intakes and basal energy requirements of the cancer and control groups.

	Cancer <sup>1</sup>	Control <sup>1</sup>
Mean Energy Intake		
kcal/day	1501	1763*
kcal/kg/day <sup>2</sup>	26	30**
Calculated BER <sup>3</sup>	1289	1288
Energy Intake		
% of BER	116	137*

<sup>1</sup>n=24.

<sup>2</sup>Based on desirable body weight.

<sup>3</sup>Basal energy requirement for desirable body weight.

\*, \*\* Significant at  $p \leq 0.05$ ,  $p \leq 0.01$ , respectively.

( $p \leq 0.01$ ) lower than 30 for the control group. Total energy intake compared to calculated basal energy requirement was 116% for the cancer group, significantly ( $p \leq 0.05$ ) less than 137% for the control group.

The frequency distributions of relative body weight and energy intake values for the cancer and control groups are shown in Table 17. The frequency distribution of relative body weights for the cancer group was similar to that for the control group. The median frequency class of relative body weights for both groups was 115 - 124%. For energy intake values, a significant ( $p \leq 0.05$ ) group difference in frequency distribution was determined by Kolmogorov-Smirnov analysis. The median frequency class of energy intake for the cancer group was 1300 - 1499 kcal/day while that for the control group was 1700 - 1899 kcal/day.

The average proportions of energy derived from protein, fat and carbohydrate for both groups are listed in Table 18. Energy from protein for the cancer and control groups, respectively averaged 15.2% and 16.4%; from fat, 31.3% and 33.3%; and from carbohydrate, 53.5% and 50.3%. The mean proportion of energy as starch for the cancer group was significantly ( $p \leq 0.05$ ) greater than that for the control group.

Table 19 gives the mean daily nutrient intakes (diet only) of the cancer and control groups. Mean intakes of several nutrients were significantly lower for the cancer subjects than for the controls: energy ( $p \leq 0.05$ ), protein ( $p \leq 0.01$ ), total fat ( $p \leq 0.05$ ), saturated fatty acids ( $p \leq 0.01$ ), monounsaturated fatty acids ( $p \leq 0.05$ ), cholesterol ( $p \leq 0.01$ ), riboflavin ( $p \leq 0.05$ ), preformed niacin ( $p \leq 0.05$ ), ascorbic acid ( $p \leq 0.05$ ), calcium ( $p \leq 0.01$ ), phosphorus ( $p \leq 0.05$ ), iron ( $p \leq 0.05$ ) and zinc ( $p \leq 0.05$ ). For the cancer group the mean intakes of calcium (688 mg) and zinc (7.7

Table 17. Frequency distributions of relative body weight<sup>1</sup> and energy intake values of the cancer and control groups.

Parameter	Cancer		Control	
	Frequency <sup>2</sup>	(%) <sup>3</sup>	Frequency <sup>2</sup>	(%) <sup>3</sup>
Relative body weight (%)				
85 - 94	4	17	2	8
95 - 104	1	4	3	13
105 - 114	6	25	8	33
115 - 124	10	42	7	29
125 - 134	1	4	3	13
135 - 144	1	4	0	0
≥ 145	1	4	1	4
Total	24	100	24	100
Energy intake (kcal/day)*				
≤ 899	1	4	0	0
900 - 1099	1	4	1	4
1100 - 1299	5	21	1	4
1300 - 1499	7	29	3	12
1500 - 1699	4	17	4	17
1700 - 1899	1	4	8	33
1900 - 2099	3	13	3	13
≥ 2100	2	8	4	17
Total	24	100	24	100

<sup>1</sup>Based on desirable body weight.

<sup>2</sup>Number of subjects.

<sup>3</sup>Percent total subjects.

\* Frequency distribution significantly different between groups at  $p \leq 0.05$ .

Table 18. Mean percentage distribution of energy intake values of the cancer and control groups.

Nutrient	Cancer <sup>1</sup>	Control <sup>1</sup>
	% of kcal	% of kcal
Protein	15.2(0.4) <sup>2</sup>	16.4(0.5)
Fat		
Total Fat	31.3(1.2)	33.3(1.0)
Saturated Fat	11.0(0.6)	12.2(0.6)
Polyunsaturated Fat	6.2(0.3)	5.5(0.3)
Monounsaturated Fat	11.2(0.5)	12.1(0.4)
Carbohydrate		
Total Carbohydrate	53.5(1.5)	50.3(1.4)
Sugar	25.3(1.0)	24.0(1.3)
Starch	24.5(1.2)	21.2(1.0)*

<sup>1</sup>n=24.

<sup>2</sup>Mean(standard error of the mean).

\* Significant at  $p \leq 0.05$ .

Table 19. Mean daily nutrient intake (diet only) of the cancer and control groups.

Nutrient	Cancer <sup>1</sup>	Control <sup>1</sup>
Energy (kcal)	1501 ± 76 <sup>2</sup>	1763 ± 67*
Protein (g)	58 ± 4	73 ± 3**
Fat		
Total (g)	53 ± 4	66 ± 4*
Saturated (g)	18.3 ± 1.3	24.0 ± 1.6**
Polyunsaturated (g)	10.6 ± 0.8	11.0 ± 0.8
P/S Ratio	0.60 ± 0.03	0.47 ± 0.03*
Monounsaturated (g)	18.9 ± 1.3	24.2 ± 1.5*
Cholesterol (mg)	204 ± 19	288 ± 22**
Carbohydrate		
Total (g)	204 ± 12	224 ± 9
Dietary Fiber (g)	17 ± 2	18 ± 1
Sugar (g)	94 ± 6	105 ± 6
Starch (g)	92 ± 7	93 ± 5
Thiamin (mg)	1.19 ± 0.07	1.30 ± 0.07
Riboflavin (mg)	1.34 ± 0.09	1.60 ± 0.08*
Preformed Niacin (mg)	14 ± 1	17 ± 1*
Vitamin B6 (mcg)	1.38 ± 0.13	1.50 ± 0.08
Vitamin B12 (mcg)	2.85 ± 0.49	3.32 ± 0.28
Folacin (mcg)	178 ± 13	195 ± 11
Ascorbic Acid (mg)	121 ± 14	168 ± 15*
Vitamin A		
Total (RE)	1037 ± 123	900 ± 67
Total (IU)	7267 ± 863	5792 ± 581
Carotene (IU)	5708 ± 760	4171 ± 566
Preformed Vitamin A (IU)	1588 ± 269	1659 ± 122
Vitamin D (IU)	153 ± 18	162 ± 18
Calcium (mg)	688 ± 59	937 ± 66**
Phosphorus (mg)	1064 ± 73	1321 ± 71*
Iron (mg)	11.7 ± 0.5	13.8 ± 0.6*
Sodium (mg)	1884 ± 149	2216 ± 140
Potassium (mg)	2560 ± 193	2778 ± 136
Zinc (mg)	7.7 ± 0.4	8.9 ± 0.4*

<sup>1</sup>n=24.<sup>2</sup>Mean ± standard error of the mean.

\*, \*\* Significant at p≤0.05, p≤0.01, respectively.

mg) were below the recommended values of 700 and 800 mg for calcium and 9 mg for zinc (Health and Welfare Canada, 1990); the mean intake of folacin was marginal (178 mcg). For the control group, average intakes of all nutrients except zinc, exceeded recommended intakes; zinc intake was marginal (8.9 mg).

Vitamin/mineral supplementation markedly increased the daily intakes of several nutrients (Table 20). Average intakes of calcium and zinc by the cancer group were augmented to values above the recommended levels; the mean folacin intake was increased to 140% of the recommended level. For the controls, zinc intake was increased to 123% of the recommended level. For the cancer group, mean intake values for thiamin, riboflavin, vitamins B6 and B12, and ascorbic acid were 838%, 700%, 1986%, 505% and 496% of the recommended levels, respectively. Corresponding values of these vitamins for the control group were 1025%, 800%, 997%, 630% and 940% of recommended levels.

Mean values for percent risk of nutrient deficiency are shown in Table 21. The cancer group had a significantly ( $p \leq 0.05$ ) higher index of overall nutritional risk than the control group for both diet only and diet plus supplements. Compared to the control group, the cancer group was at significantly greater risk of deficiency for calcium ( $p \leq 0.01$ ) and for iron ( $p \leq 0.05$ ). For the cancer group, the nutrients at greatest risk of deficiency were calcium (43%), folacin (29%), zinc (25%), vitamin B12 (23%), vitamin A (19%), ascorbic acid (9%) and iron (7%). For the control group, nutrients at greatest risk of deficiency were vitamin A (21%), folacin (15%), calcium (14%), zinc (11%) and vitamin B12 (9%).

Table 20. Mean daily nutrient intake (diet plus vitamin/mineral supplements) of the cancer and control groups.

Nutrient	Cancer <sup>1</sup>	Control <sup>1</sup>
Energy (kcal)	1501 $\pm$ 76 <sup>2</sup>	1763 $\pm$ 67*
Thiamin (mg)	6.72 $\pm$ 2.66	8.24 $\pm$ 2.98
Riboflavin (mg)	6.98 $\pm$ 2.65	7.97 $\pm$ 2.85
Preformed Niacin (mg)	24.4 $\pm$ 3.3	37.5 $\pm$ 6.8
Vitamin B6 (mg)	17.28 $\pm$ 10.63	10.97 $\pm$ 5.05
Vitamin B12 (mcg)	10.13 $\pm$ 3.10	12.57 $\pm$ 4.36
Folacin (mcg)	269 $\pm$ 50	329 $\pm$ 48
Ascorbic Acid (mg)	223 $\pm$ 49	423 $\pm$ 130
Vitamin A		
Total (RE)	2134 $\pm$ 651	1327 $\pm$ 143
Total (IU)	9018 $\pm$ 1056	7355 $\pm$ 694
Carotene (IU)	5708 $\pm$ 760	4171 $\pm$ 566
Preformed Vitamin A (IU)	3159 $\pm$ 561	3221 $\pm$ 467
Vitamin D (IU)	293 $\pm$ 44	287 $\pm$ 43
Calcium (mg)	1013 $\pm$ 96	1030 $\pm$ 73
Phosphorus (mg)	1097 $\pm$ 71	1340 $\pm$ 72*
Iron (mg)	14.7 $\pm$ 1.2	19.2 $\pm$ 2.3
Sodium (mg)	1884 $\pm$ 149	2216 $\pm$ 140
Potassium (mg)	2574 $\pm$ 191	2781 $\pm$ 136
Zinc (mg)	9.5 $\pm$ 1.0	11.1 $\pm$ 1.1

<sup>1</sup>n=24.

<sup>2</sup>Mean  $\pm$  standard error of the mean.

\* Significant at  $p \leq 0.05$ .

Table 21. Mean percent risks<sup>1</sup> of nutrient deficiency of the cancer and control groups.

Nutrient	Cancer <sup>2</sup>		Control <sup>2</sup>	
	Diet Only	Diet Plus Supplements (%)	Diet Only (%)	Diet Plus Supplements (%)
Index of Overall Nutritional Risk <sup>3</sup>	14.6 ± 2.8 <sup>4</sup>	9.6 ± 1.8	7.0 ± 2.1 <sup>*</sup>	4.2 ± 1.6 <sup>*</sup>
Protein	6.0 ± 3.2	6.0 ± 3.2	3.3 ± 3.3	3.3 ± 3.3
Thiamin	1.6 ± 0.8	1.0 ± 0.8	0.4 ± 0.3	0.2 ± 0.2
Riboflavin	7.2 ± 3.7	2.2 ± 1.6	1.2 ± 1.1	0.0 ± 0.0
Vitamin B6	1.8 ± 1.8	1.8 ± 1.8	0.9 ± 0.8	0.8 ± 0.8
Vitamin B12	23.0 ± 7.2	11.0 ± 5.3	9.4 ± 5.6	1.4 ± 1.4
Folacin	29.3 ± 7.7	23.3 ± 6.9	15.0 ± 5.8	7.7 ± 4.7
Ascorbic Acid	9.0 ± 5.2	9.0 ± 5.2	0.8 ± 0.7	0.7 ± 0.7
Vitamin A	18.6 ± 6.4	13.7 ± 5.9	21.2 ± 7.2	13.1 ± 6.4
Calcium	43.3 ± 8.6	22.8 ± 7.3	10.9 ± 5.2 <sup>**</sup>	8.4 ± 4.9
Iron	7.4 ± 1.9	5.6 ± 1.8	2.9 ± 0.8 <sup>*</sup>	2.2 ± 0.7
Zinc	24.7 ± 7.0	20.7 ± 6.6	10.6 ± 5.7	8.9 ± 5.6

<sup>1</sup>Microcomputer software package "Probability Assessment of Nutrient Intake" by G.H. Beaton.  
<sup>2</sup>n=24.

<sup>3</sup>Composite average value for 11 nutrients.

<sup>4</sup>Mean ± standard error of the mean.

\*, \*\* Significant at p≤0.05, p≤0.01, respectively between groups (cancer and control) within the same subgroup (diet only or diet plus supplements).

Vitamin/mineral supplementation by both groups reduced the calculated nutrient risk for several nutrients, but not significantly so (Table 21).

Table 22 presents mean nutrient densities for the study groups [nutrient intake (diet only) per 1000 kcal]. For both the cancer and control groups, all mean nutrient densities exceeded those calculated from recommended intakes (Health and Welfare Canada, 1990). Nutrient densities for starch, vitamin A and carotene for the cancer group were significantly ( $p \leq 0.05$ ) greater than those for the control group.

Food consumption patterns, expressed as mean daily intake of food groups are shown in Table 23. For the cancer group, consumption of total meat, poultry, fish and eggs was significantly ( $p \leq 0.01$ ) less than that of the control subjects. Cancer subjects also consumed significantly less fish ( $p \leq 0.05$ ) and citrus fruit ( $p \leq 0.01$ ) than the controls.

#### Relationships Between Taste Perception and Dietary Parameters

There were no differences in salty and sweet suprathreshold taste perception between the cancer and control groups; however, group differences in energy intake and nutritional risk suggest the possibility of relationships between taste perception and diet. Significant, meaningful relationships between taste perception and diet often exist for subjects at nutritional risk; for healthy subjects, relationships between taste perception and diet have not been found (Mattes, 1987). The frequency distributions of energy intake values for the cancer and control subjects revealed that energy intake values for the cancer group were skewed in the direction of low intake. A cancer subgroup with daily energy intakes of less than 1300 kcal ( $n=7$ ) was established (Table 17).

Table 22. Mean daily nutrient intake (diet only) expressed as nutrient density (per 1000 kcal) of the cancer and control groups.

Nutrient	Cancer <sup>1</sup>	Control <sup>1</sup>
Protein (g)	38.7 ± 1.1 <sup>2</sup>	41.7 ± 1.3
Fat		
Total (g)	34.9 ± 1.4	36.7 ± 1.1
Saturated (g)	12.2 ± 0.6	13.5 ± 0.6
Polyunsaturated (g)	6.9 ± 0.3	6.1 ± 0.4
Monounsaturated (g)	12.5 ± 0.6	13.5 ± 0.5
Cholesterol (mg)	136 ± 10	162 ± 9
Carbohydrate		
Total (g)	136.1 ± 3.7	128.4 ± 3.5
Dietary Fiber (g)	11.7 ± 0.9	10.9 ± 0.8
Sugar (g)	63.2 ± 2.4	60.1 ± 3.2
Starch (g)	61.2 ± 3.0	53.1 ± 2.4*
Thiamin (mg)	0.80 ± 0.03	0.74 ± 0.02
Riboflavin (mg)	0.90 ± 0.05	0.91 ± 0.03
Vitamin B6 (mg)	0.92 ± 0.06	0.88 ± 0.06
Vitamin B12 (mcg)	1.9 ± 0.3	1.9 ± 0.2
Folacin (mcg)	120.8 ± 7.7	112.5 ± 6.4
Ascorbic Acid (mg)	83.5 ± 9.8	97.7 ± 10.0
Vitamin A		
Total (RE)	709 ± 84	513 ± 35*
Total (IU)	5016 ± 596	3328 ± 340*
Carotene (IU)	3792 ± 531	2417 ± 343*
Preformed Vitamin A (IU)	1057 ± 183	932 ± 52
Vitamin D (IU)	100 ± 11	91 ± 9
Calcium (mg)	457 ± 32	528 ± 29
Phosphorus (mg)	712 ± 39	748 ± 29
Iron (mg)	8.0 ± 0.3	7.9 ± 0.2
Sodium (mg)	1236 ± 64	1246 ± 63
Potassium (mg)	1720 ± 101	1603 ± 82
Zinc (mg)	5.2 ± 0.2	5.1 ± 0.2

<sup>1</sup>n=24.

<sup>2</sup>Mean ± standard error of the mean.

\* Significant at p≤0.05.

Table 23. Daily intake of food groups by women in this study and in the Nutrition Canada National Survey<sup>1</sup>.

Food Group	Cancer <sup>2</sup>		Control <sup>2</sup>		Nutrition Canada <sup>1</sup>	
	mean intake		mean intake		mean intake	
	(g)	(%) <sup>3</sup>	(g)	(%) <sup>3</sup>	(g)	(%) <sup>3</sup>
Dairy Products						
Total	247 ± 39 <sup>4</sup>	20	310 ± 37	22	225	20
Milk	210 ± 38		262 ± 39			
Cheese	15 ± 4		24 ± 4			
Desserts	22 ± 4		24 ± 6			
MPFE <sup>5</sup>						
Total	92 ± 9	8	128 ± 7**	9	145	12
Meat	47 ± 4		50 ± 6			
Poultry	17 ± 4		28 ± 6			
Fish	16 ± 5		33 ± 5*			
Eggs	12 ± 3		17 ± 3			
Cereal Products						
Total	200 ± 18	16	218 ± 17	15	174	15
Refined	126 ± 17		126 ± 12			
Whole Grain	74 ± 12		92 ± 10			
Fruits/Fruit Products						
Total	310 ± 44	26	381 ± 41	27	239	21
Citrus	13 ± 4		78 ± 20**			
Other	183 ± 32		170 ± 27			
Juice	114 ± 33		133 ± 20			
Vegetables						
Total	257 ± 21	21	244 ± 18	17	215	18
High Ascorbic Acid	39 ± 10		43 ± 6			
High Carotene	89 ± 11		90 ± 14			
High Fiber	28 ± 7		20 ± 4			
Other	101 ± 10		91 ± 9			
Fats and Oils						
Total	22 ± 2	2	25 ± 3	2	20	2
Butter	4 ± 1		6 ± 1			
Margarine	9 ± 2		7 ± 1			
Oils	2 ± 0		3 ± 1			
Other	7 ± 2		9 ± 2			
Nuts	4 ± 1	<1	5 ± 1	<1	10	1
Foods Primarily Sugar	25 ± 3	2	34 ± 7	2	39	3
Miscellaneous <sup>6</sup>	53 ± 8	4	57 ± 10	4	89	8
Total	1210 ± 76	100	1402 ± 60	100	1156	100

<sup>1</sup>Health and Welfare Canada (1977), National group (40-64 years).<sup>2</sup>n=24.<sup>3</sup>Percentage of total intake.<sup>4</sup>Mean ± standard error of the mean.<sup>5</sup>Meat, Poultry, Fish and Eggs.<sup>6</sup>Mixtures of food groups; soups; condiments; unclassified items.

\*, \*\* Significant at p≤0.05, p≤0.01, respectively between study groups.

Only two control subjects had energy intake values of less than 1300 kcal. The cancer subgroup was unusual in that it was comprised of overweight, premenopausal women with inordinately low energy intakes and high nutritional risks. Mean characteristics (SEM) of the cancer subgroup were: age, 52.6 (1.0) years; weight, 73.1 (7.3) kg; relative body weight, 120.3 (4.0) %; body mass index, 26.4 (0.8); daily energy intake, 1113 (54) kcal; index of overall nutritional risk, 25.6 (6.5) %. Although suprathreshold taste perception data did not differ between the cancer subgroup and the control group, the uniqueness of the cancer subgroup warranted investigation of relationships between taste perception and dietary intake for the subgroup. Therefore, relationships between slopes for taste intensity and non-supplemented dietary intake data for the cancer subgroup and the control group were evaluated using correlation and regression analyses.

Table 24 presents the results of Pearson correlation analyses comparing slopes for taste intensity and nutrient intake (diet only). The slope for salt intensity in the aqueous system for the cancer subgroup was positively correlated with energy and vitamin B12 intake. For the control group, the slope for saltiness in the aqueous system was positively correlated with total carbohydrate and sugar intake, and negatively correlated with vitamin B12 intake. For saltiness in food, the slope for salt intensity for the cancer subgroup was positively correlated with folacin intake. For the controls, the slope for salt intensity in food was positively correlated with energy, total carbohydrate, sugar, folacin, ascorbic acid, vitamin A, iron and potassium intake. For sweetness of aqueous systems, the slope for sucrose intensity for the cancer subgroup

Table 24. Pearson correlation coefficients; slope for taste intensity vs nutrient intake (diet only).

Nutrient	Salt (NaCl)						Sucrose					
	Aqueous			Food <sup>1</sup>			Aqueous			Food <sup>1</sup>		
	Cancer Subgroup <sup>2</sup>	Control Group <sup>3</sup>	NS <sup>4</sup>	Cancer Subgroup	Control Group	NS	Cancer Subgroup <sup>2</sup>	Control Group <sup>3</sup>	NS	Cancer Subgroup	Control Group	NS
Energy	0.71*	NS	NS	NS	0.42*	NS	0.68*	NS	NS	NS	NS	NS
Protein	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.35*	NS
Carbohydrate	NS	0.49**	NS	NS	0.65**	NS	NS	NS	NS	NS	NS	NS
Dietary Fiber	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Sugar	NS	0.58**	NS	NS	0.67**	NS	NS	NS	NS	NS	NS	NS
Starch	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Thiamin	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Riboflavin	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.34*	NS
Vitamin B6	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin B12	0.74*	-0.39*	NS	NS	NS	NS	0.69*	NS	NS	NS	NS	NS
Folic acid	NS	NS	NS	0.70*	0.49**	NS	NS	NS	NS	NS	NS	NS
Ascorbic Acid	NS	NS	NS	NS	0.56**	NS	NS	NS	NS	NS	NS	NS
Vitamin A	NS	NS	NS	NS	0.35*	NS	NS	NS	NS	NS	NS	NS
Vitamin D	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.42*	NS
Calcium	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.53**	NS
Phosphorus	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.39*	NS
Iron	NS	NS	NS	NS	0.41*	NS	NS	NS	NS	NS	NS	NS
Sodium	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Potassium	NS	NS	NS	NS	0.57**	NS	NS	NS	NS	NS	NS	NS
Zinc	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

<sup>1</sup>Strained peas for salt; Applesauce for sucrose.

<sup>2</sup>n=7.

<sup>3</sup>n=24.

<sup>4</sup>p≤0.05 required for significance.

\*, \*\* Significant at p≤0.05, p≤0.01, respectively.

was positively correlated with energy and vitamin B12 intake. For sweetness of food for the controls, the slope for sucrose intensity in food was positively correlated with protein, riboflavin, vitamin D, calcium and phosphorus intake.

Table 25 shows the Pearson correlation coefficients for the slope for taste intensity vs percent risk of nutrient deficiency (diet only). For saltiness for the cancer subgroup, the slope for salt intensity in the aqueous system was negatively correlated with percent risk of vitamin B12 deficiency; the slope for salt intensity in food was negatively correlated with the index of overall nutritional risk and percent risks of protein, vitamin B12 and folacin deficiency. For the control group, a significant negative correlation was determined for the slope of saltiness in food vs the percent risk of folacin deficiency. For sweetness for the cancer subgroup, the slope for sucrose intensity in the aqueous system was negatively correlated with percent risks of vitamin B12 and vitamin A deficiency; the slope for sucrose intensity in food was positively correlated with percent risk of iron deficiency. For the controls, the slope for sweetness in food was negatively correlated with percent risks of thiamin and zinc deficiency.

Pearson correlation coefficients for the slope for taste intensity vs nutrient density [nutrient intake (diet only) per 1000 kcal] are given in Table 26. For saltiness for the cancer subgroup, the slope for saltiness in the aqueous system was positively correlated with vitamin B12 density and negatively correlated with iron density. For the controls, the slope for saltiness in the aqueous system was positively correlated with carbohydrate and sugar density and negatively correlated with

Table 25. Pearson correlation coefficients: slope for taste intensity vs percent risk of nutrient deficiency (diet only).

Nutrient	Salt (NaCl)						Sucrose					
	Aqueous			Food <sup>1</sup>			Aqueous			Food <sup>1</sup>		
	Cancer Subgroup <sup>2</sup>	Control Group <sup>3</sup>	Cancer Subgroup	Control Group	Cancer Subgroup	Control Group	Cancer Subgroup <sup>2</sup>	Control Group <sup>3</sup>	Cancer Subgroup	Control Group	Cancer Subgroup	Control Group
Index of Overall Nutritional Risk <sup>4</sup>	NS <sup>5</sup>	NS	-0.73*	NS	NS	NS	NS	NS <sup>4</sup>	NS	NS	NS	NS
Protein	NS	NS	-0.71*	NS	NS	NS	NS	NS	NS	NS	NS	NS
Thiamin	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	-0.39*	NS
Riboflavin	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin B6	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin B12	-0.74*	NS	-0.74*	NS	NS	NS	-0.84**	NS	NS	NS	NS	NS
Folate	NS	NS	-0.80*	-0.38*	NS	NS	NS	NS	NS	NS	NS	NS
Ascorbic Acid	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin A	NS	NS	NS	NS	NS	NS	-0.69*	NS	NS	NS	NS	NS
Calcium	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Iron	NS	NS	NS	NS	NS	NS	NS	NS	NS	0.78*	NS	NS
Zinc	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	-0.43*	NS

<sup>1</sup>Strained peas for salt; Applesauce for sucrose.<sup>2</sup>n=7.<sup>3</sup>n=24.<sup>4</sup>Composite average for 11 nutrients.<sup>5</sup>p<0.05 required for significance.

\*, \*\* Significant at p&lt;0.05, p&lt;0.01, respectively.

Table 26. Pearson correlation coefficients: slope for taste intensity vs nutrient density [nutrient intake (diet only) per 1000 kcal].

Nutrient	Salt (NaCl)						Sucrose					
	Aqueous			Food <sup>1</sup>			Aqueous			Food <sup>1</sup>		
	Cancer	Control	Cancer	Cancer	Control	Control	Cancer	Control	Cancer	Cancer	Control	Control
	Subgroup <sup>2</sup>	Group <sup>3</sup>	Subgroup	Subgroup	Group	Group	Subgroup <sup>2</sup>	Group <sup>3</sup>	Subgroup	Subgroup	Group	Group
Protein	NS <sup>4</sup>	-0.56**	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Carbohydrate	NS	0.39*	NS	NS	0.34*	NS	NS	0.39*	NS	NS	NS	NS
Dietary Fiber	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Sugar	NS	0.50**	NS	NS	0.41*	NS	NS	NS	NS	NS	NS	NS
Starch	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Thiamin	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Riboflavin	NS	-0.34*	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin B6	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin B12	0.69*	-0.52**	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Folacin	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Ascorbic acid	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin A	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Vitamin D	NS	-0.43*	NS	NS	NS	NS	NS	NS	NS	NS	0.35*	NS
Calcium	NS	NS	NS	NS	NS	NS	NS	NS	-0.67*	NS	0.53**	NS
Phosphorus	NS	NS	NS	NS	NS	NS	NS	NS	-0.68*	NS	0.36*	NS
Iron	-0.93**	NS	NS	NS	NS	NS	-0.81*	NS	NS	NS	NS	NS
Sodium	NS	NS	NS	NS	NS	NS	NS	-0.36*	NS	NS	NS	NS
Potassium	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS
Zinc	NS	-0.35*	NS	NS	NS	NS	NS	NS	NS	NS	NS	NS

<sup>1</sup>Strained peas for salt; Applesauce for sucrose.

<sup>2</sup>n=7.

<sup>3</sup>n=24.

\*p≤0.05 required for significance.

\*\*, \*\* Significant at p≤0.05, p≤0.01, respectively.

protein, riboflavin, vitamin B12, vitamin D and zinc density. The slope for saltiness in food for the controls was positively correlated with total carbohydrate and sugar density. For sweetness for the cancer subgroup, the slope for sucrose intensity in the aqueous system was negatively correlated with iron density. For the controls, the slope for sweetness in the aqueous system was positively correlated with total carbohydrate density and negatively correlated with sodium density. The slope for sucrose intensity in food for the cancer subgroup was negatively correlated with calcium and phosphorus density. For the controls, the slope for sucrose intensity in food was positively correlated with vitamin D, calcium and phosphorus density.

Equations for the stepwise multiple regression of nutrient intake (diet only) on the slopes for taste intensity in aqueous and food systems for the cancer subgroup and the control group are presented in Table 27. For saltiness for the cancer subgroup, vitamin B12 intake ( $p \leq 0.10$ ) predicted 56% of the variance in the slope for salt intensity in the aqueous system; folacin intake ( $p \leq 0.10$ ) explained 49% of the variance in the slope for salt intensity in food. For the controls, sugar intake ( $p \leq 0.01$ ) accounted for 33% of the variance in the slope for salt intensity in the aqueous system and for 44% of that in food; ascorbic acid intake ( $p \leq 0.05$ ) predicted an additional 11% of the variance in the slope for salt intensity in food. For sweetness for the cancer subgroup, vitamin B12 ( $p \leq 0.10$ ) intake contributed to 48% of the variance in the slope for sucrose intensity in the aqueous system. For sweetness for the controls, calcium intake ( $p \leq 0.01$ ) accounted for 28% of the variance in the slope for sucrose intensity in food.

Table 27. Stepwise regression equations for slopes for taste intensity (Y), standard error of the estimate (S) and coefficient of multiple determination (R<sup>2</sup>) [nutrient intake (diet only)].

Tastant Group	Aqueous System		Food System <sup>1</sup>	
	Equation <sup>2</sup>	(S) (R <sup>2</sup> )	Equation <sup>2</sup>	(S) (R <sup>2</sup> )
<b>Salt (NaCl)</b>				
<b>Cancer Subgroup<sup>3</sup></b>				
	Y= 5.15 + 2.16(Vit B12 intake)	1.28 0.56	Y= 2.80 + 0.03(Folacin intake)	1.51 0.49
<b>Control Group<sup>4</sup></b>				
	Y= 7.23 + 0.02(Sugar intake)**	1.05 0.33**	Y= 3.08 + 0.03(Sugar intake)** + 0.01(Ascorbic Acid intake)*	1.35 0.55***
<b>Sucrose</b>				
<b>Cancer Subgroup<sup>3</sup></b>				
	Y= 3.24 + 2.99(Vit B12 intake)	2.08 0.48	Y= no variables entered <sup>2</sup>	--- ---
<b>Control Group<sup>4</sup></b>				
	Y= no variables entered <sup>2</sup>	--- ---	Y= 1.17 + 0.01(Calcium intake)**	2.15 0.28**

<sup>1</sup>Strained peas for salt; Applesauce for sucrose.

<sup>2</sup>Variables entered into equations at p<0.10.

<sup>3</sup>n=7.

<sup>4</sup>n=24.

\*, \*\*, \*\*\* Significant at p≤0.05, p≤0.01, p≤0.001, respectively.

Table 28 gives the equations for the stepwise multiple regressions of percent risk of nutrient deficiency (diet only) on the slope for taste intensity for the cancer subgroup and the control group. For saltiness for the cancer subgroup, percent risk of vitamin B12 ( $p \leq 0.05$ ) and thiamin deficiency ( $p \leq 0.10$ ) provided 54% and 26%, respectively of the variance in the slope for salt intensity in the aqueous system; percent risk of folacin deficiency ( $p < 0.05$ ) explained 65% of the variance in the slope for salt intensity in food. For the controls, percent risk of folacin deficiency ( $p \leq 0.10$ ) predicted 14% of the variance in the slope for saltiness in food. For sweetness for the cancer subgroup, percent risk of vitamin B12 deficiency ( $p \leq 0.05$ ) contributed to 71% of the variance in the slope for sucrose intensity in the aqueous system; percent risk of iron ( $p \leq 0.01$ ) and riboflavin ( $p \leq 0.05$ ) deficiency accounted for 61% and 29%, respectively of the variance in the slope for sucrose intensity in food. For the controls, percent risk of zinc deficiency ( $p \leq 0.05$ ) explained 19% of the variance in the slopes for sucrose intensity in food.

Equations for the stepwise multiple regression of nutrient density [nutrient intake (diet only) per 1000 kcal] on slopes for taste intensity for the cancer subgroup and the control group are listed in Table 29. For saltiness for the cancer subgroup, iron ( $p \leq 0.01$ ) and ascorbic acid ( $p \leq 0.10$ ) density predicted 87% and 7% respectively, of the variance in the slope for salt intensity in the aqueous system. For the controls, protein ( $p \leq 0.01$ ) and phosphorus ( $p \leq 0.05$ ) density determined 31% and 12% respectively, of the variance in the slope for salt intensity in the aqueous system; sugar density ( $p \leq 0.05$ ) contributed to 17% of the variance in the slope for salt intensity in food. For sweetness for the cancer

Table 28. Stepwise regression equations for slopes for taste intensity (Y), standard error of the estimate (S) and coefficient of multiple determination (R<sup>2</sup>) [percent risk of nutrient deficiency (diet only)].

Tastant Group	Aqueous System		Food <sup>1</sup>	
	Equation <sup>2</sup>	(S) (R <sup>2</sup> )	Equation <sup>2</sup>	(S) (R <sup>2</sup> )
Salt (NaCl)				
Cancer Subgroup <sup>3</sup>				
	Y=10.18 - 0.06(Vit B12 risk)* + 0.18(Thiamin risk)	0.95 0.80*	Y= 8.81 - 0.04(Folacin risk)*	1.26 0.65*
Control Group <sup>4</sup>				
	Y= no variables entered <sup>2</sup>	---	Y= 8.68 - 0.03(Folacin risk)	1.83 0.14
Sucrose				
Cancer Subgroup <sup>3</sup>				
	Y=10.68 - 0.06(Vit B12 risk)*	1.54 0.71*	Y= 0.27 + 0.43(Iron risk)** - 0.06(Riboflavin risk)*	0.82 0.90*
Control Group <sup>4</sup>				
	Y= no variables entered <sup>2</sup>	---	Y= 5.44 - 0.04(Zinc risk)*	2.30 0.18*

<sup>1</sup>Strained peas for salt; Applesauce for sucrose.

<sup>2</sup>Variables entered into equations at p<0.10.

<sup>3</sup>n=7.

<sup>4</sup>n=24.

\*, \*\* Significant at p≤0.05, p≤0.01, respectively.

Table 29. Stepwise regression equations for slopes for taste intensity (Y), standard error of the estimate (S) and coefficient of multiple determination (R<sup>2</sup>) [nutrient density (nutrient intake per 1000 kcal - diet only)].

1000 kcal - diet only						
Tastant Group	Aqueous System			Food System <sup>1</sup>		
	Equation <sup>2</sup>	(S)	(R <sup>2</sup> )	Equation <sup>2</sup>	(S)	(R <sup>2</sup> )
Salt (NaCl)						
Cancer Subgroup <sup>3</sup>						
	Y=23.29 - 1.59(Iron density)** + 0.01(Ascorbic Acid density)	0.53	0.94**	Y= no variables entered <sup>2</sup>	---	---
Control Group <sup>4</sup>						
	Y=14.59 - 0.22(Protein density)** + 0.01(Phosphorus density)*	1.00	0.43**	Y= 5.23 + 0.05(Sugar density)*	1.80	0.17*
Sucrose						
Cancer Subgroup <sup>3</sup>						
	Y=26.37 - 2.38(Iron density)** + 0.04(Folacin density)*	1.08	0.89"	Y=13.11 - 0.01(Phosphorus density)	1.69	0.46
Control Group <sup>4</sup>						
	Y= 12.14 - 0.01(Sodium density)	2.01	0.13	Y= 0.09 + 0.01(Calcium density)**	2.16	0.30**

<sup>1</sup>Strained peas for salt; Applesauce for sucrose.

<sup>2</sup>Variables entered into equations at p<0.10.

<sup>3</sup>n=7.

<sup>4</sup>n=24.

\*, \*\*, \*\*\* Significant at p≤0.05, p≤0.01, p≤0.001, respectively.

subgroup, iron ( $p \leq 0.01$ ) and folacin ( $p \leq 0.05$ ) density accounted for 66% and 23% respectively, of the variance in the slope for sucrose intensity in the aqueous system; phosphorus density ( $p \leq 0.10$ ) explained 46% of the variance in the slope for sucrose intensity in food. For the controls, sodium density ( $p \leq 0.10$ ) predicted 13% of the variance in the slope for sucrose in the aqueous system while calcium density ( $p \leq 0.01$ ) predicted 46% of the variance in the slope for sucrose intensity in food.

## 5 DISCUSSION

### Taste Perception

In the present study, suprathreshold taste perception did not differ between the breast cancer and control groups. Neither the slopes for taste intensity nor the pleasantness responses for suprathreshold concentrations of salt and sucrose in aqueous and food systems differed significantly between groups. The effects of breast cancer on suprathreshold taste perception have not been reported elsewhere. However, suprathreshold taste perception in subjects with other forms of cancer has received limited attention (Settle et al., 1979; Trant et al., 1982). Trant et al. (1982) found no significant differences in suprathreshold taste intensity or pleasantness responses for salty, sweet, sour and bitter taste modalities in foods between lung and upper gastrointestinal cancer patients. There was no control group for comparison (Trant et al., 1982). Settle et al. (1979) reported that pleasantness responses for suprathreshold saltiness, sweetness, sourness and bitterness of aqueous solutions in cancer patients of mixed etiology and controls were similar.

For the breast cancer group in the present study, the mean slope for salty taste intensity in food ( $8.5 \text{ cm/Log M}$ ) was approximately 1.5x that reported by Trant et al. (1982) for lung and upper gastrointestinal cancer patients. For sweetness in food, the slope for sucrose intensity for the present breast cancer patients ( $6.0 \text{ cm/Log M}$ ) was comparable to that found by Trant et al. (1982) for their combined cancer group.

In the current study, pleasantness functions for saltiness and sweetness in aqueous and food systems for both groups were non-linear.

Lundgren et al. (1978) identified four pleasantness function shapes: monotonic increasing, monotonic decreasing, parabolic and flat. For individual subjects in our cancer and control groups, all four pleasantness function shapes were observed. For the breast cancer group, the pleasantness functions for salt and sucrose in aqueous systems were parabolic. For the control group, the pleasantness curve for salt in the aqueous system decreased with increasing concentrations of NaCl while that for sucrose in the aqueous system was parabolic. Settle et al. (1979) observed monotonic decreasing functions for salt pleasantness and parabolic pleasantness curves for sucrose in aqueous systems for cancer patients of mixed etiology and controls. For the food system in the present study, pleasantness functions for salt and sucrose for the breast cancer and control groups were generally parabolic. Mean parabolic pleasantness curves for salt and sucrose in food were found by Trant et al. (1982) for lung and upper gastro-intestinal cancer patients. However, sweetness pleasantness responses were influenced by chemotherapy and anorexia: no single pleasantness function shape characterized the majority of the cancer patient responses (Trant et al., 1982).

Inter-study comparisons are limited by methodological differences. Settle et al. (1979) determined suprathreshold pleasantness of tastants in aqueous but not food systems. Trant et al. (1982) assessed suprathreshold intensity and pleasantness of tastants in food but not aqueous systems. In the current study and that of Trant et al. (1982) respectively, the tastant carriers for the food systems were: for salt, strained peas and tomato juice; for sucrose, applesauce and cherry beverage. The range of tastant concentrations used in the current study and in those of Trant et

al. (1982) and Settle et al. (1979) also differed. Moreover, Trant et al. (1982) utilized a 10 cm vs the 15 cm line scale used in the present study; Settle et al. (1979) employed a structured category scale. Variables that are known to influence suprathreshold taste perception responses include: the tastant carrier (Riskey et al., 1979; Lawless, 1983; Mattes, 1985), the range of tastant concentration (Pangborn and Pecore, 1982; Mattes, 1985), the choice of scale (Giovanni and Pangborn, 1983) and tumor morphology (Settle et al., 1979; Trant et al., 1982).

In the present study, system-related differences in both taste intensity and pleasantness were observed for the breast cancer and control groups. Slopes for saltiness and sweetness intensity for both groups were significantly higher for the aqueous than for the food system. For salt intensity estimates significant system and system x concentration interaction effects were observed for the cancer and control groups. For saltiness pleasantness ratings, significant system effects were observed for both groups. For sucrose intensity estimates a significant system x concentration interaction effect was observed for both groups while for sucrose pleasantness ratings, a significant system effect was observed for the breast cancer group only. These results support those of others (Pangborn and Trabue, 1967; Pangborn and Pecore, 1982; Murphy, 1986; Gee et al., 1988; Ko, 1988) that have noted that suprathreshold taste perception of a given tastant in an aqueous system differed from that in a food system. Foods rather than aqueous solutions are normally consumed in the diet. Therefore, suprathreshold taste perception of tastants in foods provides more information about the everyday taste experiences of

subjects than does perception of tastants in aqueous systems (Bartoshuk, 1978).

### Dietary Intake

In the present study the mean energy intake of the breast cancer group (1501 kcal) was significantly ( $p \leq 0.05$ ) lower than that of the controls (1763 kcal). The comparable value for daily energy intake from the Nutrition Canada survey report (females 40-64 years) is 1726 kcal (Health and Welfare Canada, 1973). The finding that the cancer group consumed significantly less energy than the control group in this study was surprising. All cancer subjects had Stage I breast malignancy; surgery was the only mode of treatment; and all were free of recurrence. Therefore, no effects of cancer or treatment on dietary intake were expected. Nor were the observed group differences in energy intake attributable to age or anthropometric differences: the cancer patients and control subjects were matched by age and relative body weight. However, similar results have been reported elsewhere (Aldercreutz et al., 1989): mean energy intake of eight mastectomized postmenopausal breast cancer patients (1521 kcal) was significantly ( $p \leq 0.01$ ) lower than that of 10 control women (1799 kcal). An energy intake value of 1504 kcal for 19 postmenopausal breast cancer patients has also been recorded (Boyar et al., 1988).

In the current study, low energy intake for the cancer group resulted in significantly lower intakes of several nutrients for the cancer group compared to the control group. Similar results were reported for postmenopausal mastectomized breast cancer patients compared to

controls (Aldercroutz et al., 1989). Despite the quantitative dietary differences determined for subjects in the present study, the quality of the diets of the cancer and control groups did not differ. When nutrient intakes were expressed per 1000 kcal, the values for the two groups were similar, indicating that they consumed foods of essentially the same nutrient composition. Three interesting exceptions however, were observed: for the cancer group, the values for vitamin A, carotene and starch density were significantly ( $p \leq 0.05$ ) higher than those for the control group. Mean proportions of energy derived from protein, fat and carbohydrate did not differ between groups.

Low energy intake may result in inadequate nutrient intakes (Health and Welfare Canada, 1990). In the present study, mean daily intakes of calcium and zinc by the cancer group were below recommended levels. Averages however, tend to mask the actual range of individual nutrient intakes. The probability approach in evaluating dietary intake data enables the researcher to estimate the prevalence of inadequate nutrient intakes (Anderson et al., 1982). The cancer group was found to be at significantly greater risk of deficiency of calcium ( $p \leq 0.01$ ) and iron ( $p \leq 0.05$ ) than the control group. Moreover, the index of overall nutritional risk for the cancer group (14.6%) was twice ( $p \leq 0.05$ ) that of the controls (7.0%).

Supplement use by the cancer and control subjects did not significantly reduce mean percent risk of deficiency for any nutrient nor the index of overall nutritional risk. This suggests that the subjects at risk of nutrient deficiency were not those taking supplements and/or were taking inappropriate supplements.

Differences in food consumption patterns contributed to differences in energy and nutrient intake for the cancer group compared to the control group. The cancer group consumed significantly smaller amounts of the MFPE food group than did the controls. For women, the MFPE food group is a major source of energy (Health and Welfare Canada, 1977; Randall et al., 1989; Gorbach et al., 1990) and a primary source of protein (Health and Welfare Canada, 1977). The MFPE food group is also a major source of fat (Health and Welfare Canada, 1977; Randall et al., 1989; Buzzard et al., 1990), a significant source of both zinc (Buzzard et al., 1990) and iron (Health and Welfare Canada, 1977) and the only significant source of vitamin B12 (Ellenbogen, 1984) in the diets of women. Total fruit consumption in the present study did not differ between groups. However, cancer subjects consumed significantly less citrus fruit than did the controls. In the diets of women, fruits are primary sources of ascorbic acid (Health and Welfare Canada, 1977; Randall et al., 1989) and folacin (Health and Welfare Canada, 1977).

In the current study, the cancer subgroup is of particular interest. Cancer subgroup selection was based on inordinately low daily energy intakes ( $\leq 1300$  kcal). The cancer subgroup was at greater nutritional risk than the control group: the index of overall nutritional risk for the cancer subgroup was 25.6% compared to 7.0% for the control group. The cancer subgroup was comprised of predominately overweight, premenopausal women. Mean characteristics for the cancer subgroup compared to the control group were: weight, 73.1 vs 65.3 kg; relative body weight, 120.3 vs 113.0%; body mass index, 26.4 vs 24.7; and premenopausal status, 6/7 (86%) vs 12/24 (50%) women. Increased body weight is a risk factor for

breast cancer (de Waard, 1986). Moreover, data indicate that body weight influences breast cancer recurrence (Donegan et al., 1978; Tartter et al., 1981; Eberlein et al., 1985; Lees et al., 1991). A retrospective study (Donegan et al., 1978) of 962 mastectomized breast cancer patients demonstrated that women weighing >59 kg were at greater risk of recurrence than lighter women. This effect was significant for women with negative nodal status only (Donegan et al., 1978). Findings for similar data revealed significantly decreased disease-free survival in women weighing more than 68 kg (Tartter et al., 1981) and 66 kg (Lees et al., 1991) compared to less heavy women. In the former study, the effect of weight was greatest in women with elevated levels of serum cholesterol (Tartter et al., 1981). In the latter study when nodal status and stage of disease were considered, the effect of weight was significant for premenopausal women only (Lees et al., 1991). A large prospective study (Boyd et al., 1981) showed a significant positive effect of both weight and obesity (body mass index) on breast cancer recurrence, especially for subjects with negative nodal status. This effect was particularly strong for postmenopausal women and for premenopausal women  $\geq 45$  years (Boyd et al., 1981). Body mass index was more strongly associated with breast cancer recurrence than was weight (Boyd et al., 1981). Eiberlein et al. (1985), reported a positive association between breast cancer recurrence and body mass index (but not weight) when nodal status was considered. These findings suggest that breast cancer recurrence and survival are related to obesity for premenopausal women (Boyd et al., 1981; Lees et al., 1991) with negative nodal status (Donegan et al., 1978; Boyd et al., 1981).

### Relationships Between Taste Perception and Dietary Parameters

For the breast cancer subgroup and the control group in the present study, significant Pearson correlations were determined between slopes for taste intensity and non-supplemented nutrient intake, percent risk of nutrient deficiency and nutrient density. For the cancer subgroup, significant correlations between slopes for taste intensity and the following nutrient intake indices were observed: energy, vitamin B12 and folacin intake; the index of overall nutritional risk and percent risk of protein, vitamin B12, folacin, vitamin A and iron deficiency; and vitamin B12, iron, calcium and phosphorus density. For the controls, slopes for taste intensity were significantly correlated with energy, protein, total carbohydrate, sugar, riboflavin, vitamin B12, folacin, ascorbic acid, vitamin A, vitamin D, calcium, phosphorus, iron and potassium intake; percent risk of thiamin, folacin and zinc deficiency; and protein, total carbohydrate, sugar, riboflavin, vitamin B12, vitamin D, calcium, phosphorus, sodium and zinc density. The results of the current study contrast with those of Trant et al. (1982). They found no significant correlations between slopes for taste intensity and intake of energy, protein, fat or carbohydrate for lung and upper gastro-intestinal cancer patients (Trant et al., 1982). However, the 24-hour recall method of dietary assessment used by Trant et al. (1982) was inappropriate for correlation analyses (Beaton et al., 1979; Beaton et al., 1983; Gibson, 1987). Intraindividual variation in nutrient intake is such that data for a single 24-hour period is not representative of an individual's usual dietary intake (Beaton et al., 1979; Beaton et al., 1983; Gibson, 1987).

In the present study, stepwise multiple regression analysis was used to describe the relative contributions of nutrient intake variables to the prediction of slopes for taste intensity. In stepwise multiple regression analyses, independent variables (dietary parameters) are entered into the regression equation in the order of the highest partial correlation coefficient between that variable and the dependent variable (slope for taste intensity). The additional percent of total variance accounted for by the independent variable is conditional to the preceding independent variables entered into the equation. Thus for the stepwise regression equations in the present study, the proportion of the variance in the slope for taste intensity provided by a dietary variable was additional to that proportion of the variance provided by other dietary variables in the regression equation.

For the cancer subgroup and the control group, stepwise regression equations were determined for the regression of non-supplemented nutrient intake, percent risk of nutrient deficiency and nutrient density on the slopes for salt and sucrose intensity in aqueous and food systems. For the cancer subgroup, vitamin B12 and folacin intake; percent risk of vitamin B12, thiamin, folacin, iron and riboflavin deficiency; and iron, ascorbic acid, folacin and phosphorus density were important contributors to the variance in slopes for taste intensity. For the controls, sugar, ascorbic acid and calcium intake; percent risk of folacin and zinc deficiency; and protein, phosphorus, sugar, sodium and calcium density contributed to the variance in the slopes for taste intensity.

In the present study, larger proportions of the variance in slopes for taste intensity were predicted by dietary variables for the cancer

subgroup than for the controls. For the cancer subgroup, dietary variables explained 46-94% of the total variance in slope for taste intensity. For the controls, dietary variables explained 13-55% of the total variance in slope for taste intensity. This difference in the proportion of the total variance in slope for taste intensity explained by dietary variables may be due to the high nutritional risk of the cancer subgroup compared to the controls. Research (Murphy, 1986; Mattes, 1987; Ko, 1988; Gee et al., 1988) suggests that taste-diet relationships exist among individuals at nutritional risk. Ko (1988) found that for elderly men, percent risk of riboflavin, folacin, vitamin A and protein deficiency, and the index of overall nutritional risk contributed significantly to the variance in slopes for salt and sour taste intensity. For young men, percent risk of vitamin B12 deficiency explained a significant proportion of the variance in slopes for salt and sour taste intensity (Ko, 1988). Gee et al. (1988) reported that for elderly women, aberrations in slopes for salt and sour intensity were correlated significantly with high nutrient risks. Murphy (1986) observed that elderly subjects with low-normal protein status (based on serum albumin and blood urea nitrogen levels) preferred soups supplemented with higher levels of casein hydrolysate than did subjects with more adequate serum protein values. For subjects with dysgeusia (distorted taste), a decrease in energy and nutrient intake with increased severity of dysgeusia has been noted (Markley et al., 1983; Mattes-Kulig and Henkin, 1985; Mattes et al., 1990). Mattes (1987) observed that college-aged subjects could be classified into categories of nutrient intake based on suprathreshold taste perception measurements. He suggested a possible role for sensory

evaluation in predicting, diagnosing or monitoring the management of nutritionally-based health disorders (Mattes, 1987).

Taste receptors, because of their epithelial nature and rapid turnover rate, are particularly susceptible to nutrient deficiencies (Mattes and Mela, 1988). For the cancer subgroup in the present study, energy intake values did not contribute significantly to the variance in the slopes for salty and sweet taste intensity. However, low energy intake resulted in the low nutrient intakes and high nutrient risks of the cancer subgroup compared to the controls. For the breast cancer subgroup, energy intake, the index of overall nutritional risk and the percent risk of protein deficiency were correlated significantly with slopes for salty taste intensity. In energy insufficiency, protein is diverted to energy production (Shils, 1980). Energy and protein deficiency could inhibit receptor cell turnover in a manner similar to that known to occur in the gastrointestinal mucosa (Schiffman, 1983a).

For the present breast cancer subgroup, the variance in the slopes for suprathreshold salt and sweet taste intensity was predicted by nutrient intake indices for vitamin B12, folacin, thiamin, riboflavin, ascorbic acid, iron and phosphorus. Folacin and vitamin B12 are required for DNA synthesis and cellular reproduction (Sandstead, 1980). Therefore, deficiencies of either nutrient could retard taste receptor renewal. Furthermore, the availability of folacin for DNA synthesis is vitamin B12-dependent (Sandstead, 1980). A deficiency of vitamin B12 could result in a secondary deficiency of folacin. Neurological deficits resulting from clinical deficiency of vitamin B12 (Sandstead, 1980) or thiamin (Neal and Sauberlich, 1980) could inhibit neural transmission of information from

the taste receptor cell. Clinical manifestations of riboflavin deficiency in man are stomatitis and glossitis (Horwitt, 1980) which are characterized by atrophy of the lingual epithelium (Beutler, 1980). In animals, experimental deficiencies of both riboflavin and folate have resulted in glossitis (Afonsky, 1960). Iron deficiency in adults, especially women over the age of 40 years, has also been associated with glossitis and lingual atrophy (Beutler, 1980). However, poor intake of several nutrients in addition to iron likely contributed to the condition (Beutler, 1980). Ascorbic acid deficiency is associated with scorbutic deterioration of the teeth and gums (Jaffe, 1984). The health of the oral cavity of man has been found to influence taste perception (Langan and Yearick, 1976; Hyde et al., 1981). Phosphorus is essential for carbohydrate, fat and protein metabolism and is a cofactor in a multitude of enzyme reactions (Avioli, 1980). Phosphorus is also a structural component of DNA and RNA. Although rare, a deficiency of phosphorus could be expected to impair the cellular function and regeneration of taste receptors.

It is interesting to note that several of the dietary variables that were important predictors of taste intensity perception are known to influence immune function. Deficiencies of vitamin B12, folacin and iron depress immune responses in man (Beisel et al., 1981). Ascorbic acid deficiency may also impair immune function (Jaffe, 1984). The relationships of these nutrients to taste perception, taste receptor integrity and immunocompetence warrants further investigation of the relationships between taste perception and dietary intake. Since optimal nutritional health is required for maintenance of overall health and

because nutritional deficiencies are generally reversible, identification of breast cancer patients with nutritional problems is imperative. For breast cancer patients, it may be possible to use taste intensity perception data to identify subgroups of patients with nutritional problems.

In summary, results of the present study show that for breast cancer patients, energy and nutrient intakes were significantly lower and nutrient risks were significantly higher than those of control subjects. For a subgroup (n=7) of cancer patients with daily energy intakes of  $\leq 1300$  kcal, suprathreshold taste intensity perception was significantly influenced by dietary parameters. In particular, nutrient intake indices for vitamin B12, folacin, thiamin, riboflavin, ascorbic acid, iron, and phosphorus were important predictors of the slopes for suprathreshold salty and sweet taste intensity for the cancer subgroup. For some breast cancer patients, suprathreshold taste intensity data may be useful to identify nutritional problems.

## 6 SUMMARY AND CONCLUSIONS

A case-control study was conducted to quantitatively assess and compare suprathreshold taste perception and nutrient intake between women with and without breast cancer. Relationships between taste perception and nutrient intake were examined.

Two groups of women aged 44-56 years participated in the present study: 24 mastectomized breast cancer outpatients and 24 matched control subjects. Breast cancer subjects were obtained through the Northern Alberta Breast Cancer Registry. All cancer subjects had Stage I breast malignancy; surgery was the only mode of treatment; and all were free of recurrence. Breast cancer and control subjects were matched by age and relative body weight. For the cancer and control groups, respectively, mean ages were 50.8 and 49.2 years; mean relative body weights were 113.7 and 113.0%, respectively.

Suprathreshold intensity and pleasantness of saltiness and sweetness of aqueous and food systems were evaluated by unstructured category line scaling. Tastants were: NaCl for the salty and sucrose for the sweet taste modality, respectively. Six suprathreshold concentrations of NaCl and sucrose (40, 72, 130, 233, 420 and 756 mM) were evaluated in aqueous solutions and simple foods (strained peas for NaCl and applesauce for sucrose, respectively).

Slopes for salty and sweet taste intensity in aqueous and food systems did not differ between the breast cancer and control groups. Pleasantness responses for suprathreshold concentrations of NaCl and sucrose in aqueous and food systems did not differ between the breast cancer and control groups.

System-related differences in suprathreshold salty and sweet taste intensity estimates and pleasantness ratings were observed in the present study. The slopes for saltiness intensity in the aqueous system were 10.3 for the breast cancer patients and 9.8 for the control group. The slopes for saltiness intensity were significantly ( $p \leq 0.01$ ) higher than those for the food system, 8.5 and 8.3 for the breast cancer and control groups, respectively. The slopes for sweetness intensity in the aqueous system were 9.9 for the breast cancer group and 9.0 for the control group. The slopes for sweetness intensity were significantly ( $p \leq 0.001$ ) higher than those for the food system, 6.0 and 5.0 for the breast cancer and control groups, respectively. For salt intensity estimates, significant system ( $p \leq 0.05$ ) and system x concentration interaction ( $p \leq 0.001$ ) effects were observed for both the breast cancer and control groups. For both groups, mid and high concentrations of NaCl in aqueous systems were judged as more salty, while low concentrations were judged as equally salty compared to food systems. For sweetness intensity estimates, significant ( $p \leq 0.001$ ) system x concentration interaction effects were observed for the cancer and control groups. For both groups for sweetness in the aqueous system, low concentrations of sucrose were judged as less sweet while high concentrations were judged as more sweet than those in the food system. For pleasantness ratings, significant system effects for the salty taste modality were determined for the cancer ( $p \leq 0.01$ ) and control ( $p \leq 0.05$ ) groups. For both groups, low and high concentrations of NaCl in the aqueous system were equally pleasant while mid concentrations were less pleasant compared to those in the food system. For sweetness pleasantness scores, a system effect ( $p \leq 0.05$ ) was observed for the breast cancer group

only. However for both groups, high concentrations of sucrose were less pleasant while low concentrations were equally pleasant in aqueous compared to food systems. Because foods rather than aqueous solutions are consumed in the diet, suprathreshold taste perception of tastants in foods is more relevant to the normal taste experience of subjects than is that in aqueous solutions.

Dietary intake for four days was assessed for each subject using a combination of 24-hour recall (one day) and food records (three days). Dietary intakes for three weekdays and one weekend day were assessed.

The mean daily energy intake of the breast cancer group (1501 kcal) was significantly ( $p \leq 0.05$ ) lower than that of the controls (1763 kcal). Compared to the control group, the breast cancer group consumed significantly less of the following nutrients: protein ( $p \leq 0.01$ ), total fat ( $p \leq 0.05$ ), saturated fatty acids ( $p \leq 0.01$ ), monounsaturated fatty acids ( $p \leq 0.05$ ), cholesterol ( $p \leq 0.01$ ), riboflavin ( $p \leq 0.05$ ), preformed niacin ( $p \leq 0.05$ ), ascorbic acid ( $p \leq 0.05$ ), calcium ( $p \leq 0.01$ ), phosphorus ( $p \leq 0.05$ ), iron ( $p \leq 0.05$ ) and zinc ( $p \leq 0.05$ ). For the breast cancer group, mean intakes of calcium (688 mg) and zinc (7.7 mg) were below the recommended values.

The index of overall nutritional risk was significantly higher ( $p \leq 0.05$ ) for the breast cancer (14.6%) than for the control (7.0%) group. The nutrients at greatest risk of deficiency for the breast cancer group were: calcium, folacin, zinc, vitamin B12, vitamin A, ascorbic acid and iron. The nutrients at greatest risk of deficiency for the controls were: vitamin A, folacin, calcium, zinc and vitamin B12. Compared to the

control group, the breast cancer group was at significantly greater risk of calcium ( $p \leq 0.01$ ) and iron ( $p \leq 0.05$ ) deficiency.

Food consumption patterns differed between the study groups. The cancer group consumed significantly ( $p \leq 0.01$ ) lower amounts of the MPFE food group than did the control subjects. Cancer subjects also consumed significantly less fish ( $p \leq 0.05$ ) and citrus fruit ( $p \leq 0.01$ ) than did the controls.

The frequency distribution of energy intake values of the breast cancer group was skewed in the direction of low intake, revealing a subgroup of breast cancer subjects ( $n=7$ ) with daily energy intakes of less than 1300 kcal (average energy intake, 1113 kcal). The cancer subgroup was unusual in that it was comprised of overweight, premenopausal women with inordinately low energy intakes and high nutritional risks. Mean characteristics for the cancer subgroup compared to the control group were: age, 52.6 vs 49.2 years; weight, 73.1 vs 65.3 kg; relative body weight, 120.3 vs 113.0%; body mass index, 26.4 vs 24.7; and premenopausal status, 6/7 (86%) vs 12/24 (50%) subjects. The index of overall nutritional risk for the cancer subgroup was 25.6% compared to 7.0% for the controls.

Although there were no differences in salty and sweet suprathreshold taste perception between the breast cancer subgroup and the control group, relationships between taste perception data and nutrient intake indices were explored by correlation analyses. Stepwise multiple regression was used to describe the relative importance of the nutrient intake indices in predicting slopes for salty and sweet taste intensity.

For the cancer subgroup, significant correlations were revealed between slopes for taste intensity and the following nutrient intake indices: energy, vitamin B12 and folacin intake; the index of overall nutritional risk and percent risk of protein, vitamin B12, folacin, vitamin A and iron deficiency; and vitamin B12, iron, calcium and phosphorus density. For the controls, slopes for taste intensity were significantly correlated with energy, protein, total carbohydrate, sugar, riboflavin, vitamin B12, folacin, ascorbic acid, vitamin A, vitamin D, calcium, phosphorus, iron and potassium intake; percent risk of thiamin, folacin and zinc deficiency; and protein, total carbohydrate, sugar, riboflavin, vitamin B12, vitamin D, calcium, phosphorus, sodium and zinc density.

For the cancer subgroup, dietary variables accounted for 46-94% of the total variance in slope for taste intensity. For the control group, dietary variables accounted for 13-55% of the total variance in slope for taste intensity. For the cancer subgroup, vitamin B12 and folacin intake; percent risk of deficiency of vitamin B12, thiamin, folacin, iron and riboflavin deficiency; and iron, ascorbic acid, folacin and phosphorus density were important predictors of slopes for taste intensity. For the controls, sugar, ascorbic acid and calcium intake; percent risk of folacin and zinc deficiency; and protein, phosphorus, sugar, sodium and calcium density contributed to the variance in the slopes for taste intensity.

In summary, results of the present study show that for breast cancer patients, energy and nutrient intakes were significantly lower and nutrient risks were significantly higher than those of control subjects. For a subgroup (n=7) of cancer patients with daily energy intakes of  $\leq 1300$

kcal, suprathreshold taste intensity perception was influenced significantly by dietary parameters. In particular, nutrient intake indices for vitamin B12, folacin, thiamin, riboflavin, ascorbic acid, iron, and phosphorus were important predictors of the slopes for suprathreshold salty and sweet taste intensity for the cancer subgroup.

In conclusion, the strong relationships between taste intensity perception and dietary intake observed in the present study suggest that for some breast cancer patients, suprathreshold taste intensity data may be useful to identify nutritional problems. However, further research is required to confirm the usefulness of taste intensity perception data in such an application. In particular, the relationships between taste perception and dietary data need to be evaluated in other, larger populations of breast cancer patients to ensure that subgroups of patients with nutritional problems can be identified. In addition, further investigation of the prevalence and severity of alterations in suprathreshold taste perception in breast and other cancers, treated or untreated, and how they relate to dietary intake is required. Future study of taste perception should utilize food rather than aqueous systems to ensure that the taste perception data is relevant to the usual taste experiences of the subjects.

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**APPENDIX**

## Appendix 1. Letter to Physician.

Dear Dr. \_\_\_\_\_:

Further to our recent telephone call, this is to indicate that the study we are currently conducting is entitled "Taste Perception, Diet, and Breast Cancer: Comparison of Women With and Without Breast Cancer." Ms. (or Mrs.) \_\_\_\_\_, one of your patients, has qualified as a potential study participant.

This is to confirm that your permission to approach Ms. (or Mrs.) \_\_\_\_\_ to enter this study was granted.

Thank you for your assistance.

Sincerely,

H. Ames, M. Gee, and Z. Hawrysh

## Appendix 2. Subject Profile Questionnaire. Part I.

Subject code no: \_\_\_\_\_ Date: \_\_\_\_\_

I. Health Status

1. Would you say that your health in general is

Very good \_\_\_\_\_ Good (average) \_\_\_\_\_ Poor \_\_\_\_\_

2. Have you had any medical condition within the past five years which has caused changes in diet or changes in exercise and activity patterns?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify \_\_\_\_\_

3. Have you had a medical checkup in the last year?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify \_\_\_\_\_

4. Have you had any illnesses in the past year?

Yes \_\_\_\_\_ No \_\_\_\_\_

Specify:	anemia _____	kidney _____
	diabetes _____	infections _____
	liver _____	colds _____
	heart _____	psychological _____
		illness _____
	allergy _____	G.I. _____
	other (specify) _____	

5. In the past year, has your weight varied by five pounds or more either up or down?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify: a) How much?  
 b) Any significant reason? (eg. diet, illness)

6. Have you followed any type of a weight-reducing diet within the past year?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify: a) Name of the diet.  
 b) Duration.

## Appendix 2. Subject Profile Questionnaire. Part II.

## 7. How would you describe your appetite?

Very good \_\_\_\_\_ Good (average) \_\_\_\_\_ Poor \_\_\_\_\_

Has your appetite changed during the past year?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify \_\_\_\_\_

## 8. During the past six months, have you regularly used any of the following medications:

<u>Medication</u>	<u>When Taken</u>	<u>Duration</u>
a) diuretics		
Aldactone	_____	_____
Lasix	_____	_____
Furosemide	_____	_____
other _____	_____	_____
b) antihypertensives		
Adalat PA 20	_____	_____
Capoten	_____	_____
other _____	_____	_____
c) vasodilators		
Isordil	_____	_____
Nitroglycerine	_____	_____
other _____	_____	_____
d) antirheumatics/anti-inflammatory agents		
Indocid	_____	_____
Motrin	_____	_____
Apo-ibuprofen	_____	_____
other _____	_____	_____
e) analgesics		
Anacin	_____	_____
Aspirin	_____	_____
Motrin	_____	_____
Ibuprofen	_____	_____
Panadol	_____	_____
other _____	_____	_____
f) antihistamines		
Actifed	_____	_____
Benadryl	_____	_____
Dimetapp	_____	_____
Hismanal	_____	_____
other _____	_____	_____

continued...

## Appendix 2. Part II. (continued)

- g) antimicrobial agents (antibiotics)  
specify \_\_\_\_\_
- h) antithyroid agents  
Propyl-thyracil \_\_\_\_\_  
other \_\_\_\_\_
- i) hypoglycemics  
specify \_\_\_\_\_
- j) estrogens  
specify \_\_\_\_\_
- k) anticoagulants  
Digoxin \_\_\_\_\_  
other \_\_\_\_\_
- l) other \_\_\_\_\_
9. Do you experience any problems with taste?  
Yes \_\_\_\_\_ No \_\_\_\_\_  
Please specify \_\_\_\_\_
10. Do you experience any problems with the sense of smell?  
Yes \_\_\_\_\_ No \_\_\_\_\_  
Please specify \_\_\_\_\_
11. Do you have any biting, chewing or swallowing difficulties that  
would interfere with eating?  
Yes \_\_\_\_\_ No \_\_\_\_\_  
Please specify \_\_\_\_\_
12. Do you avoid eating foods for the following reasons?
- a) food allergies  
Yes \_\_\_\_\_ No \_\_\_\_\_  
Please specify \_\_\_\_\_
- b) Foods causing discomfort?  
Yes \_\_\_\_\_ No \_\_\_\_\_  
Please specify \_\_\_\_\_

continued...

## Appendix 2. Part II. (continued)

## c) Foods disliked?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify \_\_\_\_\_

## 13. Are you on a modified salt intake?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify \_\_\_\_\_

## 14. Do you add salt to your food at the table?

Yes \_\_\_\_\_ No \_\_\_\_\_

Please specify \_\_\_\_\_

## 15. With whom are your meals usually eaten?

Alone \_\_\_\_\_ Spouse \_\_\_\_\_ Friend \_\_\_\_\_ Family/relatives \_\_\_\_\_

## 16. Who usually prepares your food?

Self \_\_\_\_\_ Spouse \_\_\_\_\_ Other \_\_\_\_\_

## 17. Do you regularly miss any meals?

Yes \_\_\_\_\_ No \_\_\_\_\_

What is your regular meal pattern?

## 18. Are you taking any vitamin/mineral supplements?

Yes \_\_\_\_\_ No \_\_\_\_\_

a) Brand?

b) How often?

c) How many? Each day? Each week?

d) Were they prescribed by a physician?

## 19. Do you drink alcoholic beverages?

Yes \_\_\_\_\_ No \_\_\_\_\_

a) Usual type of beverage?

b) Number of drinks per day? Per week?

continued...

## Appendix 2. Part II. (continued)

20. Have you ever smoked?

Yes \_\_\_\_\_ No \_\_\_\_\_

- a) Brand?
- b) Number per day?
- c) How long ago did you quit?

II. Demographic Information1. What is the highest level of education you have completed?  
(Check all that apply)

- \_\_\_\_\_ elementary school
- \_\_\_\_\_ some high school
- \_\_\_\_\_ high school graduate
- \_\_\_\_\_ career training (trades, business school, armed forces)
- \_\_\_\_\_ some university
- \_\_\_\_\_ university degree
- \_\_\_\_\_ no formal training

2. What is your marital status?

- |                              |               |
|------------------------------|---------------|
| _____ single (never married) | _____ married |
| _____ divorced/separated     | _____ widowed |

3. What type of dwelling do you live in?

- |                          |                       |
|--------------------------|-----------------------|
| _____ single family home | _____ duplex          |
| _____ condominium        | _____ apartment       |
| _____ rented room        | _____ other (specify) |

4. What is your present yearly income? (self and spouse)

- |                           |                           |
|---------------------------|---------------------------|
| _____ ≤ \$19,999          | _____ \$40,000 - \$49,999 |
| _____ \$20,000 - \$29,999 | _____ \$50,000 - \$59,999 |
| _____ \$30,000 - \$39,999 | _____ ≥ \$60,000          |

Appendix 3. Consent Form (Breast Cancer Subjects). Part I.

Title: Taste Perception, Diet, and Breast Cancer: Comparison of Women With and Without Breast Cancer.

Cancer patients often have nutritional problems. I understand that the purpose of this study, in which I have been invited to participate, is to evaluate and compare taste perception and dietary intake data of women (45-55 yrs) with and without breast cancer. My physician has been contacted and has agreed that I may be approached and asked to participate in this study.

I understand that if I agree to participate in this study, I will be visited six times by a researcher, either in my home or at the Cross Cancer Institute. Each visit will require about one hour. I understand that I will be asked on the initial visit to complete a 15 minute questionnaire about my eating habits, general health and socioeconomic status. I understand that I will be measured for height, weight, and arm skinfold thickness. I understand that during my first and subsequent visits, I will be asked to:

1. taste nine (9) samples each of either water and pureed peas with varying amounts of added salt, or water and applesauce with varying amounts of added sugar.
2. rate the samples according to:
  - a) how salty or sweet they are.
  - b) how pleasant each sample is to me.
3. provide information about all the food and beverages, medications, and supplements that I have recently consumed. I understand that I will be asked to provide this information on three (3) to six (6) occasions.

I understand that I will be asked to taste salty and sweet samples on three (3) to six (6) occasions and that each sample will be tasted a total of three times. I understand that the acidity of my saliva will be measured before each tasting session by placing a small strip of paper that indicates acidity (pH paper) in my mouth.

I understand that I will be involved in this study for approximately three (3) weeks. I have discussed the above information with the Foods and Nutrition Researcher and have had any questions that I have asked, answered to my satisfaction. The name of the graduate student conducting this research is Holly Ames (ph: 492-7674) and I understand that I am free to contact her at any time to discuss any questions concerning this study. The names of the Project Supervisors from the Department of Foods and Nutrition, University of Alberta are: M. Gee (ph: 492-5031) and Z. Hawrysh (ph: 492-3830).

\_\_\_\_\_ Subject's Initials

continued...

## Appendix 3. Part I. (continued)

Title: Taste Perception, Diet, and Breast Cancer: Comparison of Women With and Without Breast Cancer.

I understand that I have no obligation to consent to enter this study. I understand that I am free to withdraw from the study at any time for any reason without prejudice to my care.

I understand that records and documents relating to me are confidential and that no information will be released or printed that would expose my personal identity. I will not be identifiable as an individual in any report resulting from this study.

With full knowledge of the above information, I agree to participate in this study.

---

Signature of Subject

---

Signature of Researcher

---

Signature of Witness

---

Date

## Appendix 3. Consent Form (Control Subjects). Part II.

Title: Taste Perception, Diet, and Breast Cancer: Comparison of Women With and Without Breast Cancer.

I understand that the purpose of this study, in which I have been invited to participate, is to evaluate and compare taste perception and dietary intake data of women (45-55 yrs) with and without breast cancer.

I understand that if I agree to participate in this study, I will be visited six times by a researcher, either in my home or at the Foods and Nutrition Department, University of Alberta. Each visit will require about one hour. I understand that I will be asked on the initial visit to complete a 15 minute questionnaire about my eating habits, general health and socioeconomic status. I understand that I will be measured for height, weight, and arm skinfold thickness. I understand that during my first and subsequent visits, I will be asked to:

1. taste nine (9) samples each of either water and pureed peas with varying amounts of added salt, or water and applesauce with varying amounts of added sugar.
2. rate the samples according to:
  - a) how salty or sweet they are.
  - b) how pleasant each sample is to me.
3. provide information about all the food and beverages, medications and supplements that I have recently consumed. I understand that I will be asked to provide this information on three (3) to six (6) occasions.

I understand that I will be asked to taste salty and sweet samples on three (3) to six (6) occasions and that each sample will be tasted a total of three times. I understand that the acidity of my saliva will be measured before each tasting session by placing a small strip of paper that indicates acidity (pH paper) in my mouth.

I understand that I will be involved in this study for approximately three (3) weeks. I have discussed the above information with the Foods and Nutrition Researcher and have had any questions that I have asked, answered to my satisfaction. The name of the graduate student conducting this research is Holly Ames (ph: 492-7674) and I understand that I am free to contact her at any time to discuss any questions concerning this study. The names of the Project Supervisors from the Department of Foods and Nutrition, University of Alberta are: M. Gee (ph: 492-5031) and Z. Hawrysh (ph: 492-3830).

\_\_\_\_\_Subject's Initials

continued...

## Appendix 3. Part II. (continued)

Title: Taste Perception, Diet, and Breast Cancer: Comparison of Women  
With and Without Breast Cancer.

I understand that I have no obligation to consent to enter this study. I understand that I am free to withdraw from the study at any time for any reason without prejudice to my care.

I understand that records and documents relating to me are confidential and that no information will be released or printed that would expose my personal identity. I will not be identifiable as an individual in any report resulting from this study.

With full knowledge of the above information, I agree to participate in this study.

---

Signature of Subject

---

Signature of Researcher

---

Signature of Witness

---

Date

Appendix 4. Analytical Composition of Windsor<sup>R</sup> Table Salt.

<u>Chemical Analysis</u> <sup>1</sup>	<u>Typical</u>	<u>Limits</u>
Calcium Sulphate ( $\text{CaSO}_4$ )	0.16%	0.4% max
Calcium Chloride ( $\text{CaCl}_2$ )	0.04%	0.4% max
Magnesium Chloride ( $\text{MgCl}_2$ )	0.002%	0.4% max
Filter Pad - APHA Test	0.10mg	0.3mg max
Iron (Fe)	1.0ppm	2.0ppm max
Copper (Cu)	0.5ppm	1.0ppm max
Moisture ( $\text{H}_2\text{O}$ )	0.03%	0.1% max
Net Salt - Dry basis ( $\text{NaCl}$ )	99.8%	99.6% min
Added:		
Yellow prussiate of soda anti-caking agent	3.0ppm	13.0ppm max
Zeolex, free running agent	0.6%	1.0% max
Potassium iodide ( $\text{KCl}$ )	0.013%	0.010% min
Invert sugar, iodide stabilizer	0.02%	

<sup>1</sup>data provided by the Canadian Salt Company Ltd., Windsor<sup>R</sup>, August, 1985.

7. 10. 1944

DATE:

AQUEOUS TASTANT

STANDARD SAMPLE ORDER:

REFERENCE SAMPLE "R":

SAMPLE NO.

least salty

most salty

least pleasant

most pleasant

least salty

most salty

least pleasant

most pleasant

least salty

most salty

least pleasant

most pleasant

least salty

most salty

least pleasant

most pleasant

least salty

most salty

least pleasant

most pleasant

least salty

most salty

least pleasant

Most pleasant

<sup>1</sup>Not to scale: minimum score (least intense/pleasant)=0 cm; maximum score (most intense/pleasant)=15 cm.



#### Appendix 7. Food Record Form.

Please use the following sheets to record everything that you eat or drink for the specified day. Eat as you normally would if you were not keeping this record. We suggest that you write the information down while eating or just after you finish eating since meals are difficult to recall in detail later.

At the top of each page please write the date of the day for which you are providing the information. Use as many of the following pages as you need to record your meals. In the first column list the time of day the food or beverage was consumed. In the second column list the amount consumed as a volume, weight, number of pieces, etc. Whenever possible, copy the portion eaten from cans, bottles, and packages.

For the food description, please give as many details as possible and also the brand names of commercial products. Please record the method of preparation, for example: raw, baked, boiled, etc., and whether the item was fresh, canned, or frozen. Please record any additions such as salt, sugar, butter, or gravy. If you eat a casserole, stew or other mixed dishes, we would be very grateful if you could include the recipe on the blank pages at the back and remember to record how much of the whole recipe you actually consumed, for example: one half, one third, etc.

Remember to record all snacks, gum, candy, alcoholic or other beverages, cough drops, vitamin or mineral supplements and the amount that you consumed during this day. An example of the correct method of filling out your Food Record is shown on the following pages. When you are writing your Food Record, imagine that someone wants to duplicate your meals as closely as possible.

continued...

## Appendix 7. (continued)

### 24-HOUR FOOD INTAKE

NAME \_\_\_\_\_

DATE \_\_\_\_\_

TIME OF DAY

AMOUNT

DESCRIPTION

continued...

## Appendix 7. (continued)

## CORRECT METHOD OF COMPLETING A 24-HOUR FOOD RECORD

TIME OF DAY	AMOUNT	DESCRIPTION
7:00 am	2 slices 3 Tbsp 6 oz 1 tsp 2 Tbsp	toasted white bread Kraft <sup>R</sup> strawberry jam perked coffee white sugar homo milk
12:15 pm	1/2 10 oz can  1 slice 3"x3"x1/4" slice 1 Tbsp 3 - 2" diameter	Campbell's <sup>R</sup> chicken noodle soup rye bread baked ham mayonnaise Oreo cookies
6:30 pm	1 3"wide x 1" thick 1 - 8 oz cup 1 - 6" 4 oz	white hamburger bun broiled beef patty frozen peas, boiled banana 2% milk
10:30 pm	6 oz 2 - 3" square 2"x1"x1/2" slice	tea unsalted soda crackers cheddar cheese