UNIVERSITY OF CALGARY

The Determinants of Food Intake in Individuals with Mood Disorders

by

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A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES
IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE
DEGREE OF DOCTOR OF PHILOSOPHY

DEPARTMENT OF COMMUNITY HEALTH SCIENCES, FACULTY OF MEDICINE

CALGARY, ALBERTA

(JANUARY, 2010)

UNIVERSITY OF
CALGARY

ABSTRACT

A study was carried out in 97 community-based adults with verified bipolar or major depressive disorder to evaluate nutrient intakes and their relationship with mental status. The participants were mainly single females who had less than a university degree, had lower incomes, and tended to have excess body weight.

Based on dietary guidelines, their nutrient intakes from food sources showed potential inadequacies for α-linolenic acid, linoleic acid, fibre, protein, most of the B vitamins, vitamin C, magnesium, potassium, iron, phosphorus, and zinc. In comparison to provincial survey data, the prevalence of nutrient inadequacies tended to be higher. When supplement intakes were added to food sources, many participants no longer displayed inadequate levels; some exceeded the Tolerable Upper Intake Levels for folate, niacin, vitamins B_6, C, D, and E, calcium, iron, magnesium, zinc, and manganese. Higher intakes of energy, protein, fibre, thiamin, niacin, vitamin B_6, iron, sodium, magnesium, phosphorus, potassium, and zinc tended to be associated with higher income, having a partner, lower levels of cognitive dietary restraint and disinhibition, and being male and older.

Measures of association between nutrient intakes from food and psychiatric functioning showed many weak significant positive correlations, suggesting better psychological function was associated with higher dietary nutrient intake. When supplement sources were added to food, significant negative correlations were found between mania and zinc as well as depression and iron. Higher levels of psychological functioning were associated with greater intake of selected dietary minerals derived from food plus supplements. There was no demonstrated relationship between mental functioning and micronutrient intakes when linear regression analysis was conducted; significant associations were found only for fat intake and psychological functioning scores. The lack of association in the regression analyses may be attributed to the limited sample-to-variable ratio.

These results suggest that the relative sufficiency and quality of food, individual and contextual factors influencing food selection, and supplemental mineral intakes are considerations for nutritional interventions in mood disorders. Further study on nutrient intakes, supplement use, mental status, as well as social and environmental factors related to eating behaviours in this population is warranted.

ACKNOWLEDGEMENTS

This thesis would not appear in its present form without the kind assistance and support of many individuals and organizations.

First, to the participants who spared the time to write food records and engage in the nutrition and clinical interviews; I am very grateful. Without you, this study would not have been possible.

I would like to extend a very heartfelt thank you to my supervisor – Dr. Bonnie Kaplan. She has been incredibly supportive and patient during my studies. She has also provided extensive advice and knowledge. I am very fortunate to have had her as my mentor for this project.

To the members of my supervisory committee – Drs. Marja Verhoef, Scott B. Patten, and Misha Eliasziw – I am grateful for your support and theoretical insights. I could not have hoped for a better ensemble of academics to help guide me.

I would like to acknowledge the tremendous support and assistance of the Mood Disorders Association of British Columbia (MDABC) as well as the staff of this research project. The MDABC is an organization with a genuine motivation to help those suffering from mental illness. They provided support staff, office space and assistance with recruitment for this research project. Thanks to Ed Rogers, Vicki Rogers, Julia Zohab, Johannes Presley and Rennie Hoffman. Also, gratitude is extended to Registered Dietitian Sue Maxwell who did an excellent job with the nutrition component of the interviews. Thank you also to the clinical interviewers – Erin Putterman, Maggie Hanson, Lindsay Thomas and Janine Moseley.

I am also thankful to those who provided the financial resources for this project and helped coordinate the blood collection component of this study. I would like to acknowledge The Danone Research Institute for their generous support in funding for this investigation. Thank you to Dr. Ronald A. Remick, consultant psychiatrist with the Department of Psychiatry, St. Paul's Hospital in Vancouver who signed the laboratory requisitions. Also, to Wendy Heathcote of BC Biomedical Laboratories Limited for helping to organize the participant bloodwork collection and analysis both with her organization and with the laboratory at British Columbia’s Children’s Hospital.

I am also appreciative of the staff of Health Canada and the BC Ministry of Health, as they provided assistance in the utilization of the BC Nutrition Survey data. I am also grateful to Pardeep Hans for her hard work and dedication with the data entry of the food records.

Finally, I would like to thank my parents. Being a new mom of two children during my PhD brought great joy, but also significant challenges in getting this project completed. My parents were able to help me overcome those challenges by providing countless hours of childcare. My family is so thankful for this support. We love you.

DEDICATION

First, I would like to dedicate this thesis to my children, Serena and Breslin. You are two precious gifts that arrived during my doctoral studies and added a whole new dimension to my life. I love both of you with all of my heart.

I would also like to devote this work to my third child, who at the time of writing is an 8-month-old fetus. Your siblings, dad and mom are excited that you will be arriving soon!

Finally, I want to dedicate this thesis to my wonderful husband Scott. I will forever be indebted for his remarkable support and encouragement. His approach to life is one of unfailing honesty and integrity. Each day I can't help but love and admire him more. He was there for me every step of the way and reminded me to have a sense of humor; "Hey! How's the PhD?"

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LIST OF SYMBOLS, ABBREVIATIONS, NOMENCLATURE

Abbreviation	Definition
ACTH	Adrenocortico-tropic Hormone
ADD	Attention Deficit Disorder
ADHD	Attention Deficit Hyperactive Disorder
AGE	Advanced Glycation End Products
AI	Adequate Intakes
ALA	Alpha-Linoleic Acid
ANOVA	Analysis of Variance
AMDR	Adequate Macronutrient Distribution Range
ATP	Adenosine Triphosphate
BCNS	British Columbia Nutrition Survey
BD	Bipolar Disorder
BDI	Beck Depression Inventory
BEE	Basal Energy Expenditure
BMI	Body Mass Index
BPRS	Brief Psychiatric Rating Scale
CCHS	Canadian Community Health Survey
CH_3	Methyl Group
CI	Confidence Interval
CNF	Canadian Nutrient File
CNS	Central Nervous System
DAST	Drug Abuse Screening Test
df or DF	Degrees of Freedom
DFE	Dietary Folate Equivalents
DHA	Docosahexaenoic Acid
DINs	Drug Identification Numbers
dl	Decilitre
DLC	Digitalis-like Compounds
DNA	Deoxyribonucleic Acid

Abbreviation	**Definition**
DRI	*Dietary Reference Intakes*
DSM	The Diagnostic and Statistical Manual of Mental Disorders
EAR	Estimated Average Requirement
EDTA	Ethylenediaminetetraacetic Acid
EEG	Electroencephalograms
EFA	Essential Fatty Acid
EPA	Eicosapentaenoic Acid
ES	Effect Size
F	F test statistic
FAD	Flavin Adenine Dinucleotide
FFQ	Food Frequency Questionnaire
FSQ	Food Selection Questionnaire
g	Grams
GABA	Gamma-Aminobutyric Acid
GAF	Global Assessment of Functioning
GAS	Global Assessment Scale
Ham-D	Hamilton Depression Scale
HC	High-Calcium Diet
Hcy	Homocysteine
HNE	Hydroxynonenal
HPA	Hypothalamic–Pituitary–Adrenal
ICC	Intraclass Correlation
ICD	International Classification of Diseases
IU	International Units
K^+	Potassium
kcal	Kilocalories
kg	Kilogram
LNAHK	High-Potassium Diet
m	Metre
MAOI	Monoamine Oxidase Inhibitors

Abbreviation	Definition
MDABC	Mood Disorders Association of British Columbia
MDD	Major Depressive Disorder
MDE	Major Depressive Episode
MEq	Milliequivalent
METS	Metabolic Equivalents
Mg	Magnesium
mcg	Micrograms
mg	Milligrams
mmHg	Millimetres Mercury
MTHFR	Methylenetetrahydrofolate Reductase
MW	Molecular Weight
N or n	Number in sample
nmol	Nanomole
NADH	Nicotinamide Adenine Dinucleotide
NE	Niacin Equivalents
NHANES	National Health and Nutrition Examination Survey
NMDA	N-methyl-D-aspartate
NPHS	National Population Health Survey
Na^+	Sodium
ng	Nanograms
ns	Not Significant
OC	Oral Contraceptive
OD	Moderate-Sodium, High-Potassium, High-Calcium Dietary Approaches to Stop Hypertension Diet
OR	Odds Ratio
p	Probability, statistical significance level
PA	Physical Activity Coefficient
PEA	Phenylethylamine
pmol	Picomole
p.o	By Mouth

Abbreviation	Definition
POMS	Profile of Mood States
PUFA	Polyunsaturated Fat
q.i.d.	Four Times A Day
r	Correlation Coefficient
R^2	Coefficient of Determination
RAE	Retinol Activity Equivalent
RBC folate	Red Blood Cell Folate
RCT	Randomized Clinical Trial
RDA	Recommended Dietary Allowance
RE	Retinol Equivalent
RNA	Ribonucleic Acid
RR	Relative Risk
SAD	Seasonal Affective Disorder
SCL-90-R	Symptom Checklist-90-R
SIGH-SAD	Structured Interview Guide for the Hamilton Depression Rating Scale, Seasonal Affective Disorders
SAMe	S-adenosyl-L-methionine
SCID-P	Structured Clinical Interview for DSM-IV Axis I Disorders – Patient Edition
SD	Standard Deviation
Sec	Selenocysteine
SE	Standard Error
SEM	Standard Error of the Mean
SNRI	Serotonin and Norepinephrine Reuptake Inhibitor
SSRI	Selective Serotonin Reuptake Inhibitors
t	T-test statistic
TEE	Total Energy Expenditure
TFEQ	Three-Factor Eating Questionnaire
TYR/LNAA	Tyrosine to Large Neutral Amino Acid Ratio
TRP-LNAA	Tryptophan to Large Neutral Amino Acid Ratio

Abbreviation	Definition
UBC	University of British Columbia
UL	Tolerable Upper Intake Levels
YMRS	Young Mania Rating Scale
yrs	Years
z	Z test statistic
μmol	Micromole
25-OH D	25-hydroxy Vitamin D
χ^2	Chi square test statistic
≤	Less than or equal to
≥	Greater than or equal to
%	Percent

CHAPTER ONE: LITERATURE REVIEW

Mood disorders as defined by the American Psychiatric Association include bipolar disorder and depression[1]. Bipolar disorder, subdivided as bipolar I and II, is characterized by alternating periods of depression and hypomania or mania, whereas depression is characterized by sustained periods of depression without manic episodes. Both Axis I disorders are associated with long-lasting disability and significant mortality through suicide, medical illness, and accidental death. Current treatment guidelines for mood disorders rely on pharmaceuticals; depression is treated primarily with anti-depressant medication, those with bipolar disorder require a mood-stabilizing medication. These medications have only partial benefit and in many cases have adverse effects. This has created an ongoing need for research into other treatments, including better medications and adjunctive therapies such as nutritional interventions.

It has been hypothesized that many abnormalities of brain function associated with mood disorders can be explained as a disturbance in the communication process. These anomalies in brain function may also be partially due to deficiencies in or excesses of nutrients known to alter brain biochemistry. A number of proposed links between diet and mental health have been popularly accepted without benefit of adequate scientific support. However, while the role of diet in preventing and treating physical health disorders is both understood and accepted (e.g., diet's role in the prevention of coronary heart disease), there is generally less awareness and evidence that diet could play an important role in the management of mood disorders. The available research has produced some contradictory findings, but also some recurring trends that support the hypothesis of diet influencing mood. This chapter will examine the research, beginning with some thoughts about the frameworks that explain links between nutrients and mood. This is followed by a review of the literature, including a discussion of the knowledge base on the relationship between nutrition and mood disorders based on the categories of macronutrients, vitamins, minerals, phytochemicals, other nutritional factors, non-nutritive substances, dietary risk factors, and determinants of food intake.

1.1 Explanatory Models

Brain imaging techniques have shown brain structure changes associated with mood disorders in a number of brain areas including the limbic, orbital and prefrontal cortex,

amygdala, ventral striatum, and hippocampus. Increasing evidence suggests that nutrition affects the structure and functioning of the brain because of its high metabolic activity. A review of the research suggests that 9 common inter-related frameworks can explain nutrient effects on mental function. These include evolution of the typical diet, inborn errors of metabolism, prenatal nutrition, deficient methylation process, alteration of gene expression, mitochondrial disease, long latency disease, mechanisms of oxidative stress and cortisol depletion.

The Mental Health Foundation[2] suggests that the increased incidence in mental disorders such as clinical depression over recent years might be linked to the change in our diet over the same time frame, with a shift from a diet based on various whole foods to eating patterns based on more processed foods. Nutrient content of our food supply could also be considered in conjunction with these hypotheses. Data indicates that the mineral and trace elements of fruits and vegetables have been decreasing significantly over the last 50 years[3;4], at least partially as a result of the poor remineralization of the soil. It is possible that some individuals are highly sensitive to these nutritional depletions present in food as their biochemical needs are different. Clearly the incidence of mental health problems is a complex issue most likely to be associated with a range of biological, social, and economic factors and diet might be just one part of this.

Inborn errors of metabolism can have many effects, including influencing enzyme and coenzyme reactions and ultimately brain function. In a review of 50 human diseases attributed to an enzyme having decreased binding affinity for a coenzyme[5], Ames showed that in the majority of cases this type of mutation is correctable by feeding the affected person additional cofactors or coenzymes (i.e., vitamins), thereby raising the coenzyme levels and enhancing enzymatic activity.

Human neurodevelopment is the result of genetic and environmental interactions. Epidemiologic studies that examined the role of prenatal nutrition relative to psychiatric disorders have found that prenatal caloric malnutrition, low birth weight, and prematurity increase the risk for neurodevelopmental disorders, schizophrenia, affective disorders, and schizoid and antisocial personality disorders[6]. Placebo-controlled studies in medicated pregnant women suggest that add-on treatment with omega-3 fatty acids,

particularly eicosapentaenoic acid, may ameliorate symptoms of major depressive disorder[7]. Additional studies are necessary to confirm any benefits for mood disorders.

Methylation reactions (i.e., adding a methyl group (CH_3) to a molecule) represent one interface between nutrients and genetic expression. There are hundreds of methylation reactions in our bodies, including those needed for DNA transcription and neurotransmitter synthesis. There is evidence of deficient methylation processes in relation to mood symptoms, leading researchers to examine compounds called "methyl donors" that transfer CH_3 in the synthesis of important compounds[8]. For instance, the biochemical interrelationship between folate and cobalamin (Vitamin B_{12}) lies in the maintenance of nucleic acid synthesis and methylation reactions, such as the methylation of homocysteine to methionine and the synthesis of S-adenosyl-L-methionine (SAMe)[9].

As this discussion also suggests, nutrients can alter gene expression. Norwegian research[10] has shown increased risk for depression in patients with a particular genotype that is associated with increased homocysteine and decreased folate.

An alternative model for explaining underlying nutrient related mechanisms and mental function involves energy metabolism. For example, it has been proposed that some mood symptoms represent a mitochondrial disease associated with decreased mitochondrial energy metabolism[10-12]. Treatment benefits from micronutrients would be consistent with this framework as they improve ATP activity.

Heaney[13] has proposed that many human chronic diseases (e.g., central nervous system degeneration) are long-latency effects. The fact that many individuals do not experience their first episode of mental illness until after decades of life suggests that long-latency deficiencies may be relevant. Deicken, Pegues, Anzalone, Feiwell, and Soher[14] have provided proton magnetic resonance spectroscopic evidence of progressive changes in the right hippocampus in 15 patients with familial bipolar I disorder. The correlation between years of illness and reduced N-acetyl-aspartate concentrations was quite high, suggesting that the brain is gradually less able to produce this amino acid. However, the direction of causality requires scrutiny as it is plausible that long-term psychological stress alters nutrient absorption or even directly (perhaps via elevated cortisol secretion) influences brain development[8].

In relation to the long-latency theory, research suggests that free radical production (oxidative stress) mechanisms appear to be a common thread among various neurological and emotional disorders such as Alzheimer's Disease, Amyotropic Lateral Sclerosis, anxiety disorders, Attention Deficit Hyperactivity Disorder, Autism, dementia, depression, fibromyalgia, Huntington's disease, multiple sclerosis and schizophrenia. For example, a recent study that examined post-mortem anterior cingulate brain sections in four groups (i.e., bipolar disorder, major depressive disorder, schizophrenia, and non-neurological, nonpsychiatric controls) of 15 subjects each revealed that 4-hydroxynonenal (4-HNE) levels, a major product of lipid peroxidation, were significantly increased in bipolar and schizophrenia subjects even after controlling for pH[15]. Other studies have shown that high antioxidant intake prevents oxidation tissue stress in the brain and that when lipid peroxidation is high, depression is more likely[16]. A recent review suggested that a solid foundation for oxidative stress hypotheses in mood disorders and depression has been provided by biochemical, genetic, pharmacological, preclinical therapeutic studies and one clinical trial[17]. These data not only suggest that oxidative mechanisms may form unifying pathogenic pathways in psychiatric disorders, but also introduce new targets for the development of therapeutic interventions[17].

Cortisol, an important steroid hormone secreted in response to stress, may affect mental health. Cortisol secretion is a marker of hypothalamic-pituitary-adrenal (HPA) axis activity, and it follows a diurnal pattern with peak levels following awakening and declining levels thereafter[18]. Negative mood states, including depression in men[19] and fatigue in women[20], have been associated with higher levels of cortisol in the afternoon/evening period. Conversely, "burnout," as a result of chronic stress, and post-traumatic stress disorder[21] have been associated with lower levels of cortisol in the afternoon/evening period. Psychological factors associated with dietary intake (e.g., cognitive dietary restraint) may alter cortisol secretion[22] and therefore mood stability. Furthermore, some studies have shown that consumption of dietary proteins enriched with tryptophan increases the tryptophan-large neutral amino acid ratio and, in stress-vulnerable subjects, improves coping ability and mood, probably through alterations in brain serotonin[23].

The conceptual frameworks outlined here are compatible views of how brain metabolic pathways may be affected by diet. From here a review of the nutrition-related factors related to mood disorders are considered further.

1.2 Review of the Literature

The vast majority of studies on diet and mood disorders exhibit several flaws in the research methodology. First, many studies relied on advertisements to recruit their subjects, for example, resulting in a self-selection bias. Second, intervention studies used extracted dietary substances rather than actual food, thereby making it difficult to generalize the results to whole foods that contain many interacting substances. Furthermore, participation in trials can artificially inflate the effect of treatment due to care provided, "force" participants into a better daily routine thereby creating positive effects, or change may occur simply due to natural remission of symptoms over time. Third, there is a consistent failure to consider possible intervening variables when interpreting the data. For example, many studies take place, for the sake of convenience and control, in institutional settings. The dependent variables (e.g., behaviour, symptoms) that are allegedly affected by dietary manipulation often include measures of mood state and antisocial tendencies. Both these dependent variables may also be affected by the environmental variables that are inherent in an institutional setting. Therefore, although these studies aid in the understanding of relationships between nutrition and mood, they tend to lack a comprehensive appreciation of the complexities of nutrition and the multi-determination of mood symptoms.

A computer-generated literature review to locate English-language, peer-reviewed studies reporting on nutrition and mood disorders was undertaken in a set of medical-based search engines using keyword searches related to mood disorders and nutrition. Additional relevant literature was found by searching reference lists of retrieved articles. The search focused on studies published between January 1, 1980, and September 1, 2009, as they reflected classifications of mood disorders according to accepted psychiatric diagnostic standards commencing from the publication of the DSM-III and ICD-9. The articles were then divided into subgroups of interest.

The literature search yielded 530 articles, but many of these (n = 266) were discarded because of flaws in their measures or they did not use the defined psychiatric criteria.

Screening of the article reference lists yielded an additional two studies that met the defined criteria. There were an insufficient number of controlled trials to warrant a meta-analysis, and so for each category discussed, studies that linked nutritional variables to mood symptoms were discussed. The studies tended to include anecdotal, case, case-control, or correlational studies. There were few interventional studies.

1.2.1 Energy and Macronutrients

Energy

Energy, measured as calories, is derived from the carbohydrate, protein, fat, and alcohol found in foods and beverages. The human brain is metabolically very active and uses about 20% to 30% of a person's energy intake at rest. Inadequate caloric intakes can lead to changes in mental functioning, as shown in classic studies on the effects of fasting conducted during the 1950s[24]. The body responds to energy deprivation by ceasing or slowing down nonessential functions, altering activity levels, hormonal levels, oxygen and nutrient transport, and many other bodily functions that directly or indirectly affect brain function.

Studies of restricted energy diets and mood have mainly been done in healthy subjects and have shown mixed results. Restricted-energy diets for the treatment of excess weight have been reported to increase fatigue and tension[25], which may be associated with feelings of deprivation and hunger. One study, however, reported a neutral effect on mood of an energy-restricted diet[26]. The ketogenic diet (i.e., a high-fat, low-carbohydrate, low-protein diet), traditionally used in the treatment of drug-resistant epilepsy, has been investigated for its mood stabilizing effects[27;28]. These studies have largely been based on anecdotal reports, but suggest that the ketogenic diet (without resulting in ketosis) deserves further study as a possible treatment for depression. This ketone-producing diet includes manipulation of all three macronutrients; the effects of each are discussed separately in the following.

Carbohydrates

Glucose, a form of sugar that the body creates from the carbohydrates one consumes, is the preferred energy source for erythrocytes and nerve cells, including those of the brain. Carbohydrates significantly affect mood and behaviour for various reasons. Eating

a meal high in carbohydrates triggers the release of the hormone insulin in the body that helps blood glucose enter the cells. In addition, as insulin levels rise, more of the amino acid tryptophan crosses the blood brain barrier that affects levels of neurotransmitters such as serotonin in the brain. Higher serotonin levels in the brain enhance mood. Researchers suggest that those who are depressed and crave carbohydrates may be self-medicating with this dietary substance because of its biochemical effect of raising brain serotonin levels.

Foods that are primary sources of carbohydrates are important to brain function. Carbohydrates include starches, naturally occurring and refined sugars (simple carbohydrates), and dietary fiber. Simple carbohydrates contain either one or two molecules of monosaccharides (i.e., glucose, fructose, galactose), while complex carbohydrates contain hundreds to thousands of monosaccharides. The main food sources where carbohydrates can be found include the fruits and vegetables as well as grains. These foods are also important sources of many B vitamins, antioxidants, and phytochemicals. Carbohydrates are also found in milk and milk alternatives as well as the meat alternatives (e.g., nuts, seeds, legumes). It is the macronutrient that is most widely distributed in our food supply.

Several studies have examined the effects of carbohydrates on mental function. In a 5-year diet intervention study (low-fat, high complex-carbohydrate diet) aimed at reducing plasma cholesterol in 149 men and 156 women, significant improvements in overall emotional state (i.e., depression and aggression) based on the Hopkins Symptom Checklist (SCL-90) were found when compared to those who ate a high-fat diet[29]. Those who consumed a low-fat, high complex-carbohydrate diet at the end of the study showed significantly greater improvements in depression ($p = 0.044$; difference in improvement, 2.9 points) and aggressive hostility ($p = 0.035$; difference in improvement, 3.3 points) compared with those who ate a high-fat diet.

Diets of depressed patients have indicated higher sucrose content compared to normal controls[30], which may be due to impairment of glucose utilization[31]. Studies of mental health outcomes and sugar intake have suggested that background medication use has a confounding effect. For example, patients taking the antipsychotic drug clozapine consumed twice as much sugar as those taking other antipsychotic drugs[32]. A small

intervention study that examined the effects of a 6-week carbohydrate-restricted diet (i.e., 8% carbohydrate and overall caloric level to maintain body mass) on body composition and the relationships with fasting hormone concentrations in 12 healthy, normal-weight men showed that the carbohydrate-restricted diet had no effect on cortisol secretion which can affect mood state[33]; however, these results must be interpreted with caution for those with mood disorders.

Inositol, an isomer of glucose found in food as a fibre component known as phytic acid, is a precursor of intracellular second messengers[34]. Levels of inositol, a key intermediate of noradrenergic, serotonergic, and cholinergic mechanisms, were reduced in the cerebrospinal fluid of individuals with mood disorders[35]. In addition, patients with bipolar disorder tend to have lower uptakes of inositol when compared with controls[36].

Clinical trials have demonstrated benefits for the treatment of depression with inositol[37]. In the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) study, 66 patients with bipolar I or bipolar II disorder in a current major depressive episode that was nonresponsive to a combination of adequate doses of established mood stabilizers plus at least one antidepressant were examined. Patients were randomly assigned to open-label adjunctive treatment with lamotrigine, inositol, or risperidone for up to 16 weeks. The primary outcome measure was the rate of recovery, defined as no more than two symptoms meeting DSM-IV threshold criteria for a mood episode and no significant symptoms present for 8 weeks. No significant between-group differences were seen when any pair of treatments were compared on the primary outcome measure. However, the recovery rate with lamotrigine was 23.8%, whereas the recovery rates with inositol and risperidone were 17.4% and 4.6%, respectively[38]. Investigations examining inositol as an intracellular target in treatments for bipolar disorder are in progress.

An area of particular interest in brain health and carbohydrates is that of the glyconutrients or plant monosaccharides. While there are over 200 carbohydrates or sugars, 8 are thought to be essential to bodily function: xylose, fucose, galactose, glucose, mannose, N-acetylglucosamine, N-acetylgalactosamine, and N-acetylneuraminic acid (a sialic acid). Glyconutrients are involved in cellular communication and function. Synapses are enriched in glycoconjugates and the biosynthesis of a carbohydrate chain is

catalyzed step-by-step by specific glycosyltransferases whose activity and specificity are influenced by several environmental parameters (ions, salts, temperature, concentration and competition of sugar donors and acceptors, etc.)[39]. Glyconutrients are believed to be involved in mental conditions such as ADD, ADHD, depression and schizophrenia, however the research to date is preliminary.

Fats

The lipid concentration of the brain partly reflects the dietary intake of different lipids. The mechanism that brings about these changes presumably involves alterations in the properties of neuronal cell membranes and, therefore, changes in the receptors embedded in the membranes. Different human populations have varying fat intakes, but to what extent any differences in cognition that may occur in populations (e.g., comparing traditional Inuit or Hindu diets to the current North American diet) are caused by different brain lipid content is entirely speculative.

The lipid concentration in the brain/nervous system (50% to 60% lipid) is second only to the lipid concentration in adipose tissue[40]. Furthermore, about 35% of the brain/nervous system tissue comprises polyunsaturated fatty acids (PUFA) that include the essential fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). EPA and DHA form phospholipids in brain cell membranes and have important roles in signal transduction. These two essential fatty acids cannot be sufficiently produced de novo in the body, but they can be synthesized from alpha-linoleic acid (ALA). In the diet, the omega-3 fatty acids can be found in plants and marine organisms.

A substantial number of observational, epidemiological, and clinical studies suggest that the essential fatty acids (EFAs) could prevent and improve psychiatric disorders. Omega-3s regulate corticotrophin factor, increase serotonergic function, increase dendritic arborization, prevent neural apoptosis, improve cerebral blood flow, and regulate gene expression[41]. A lack of omega-3s results in the use of other types of fatty acids, which weakens cellular networks and brain stability[42].

The research literature has suggested that EFAs have successfully reduced the Hamilton Rating Scale of Depression scores in subjects with major depressive disorder[43] and prevented depressive episodes in subjects with bipolar disorder[44]. EFAs have also been used to successfully treat the mania associated with bipolar disorder. In one study[44],

patients with bipolar disorder were given an EPA/DHA combination capsule, or olive oil as a placebo. Patients who received the EFA dose no longer met the criteria for a manic mood episode, and showed both a lack of recurrence and a significant reduction in psychiatric symptoms, based on the Psychiatric Symptom Rating Scale. A meta-analysis of 10 double-blind, randomized controlled trials in patients with mood disorders receiving omega-3 PUFAs with the treatment period lasting 4 weeks or longer demonstrated a statistically significant antidepressant effect (ES = 0.61, p = 0.003). Omega-3 PUFAs significantly improved depression in patients with clearly defined depression (ES = 0.69, p = 0.002) or with bipolar disorder (ES = 0.69, p = 0.0009). The dosage of EPA did not change the antidepressant efficacy significantly. However, heterogeneity and publication bias were limitations of these studies[45]. It is not currently possible to recommend omega-3 PUFA as either mono- or adjunctive-therapy, but the available evidence is strong enough to justify continued study in mood disorders.

Restricted intakes such as low-fat diets might adversely affect psychological well-being. Evidence suggests that serum lipid levels may be correlated with variations in mental state including suicidal tendencies[46;47], depression[48;49], manic episodes[50;51], mixed episodes[52;53], and remitted states[51;52;54]. However, a variety of factors influence serum lipid levels, including genetics, gender, age, body mass index, alcohol use, medications, comorbid physical illness, symptom severity, and the subtype of mood episode.

Most studies among individuals suffering from major depressive disorder have signalled an association between low cholesterol and major depression. Furthermore, some trials showed that recovery may be associated with a significant increase of total cholesterol[55]. However, in a recent study examining serum lipids and symptoms during acute mood episodes of hospitalized patients with bipolar I disorder (68 manic, 8 depressive, and 6 mixed), mean serum levels of cholesterol and triglycerides of participants were comparable to a general sample similar in age[56]. Severe depressive symptoms and comorbid atopic diseases were associated with higher serum cholesterol levels. It is speculated that increased serum cholesterol levels may have greater relevance to immunomodulatory systems and depressive symptoms, in comparison with manic symptoms.

The relationship between plasma cholesterol-reducing interventions to mental status, such as depression, remains a topic of debate. A low-fat diet (i.e., 25% of calories from fat) administered over four weeks increased ratings of anger-hostility based on a profile of mood states ratings in healthy volunteers with normal blood cholesterol levels[57]. Another study over a 12-week period in individuals with mild hyper-cholesterolemia indicated that a low-fat diet (i.e., < 20% of calories from fat with a focus on polyunsaturated fats) improved depression and anxiety[58]. A more recent randomised, controlled study tested the effect of dietary cholesterol-lowering on psychological symptoms. Ten women and 8 men with blood cholesterol levels ranging from 180 to 260 mg/dl were randomly assigned to one of two counter-balanced diet cycles. One was comprised of a low-fat, high-complex carbohydrate phase (< 20% of calories from fat; < 6% from saturated fat; 15% protein; < 100 mg cholesterol/3000 kcal), followed by a washout phase (return to typical eating pattern), followed by a final high-fat phase (37% of calories from fat; 20% as saturated fat; 1000 mg cholesterol/3000 kcal). The other diet cycle was in the reverse order. Each phase was approximately 6 weeks long. To control for dietary intake, the study provided meals to participants six days per week. To ensure compliance, meals were taken home for the 7th day. Participants were seen 6 days a week in the research centre. The caloric intake was adjusted, as needed, to maintain stable body weight. Analyses for repeated measures revealed that the low-fat diet significantly reduced total, LDL and HDL cholesterol, when compared with baseline and the high-fat diet. Ratings of depression, hostility and global severity of psychological symptoms as measured by the SCL-90-R improved significantly on the low-fat, high-complex carbohydrate diet when compared with baseline. Thus, the effects of a low-fat diet on depression appear to be based on underlying blood cholesterol levels; improved effects are seen if pre-existing blood levels of total cholesterol are elevated. High-fat diets appear to have no effect on cortisol secretion which can affect mood[59].

Proteins

The study of dietary proteins and mood disorders largely focuses on the amino acids that facilitate neurotransmission and neuromodulation. The dietary precursors of serotonin and norepinephrine have been the main protein derivatives investigated. Studies of L-tryptophan, a derivative of serotonin, have shown that tryptophan-free diets may

lower mood in those with a history or family history of affective illness[60]. Small-sample, double-blind studies report mixed results for L-tryptophan and L-5-hydroxytryptophan as treatments of mood disorders[61;62] and a systematic review concluded that more studies are needed to evaluate their efficacy and safety before their use is recommended[62].

Two other amino acids discussed in the research include phenylalanine and tyrosine. L-phenylalanine, a derivative of norepinephrine that can be decarboxylated to 2-phenylethylamine (PEA), may affect mood. Urinary phenylacetic acid levels, a measure of brain PEA, are reduced in depression, and levels rise towards normal when anti-depressant therapy is effective[63].

Small-sample double-blind studies done over 20 years ago have suggested that L-tyrosine supplementation may be effective for unipolar depression[64;65]. It has also been suggested that tyrosine to large neutral amino acid (TYR/LNAA) ratios correlate positively with response to the noradrenergic agents maprotiline, nortriptyline and the SSRI citalopram[66]. However, a more recent study[67] of 147 patients with a DSM-III-R diagnosis of major depressive episode not taking any psychotropic drugs (except the occasional benzodiazepine for sleep) for two weeks (or 5 half lives of any drug if this was longer) showed dissimilar findings. Total TRP and LNAAs (i.e., leucine, isoleucine, valine, phenylalanine, tyrosine) were measured by ion exchange and revised spectro-photofluorimetric technique. After completion of the initial assessment, patients were randomly assigned to receive either fluoxetine or nortriptyline, in a non-blind design, for 6 weeks. Patients on fluoxetine were prescribed 20 mg per day for 3 weeks and nortriptyline 25 mg, 50 mg and 75 mg on successive nights, then 75 mg for 1 week. After this the dose of both drugs was adjusted on the basis of clinical need and in the case of nortriptyline blood level (aiming for 200–650 nmol/L). Patients were deemed randomised if they took at least one dose of medication. The Montgomery–Asberg depression rating scale was performed at baseline, 3 weeks and 6 weeks. There was no main effect on 6-week outcome of TRP/LNAA ratio or TYR/LNAA ratio and no interaction between these factors and treatment (fluoxetine vs nortriptyline). However a limitation of this study was that alterations in antidepressant dose were allowed, therefore possibly reducing the effect of TRP or TYR availability on response.

Some studies have examined other amino acids. Some results suggest plasma concentrations of taurine and lysine were increased for patients with major depression[68;69] whereas Tachiki et al.[70] showed decreased levels of taurine in depressed patients. Mathis et al.[71] found increased levels of valine, leucine and isoleucine in depressive patients. Sabelli et al.[72] reported successful therapy with phenylalanine of depressed patients compared to healthy controls. No differences in effects of the anti-depressant imipramine and of DL-phenylalanine were noted based on the Hamilton Depression scale.

Some population-based studies have suggested that low serum albumin may be an independent risk factor for poor cognitive status and depression symptoms. A study comparing individuals who had either bipolar depression, unipolar depression, or schizophrenia, and non-psychiatric surgical controls on hospital admission showed that the psychiatric group had significantly lower serum albumin than non-psychiatric controls[50]. However, the direction of association cannot be substantiated. Serum albumin is conventionally regarded as a biochemical and hematological marker of protein-calorie malnutrition, but it is also involved in chronic inflammation. Albumin is a negative acute-phase protein whose plasma concentration in patients with chronic diseases typically decreases in response to chronic inflammation[73].

Other research appears to suggest that dietary protein modifications such as the lactovegetarian diet in healthy volunteers may help reduce cortisol secretion and improve mood[74]. High-protein, low-carbohydrate diets appear to show a general rise in cortisol[75], however, these studies have been limited to men with type 2 diabetes mellitus.

1.2.2 Micronutrients

The availability and balance of the macronutrients available for brain function are dependent on the action of enzymes and coenzymes. Micronutrients are essential to many coenzyme systems and may be responsible for the full activation of enzymes that synthesize neurotransmitters. For instance, riboflavin, vitamin B_6, and iron are three of the substances needed for the synthesis of biogenic amines.

The neuropsychiatric effects of specific frank deficiencies are well known. Common examples include Wernicke encephalopathy associated with thiamin deficiency, pellagra due to niacin deficiency, psychosis of pernicious anemia from cyanocobalamin

deficiency, and myxedema madness from iodine deficiency[76]. Subclinical deficiencies in individuals with mood disorders may be due to genetic variations in which some are more vulnerable or to historical changes in diet composition. The most widely studied micronutrients in relation to mood disorders are discussed here.

1.2.2.1 Vitamins

Various vitamins are essential to neurotransmission. Those that have been studied in mood disorders include folate, niacin, riboflavin, thiamin, vitamin B_6, vitamin B_{12}, vitamin C, vitamin D, vitamin E, and the vitamin-like compound choline. Table 1.0.1 highlights the reported brain functions of each of these and other nutrients as well as indicates the randomized clinical trials carried out for each. Each of these vitamins is also detailed in the following.

Folate

Folate heightens serotonin function by slowing destruction of tryptophan, acts as an enzyme cofactor that convert tryptophan into serotonin[77] and tyrosine into norepinephine/ noradrenaline, contributes to the formation of compounds involved in brain energy metabolism[78], and is involved in the synthesis of dopamine. Reported prevalence of folate deficiency in mood disorders has ranged between 15% and 38%[79]. A few studies have found a high prevalence of elevated homocysteine secondary to folate deficiency in mood disorders[78;80]. A large population-based study also strongly supports the association between the methylenetetrahydrofolate reductase (MTHFR) 677C>T polymorphism, altered one-carbon metabolism and predisposition to depression[11].

While it has been thought that investigations of folate deficiency in mood disorders are no longer relevant due to folate fortification programs, a review of the folate intake literature suggests it remains a nutrient of concern. The author conducted a computer-generated literature review to extract investigations that reported prevalence estimates or mean levels of folate status (i.e., RBC folate or serum homocysteine) in mood disorders. Of the 21 investigations located and reviewed, there were higher rates of RBC folate deficiency in older adults (prevalence estimates of 12.5 to 32.5%) and inpatients (maximum prevalence estimate of 100%) with mood disorders.

Table 1.0.1: Known brain functions of selected vitamins and clinical trials conducted in mood disorder populations		
Nutrient	**Brain function***	**Clinical trials in mood disorders**
Folate, folic acid (Vitamin B_9)	• Cofactor for enzymes that convert tryptophan into serotonin and tyrosine into norepinephrine / noradrenalin. Heightens serotonin function by slowing destruction of brain tryptophan[77] • Helps form brain energy metabolic compounds[81] and involved in the synthesis of dopamine[82;83]	Meta-analysis of 3 randomized trials (247 participants). Two studies assessed folate as an adjunct (e.g., with fluoxetine and other standard treatments) and found that adding it reduced Ham-D scores on average by 2.65 points [95% CI 0.38-4.93]. Fewer patients treated with folate experienced a reduction in Ham-D score of less than 50% at 10 weeks (RR 0.47, 95% CI 0.24-0.92) The third study found no significant difference when folate alone was compared with trazodone[84]. Folate may supplement other treatments for depression. It is unclear if this is also the case for people with normal folate levels.
Niacin (Vitamin B_3)	• Nicotinamide adenine dinucleotide increases tyrosine hydroxylase activity and dopamine production in pheochromocytoma cells[85]	An open-label study found that nicotinamide adenine dinucleotide (NADH) was superior to placebo and reduced depression symptoms in major depression[86].
Riboflavin	• Involved in vitamin B_6, niacin, folic acid and homocysteine metabolism	No specific clinical trial conducted. Has been investigated in clinical trials in combination with other vitamins.
Thiamine (Vitamin B_1)	• Coenzyme in the synthesis of acetylcholine, γ-aminobutyric acid (GABA), and glutamate[80]. Can mimic action of acetylcholine[87]	No specific clinical trial conducted. Has been investigated in clinical trials in combination with other vitamins.
Pyridoxine (Vitamin B_6)	• Role in the synthesis of dopamine, serotonin, norepinephrine, epinephrine, histamine, GABA[88]. Deficiency tends to reduce production of serotonin and GABA[89]	No specific clinical trial conducted. Has been investigated in clinical trials in combination with other vitamins.

*Brain function section of table adapted from Kaplan BJ, Crawford SG, Field CJ, Simpson JSA (2007). "Vitamins, Minerals and Mood." *Psychological Bulletin*, Volume 133, Number 5, 747-760. RR = relative risk; Ham-D = Hamilton Depression Scale; CI = confidence intervals

Table 1.0.1: Known brain functions of selected vitamins and clinical trials conducted in mood disorder populations/continued

Nutrient	Brain function*	Clinical trials in mood disorders
Cobalamin (Vitamin B_{12})	• Involved in the synthesis of monoamine neurotransmitters[82] • Helps maintain myelin sheaths for nerve conductance[90] • Functions in folate metabolism; deficiency can result in a secondary folate deficiency	No specific clinical trial conducted. Has been investigated in clinical trials in combination with other vitamins.
Panto-thenic acid (Vitamin B_5)	• Changes to coenzyme A that helps convert macro-nutrients into energy • Production of red blood cells, hormones, and nerve regulators[91] • Needed for the uptake of amino acids and acetylcholine, which combine to prevent certain types of depression • Is necessary to make Vitamin D and works closely with biotin and vitamins B_1, B_2, B_3 and B_6	None.
Vitamin C	• Acts as part of the intracellular antioxidant network; a neuroprotective constituent[92] • May also acts as a neuromodulator[93]	Double-blind study of ascorbic acid and ethylene diamine tetra acetic acid (EDTA) in the treatment of manic-depressive psychosis with recognized treatment regimes showed no significant difference between the response of depressed patients to amitriptyline or ascorbic acid and EDTA. Patients responded significantly better to lithium than to ascorbic acid and EDTA[94].
Vitamin A	• Retinoids influence hormone pathways (steroid and thyroid) known to cause mood elevation and depression[95]	None.

*Brain function section of table adapted from Kaplan BJ, Crawford SG, Field CJ, Simpson JSA (2007). "Vitamins, Minerals and Mood." *Psychological Bulletin*, Volume 133, Number 5, 747-760

Table 1.0.1: Known brain functions of selected vitamins and clinical trials conducted in mood disorder populations/continued

Nutrient	Brain function*	Clinical trials in mood disorders
Vitamin D	• 1,25-Dihydroxyvitamin D_3 affects cholinergic activity in several discrete brain regions and may play a role in the neuroendocrine regulation of certain aspects of anterior pituitary function[96]	Randomized controlled trial in 15 subjects with seasonal affective disorder (SAD); 8 received 100,000 IU of vitamin D and 7 received phototherapy. At baseline psychiatric functioning (and after 1 month) and serum levels of 25-hydroxyvitamin D (25-OH D) (and at 1 week after intervention). Subjects receiving vitamin D improved in all outcome measures (i.e., Ham-D, SIGH-SAD, and SAD-8); the phototherapy group showed no significant change. Vitamin D status improved in the vitamin D (74%, $p < 0.005$) and phototherapy group, (36%, $p < 0.01$). Improvement in 25-OH D was significantly associated with improvement in depression scale scores ($r^2 = 0.26$; $p = 0.05$).
Vitamin E	• Alpha-tocopherol protects cells from damage by free radicals[97] • May reduce brain amyloid beta peptide accumulation, relevant in Alzheimer's disease[98]	None.
Vitamin K	• Involved in nervous system development[99]; affects brain calcium regulation through osteocalcin[100]	None.
Choline	• Roles in structural integrity of cell membranes, cell signalling (precursor to acetylcholine); also a major source of methyl groups for methylation reactions[101]	A randomized controlled trial found that lecithin significantly improved symptoms in 5 of 6 patients with mania[102].

*Brain function section of table adapted from Kaplan BJ, Crawford SG, Field CJ, Simpson JSA (2007). "Vitamins, Minerals and Mood." *Psychological Bulletin*, Volume 133, Number 5, 747-760

The review of folate and RBC deficiency also found that there was a high prevalence (9% to 50%) of elevated plasma homocysteine (Hcy). Other studies have suggested that approximately 45-55% of patients with depression develop significantly elevated serum homocysteine[103]. However, these results must be interpreted with caution as many factors contribute to homocysteine levels (e.g., substance use, malabsorption of vitamins, common defects of MTHFR gene, age-related conditions such as common atrophic gastritis, autoimmune diseases such as hypothyroidism, rheumatoid arthritis, systemic lupus erythematosis and diabetes as well as many drug interactions that reduce absorption of cofactors (e.g., anti-ulcer agents and biguanides) or increase the catabolism/ requirements of the vitamins (e.g., antiepileptics and levodopa))[104].

A study of Chinese psychiatric patients showed that although folate deficiency was uncommon, patients with a good response to lithium treatment over one year had higher blood folate levels than those showing an unsatisfactory response[105], suggesting that folate supplementation may be an effective adjunct to this medication. In a larger European study, blood folate, vitamin $B_{12,}$ and Hcy were determined in 3884 elderly subjects. Hyperhomocysteinemia, folate, and vitamin B_{12} deficiency were all significantly related to depressive disorders[106]. Bjelland et al.[11] studied associations between folate, vitamin B_{12}, Hcy and anxiety and depression in 5948 subjects. For the total study group, hyperhomocysteinemia (plasma tHcy level > 15 μmol/L) conferred almost a two-fold increased risk for depression, whereas plasma folate was inversely associated with depression only in the subgroup of middle-aged women. It was unclear if Hcy has a primary role in the expression of mood disorders, or if it has a more causal role as a marker for folate and vitamin B_{12} deficiency.

Associations between depressive disease severity and duration with folate status have also been found. A study by Morris et al.[107] found low folate status in depressed members of a general US population sample aged 15 to 39 years. After adjustment for relevant factors, subjects with a lifetime diagnosis of major depression had folate concentrations in RBC folate that were lower than never-depressed subjects. Subjects with dysthymia alone had lower RBC-levels of folate than never-depressed. Low folate status was found to be most characteristic of recently recovered subjects. In a study of 110 outpatients with major depressive disorder who responded to an 8-week trial of fluoxetine, those with low

folate levels (≤ 2.5 ng/ml) were more likely to experience a later onset of clinical improvement than patients with normal folate levels[108]. Plasma Hcy and vitamin B_{12} level status did not predict the time to clinical improvement.

Niacin, Riboflavin, and Thiamin

Several of the B vitamins have interrelated functions. Thiamin functions as a coenzyme involved in the synthesis of acetycholine, γ-Aminobutyric acid (GABA), and glutamate[80]. Niacin plays a role in cell signalling. Riboflavin is involved in the metabolism of several other vitamins (vitamin B_6, niacin, and folic acid), and therefore severe riboflavin deficiency may affect many enzyme systems.

Poor thiamin status (not necessarily clinical deficiency) may be linked to negative mood states such as introversion, inactivity, fatigue, and decreased self-confidence[109]. However, thiamin deficiency is considered to be rare and it is unlikely to be a determinant of mood. Thiamin supplementation has been shown to significantly inhibit platelet monoamine oxidase activity in general samples[110;111], an action that is the basis of the monoamine oxidase inhibitors.

Depression is a possible manifestation of niacin and riboflavin deficiency[112-114]. The pathway for *de novo* synthesis of the niacin congeners has been shown to be upregulated in postmortem brain tissue in individuals with schizophrenia. In a recent study, high- (HM74A) and low-affinity (HM74) receptors for niacin were investigated in postmortem brains of individuals with schizophrenia and bipolar disorder. HM74 proteins were unchanged and while HM74A was significantly decreased in the schizophrenia group, no differences were found between the bipolar group and controls[115]. Studies undertaken in the 1950s and 1960s indicated conflicting results with supplementation of niacin in depression[114;116]. The efficacy of nicotinamide adenine dinucleotide has been investigated and in an open trial of 200 patients with depression, 93% of the patients improved[86].
In addition to folate, riboflavin (in its coenzymatic form flavin adenine dinucleotide [FAD]) is required as a cofactor for the MTHFR enzyme. The reduced activity of the thermolabile variant of MTHFR has been shown to result from inappropriate loss of its FAD cofactor[117]. In a study that randomized 35 participants with homozygous (TT) genotype and age-matched individuals with heterozygous (CT, n = 26) or wild-type (CC, n = 28) genotypes to receive 1.6 mg/day riboflavin or placebo for a 12-week period,

supplementation increased riboflavin status to the same extent in all genotype groups. However, homocysteine responded only in the TT group, with levels decreasing as much as 22% overall ($p = 0.003$). These findings suggest homocysteine is highly responsive to riboflavin, and may explain why the common polymorphism (*MTHFR 677 TT* genotype) carries an increased risk of certain diseases in Europe (e.g., coronary heart disease) but not in North America, where riboflavin fortification has existed for over 50 years.

Vitamin B_6, B_{12}, Pantothenic Acid and Vitamin C

Three other water-soluble vitamins that have been investigated in mood disorders are vitamins B_6, B_{12}, and C[82;118]. Laboratory evidence suggests that vitamin B_6 deficiency is common in depression[119] and the underlying mechanism may be associated with elevated serotonin[120]. Pyridoxine or vitamin B_6 is involved in the synthesis of many neuro-transmitters (i.e., dopamine, serotonin, norepinephrine, epinephrine, histamine, GABA)[88]. Vitamin B_6 deficiency can selectively reduce brain production of serotonin and GABA[89].

Vitamin B_{12} has been linked with central nervous system processes mainly by its relationship with folate in the synthesis of purines and pyrimidines, as well as nucleic acid and nucleoproteins. Earlier small sample studies had suggested that vitamin B_{12} deficiency is higher in hospitalized mental patients than in the general population[121], and that supplementation appeared to be effective in resolving manic symptoms[122]. However, larger more recent studies have indicated that serum cobalamin levels in newly admitted psychiatric patients did not differ significantly from controls with no mental illness[123].

The role of pantothenic acid in mood disorders has not been specifically investigated. Pantethine, the stable disulfide form of pantetheine, is the major precursor of coenzyme A, which plays a central role in the metabolism of lipids and carbohydrates. Coenzyme A is a cofactor in over 70 enzymatic pathways, including fatty acid oxidation, carbohydrate metabolism, pyruvate degradation, amino acid catabolism, heme synthesis, acetylcholine synthesis, phase II detoxification, and acetylation[124].

In the brain, the nerve endings contain the highest concentrations of vitamin C in the body (after the suprarenal glands)[125]. Increased breakdown of ascorbic acid has been found in manic and depressed individuals. One double-blind study conducted over 25 years ago suggested that vitamin C supplementation may reduce manic symptoms[126].

Vitamins A, D, E, and K

Retinoids, a class of chemical compounds related chemically to vitamin A, have diverse mechanisms of action, including effects on cell division, RNA synthesis, protein synthesis, post-translational glycosylation, prostaglandin biosynthesis, lysosomal membrane stability, and cell membrane structures[95]. They can act via steroid hormone pathways and directly insert themselves into cell membranes; they are therefore probably involved in several molecular pathways. There is also evidence that retinoids influence central nervous system function. For example, isotretinoin, a synthetic oral retinoid, can stunt neuronal development and influence neurotransmitter receptors, including systems involved in psychopathology. A case report of a patient with a history of bipolar disorder indicated an exacerbation of psychosis while taking isotretinoin[127]. The patient demonstrated a return to baseline functioning when she discontinued the isotretinoin. Another patient demonstrated a positive de-challenge and a positive re-challenge (return of psychiatric symptoms upon restarting isotretinoin), but improved with the addition of a selective serotonin reuptake inhibitor[128]. Through heterodimerization, retinoids can influence particular hormone pathways (steroid and thyroid hormones) known to cause mood elevations and depression. Retinoids have been associated with hippocampal suppression, which is particularly interesting as this is a known risk factor for depression.

Because seasonal affective disorder (SAD) has been associated with sunlight deprivation[129], vitamin D deficiency may be a possible contributor to SAD. Early studies found no association with depression and 1,25-dihydroxyvitamin D[129], but subsequent studies found correlations between 25-hydroxyvitamin D and SAD and depression[130]. Additionally, in a randomized controlled trial, vitamin D supplementation of 100,000 IU in 15 patients with SAD was superior to phototherapy based on measures from three depression scales[131]. Expression of CP27B (vitamin D-activating enzyme) and vitamin D receptor ($1,25(OH)_2D$) has been reported in the human brain[132] and low maternal vitamin D status has been shown to affect neurogenesis in animals[133]. Conclusions from these observations are that vitamin D exerts autocrine and paracrine effects on the brain that might possibly underpin the link between 25-OH D status and psychological disorders such as depression[131]. A recent study conducted in the United Kingdom examined assays of the serum 25-hydroxyvitamin D levels of all patients in an old-age psychiatry

rehabilitation unit[134]. Results indicated that 10 of 12 (83%) of patients had vitamin D deficiency, and 92% had suboptimal vitamin D levels. Vitamin D status was strongly predicted by dietary supplementation. Of those not taking vitamin D supplements, 100% had vitamin D deficiency. This study suggested that all psychogeriatric patients be screened for vitamin D levels. Later in this chapter, calcium will be discussed. Because calcium status is dependent on the availability of vitamin D, the role of this vitamin in mood disorders may be attributed to its functions related to this mineral.

Among the various vitamin E components (tocopherols and tocotrienols), only alpha-tocopherol is actively utilized by the brain and is directly involved in nerve membrane protection[125]. Serum vitamin E concentrations were measured in healthy volunteers and depressed patients, with the latter showing significantly lower serum levels, suggesting lower antioxidant defences in those with major depression[135]. In a more recent Australian study plasma vitamin E (α-tocopherol) was measured in 49 adults (average age 47 ± 12 years) with major depression. In a subset (n = 19), usual dietary intake of vitamin E was determined by diet history. Subjects had significantly lower plasma α-tocopherol than had been previously reported for healthy Australians, and plasma α-tocopherol was inversely related to depression core (Beck Depression Inventory). Diet analysis indicated that 89% of subjects met or exceeded the recommended intake for vitamin E, and dietary intake was not related to plasma α-tocopherol in this subset. These findings suggest that plasma levels of α-tocopherol are lower in depression, and this is likely not to be due to inadequate intake of vitamin E from food sources.

Vitamin K is involved in the development of the nervous system[99] and regulates calcium through osteocalcin and the matrix G1A protein. Osteocalcin is found in the brain; in its absence, it appears that brain cells become more vulnerable to the effects of calcium[100]. It is speculated that vitamin K causes unregulated calcium movement and deposition in the body, which may be a cause of psychiatric symptoms. There has been limited research examining possible associations between vitamin K and mood disorders.

Choline

Although choline is not by strict definition a vitamin, it is an essential nutrient. The majority of the body's choline is found in phospholipids, the most common of which is phosphatidylcholine or lecithin[101]. When choline bitartrate (providing two–8 g of free

choline per day) was administered to 6 patients with rapid-cycling bipolar disorder who were being treated concurrently with lithium, 5 had significant reductions in manic symptoms, along with a substantial rise in the concentration of choline-containing compounds in the basal ganglia as measured by proton magnetic resonance spectroscopy[136]. In a study of hospitalized patients with bipolar disorder[137], patients with mania had significantly higher mean levels of erythrocyte choline, although this result was due primarily to a subgroup with particularly high levels. A randomized controlled trial [102] found that lecithin significantly improved symptoms in 5 of 6 patients with mania.

Alterations in the metabolism of choline-containing compounds, particularly phosphtidylcholine, in the anterior cingulate cortex of patients with bipolar disorder (n = 9) compared with controls (n = 14) have been found in proton magnetic resonance spectroscopic imaging studies[138] and were consistent with impairments in intraneuronal signalling mechanisms in patients with bipolar disorder. Other neuro-imaging studies have documented abnormalities in choline metabolism and abnormalities in frontal lobe phospholipid metabolism in individuals with bipolar disorder[139;140].

1.2.2.2 Minerals

Table 1.0.2 summarizes key minerals essential to neurotransmitter systems and each of these are discussed as follows.

Calcium

Calcium mediates vasoconstriction, vasodilation, and nerve impulse transmission. Calcium activity has been shown to be abnormal in mood disorders and mood-stabilizing treatments may regulate calcium ion hyperactivity[141]. In a review of 18 studies on mood disorders, the majority showed abnormal intracellular calcium ion homeostasis, with evidence that depressed patients differed from those with bipolar disorder[142]. Depression in particular was associated with elevated platelet serotonin-stimulated intracellular calcium mobilization[142]. Kamei et al.[143] reported that 31 patients with major depression had significantly lower erythrocyte calcium concentrations compared with controls, regardless of whether they were in an active (n = 12) or remission (n = 19) stage. A predictor of good prophylactic response to lithium includes higher platelet serotonin-induced calcium mobilization[144]. However, the role that dietary calcium may have in these mechanisms is unclear.

Table 1.0.2: Known brain functions of selected minerals and clinical trials conducted in mood disorder populations		
Mineral	**Brain function***	**Clinical trials in mood disorders**
Calcium	• Important intracellular messenger, cofactor for enzymes[145] and release of neurotransmitters	None.
Iron	• Cofactor for ATP production[90]; produces energy in the cerebral parenchyma (by cytochrome oxidase)[125] • Oxidative metabolism[90] • Helps produce serotonin, norepinephrine, epinephrine, and dopamine[77] and myelin[125]	None.
Lithium	• Role in the brain arachidonic acid cascade[146]	None using non-pharmacological doses.
Bromine	• May aid the therapeutic effect of lithium[147]	None.
Rubidium	• May increase dopamine at the receptor sites[148]	None.
Magnesium	• Coenzyme; role in the metabolism of carbohydrates and fats and in the synthesis of nucleic acids and proteins[90] • Important for the active transport of ions across cell membranes and for cell signaling[90]	Study of 20 white male volunteers with bipolar disorder, 22 to 30 years, with prior manic episodes and receiving verapamil for at least 6 months. Ten patients were each randomly assigned to receive verapamil 80 mg q.i.d. and magnesium oxide 375 mg or verapamil 80 mg q.i.d. and placebo. Manic symptoms decreased significantly while serum magnesium rose significantly in the experimental group[149].
Potassium	• Regulate neuronal signalling; may regulate cell volume and protect neurons under metabolic stress	None.
Sodium	• Voltage-gated channels allow sodium ions to enter brain cells[150]	None.

*Brain function section of table adapted from Kaplan BJ, Crawford SG, Field CJ, Simpson JSA (2007). "Vitamins, Minerals and Mood." *Psychological Bulletin*, Volume 133, Number 5, 747-760

Table 1.0.2: Known brain functions of selected minerals and clinical trials conducted in mood disorder populations/continued		
Mineral	**Brain function***	**Clinical trials in mood disorders**
Copper	• Modulator of NMDA-receptor activity • Oxygen metabolism; facilitates electron transfer and regulation • Metabolism of norepinephrine and may affect catabolism and flux at synapses[151]	None.
Zinc	• Protein synthesis and structure and regulation of gene expression[145] • Serves in neurons and glial cells. Certain zinc-enriched regions (e.g., hippo-campus) are responsive to dietary zinc deprivation, which can cause learning impairment and olfactory dysfunction[152]	A double blind pilot study of zinc supplementation (6 received 25 mg of Zn^{2+} once daily and 8 received placebo) was conducted in patients with major depression who were treated with tricyclic antidepressants and/or selective serotonin reuptake inhibitors. Zinc supplementation significantly reduced Ham-D and BDI scores after 6- and 12-week supplementation when compared with placebo treatment[153].
Selenium	• Glutathione peroxidase (selenium dependent) maintains cell membranes and antioxidative protective system[90]	None.
Vanadium	• Inhibits Na+-K+-ATPase pump activity	None.
Chromium	• Involved in glucose and lipid homeostasis[154]	Fifteen medication-free patients with atypical depression received either 600 mcg of chromium picolinate or a placebo over an 8-week RCT. Seventy percent who received chromium were responders (i.e., at least a 66% drop in Ham-D score plus improvement on the Clinical Global Impressions of Improvement Scale), compared with 0% in the placebo group[155].

*Brain function section of table adapted from Kaplan BJ, Crawford SG, Field CJ, Simpson JSA (2007). "Vitamins, Minerals and Mood." *Psychological Bulletin*, Volume 133, Number 5, 747-760. Ham-D = Hamilton Depression Scale; BDI = Beck Depression Inventory

Table 1.0.2: Known brain functions of selected minerals and clinical trials conducted in mood disorder populations/continued		
Mineral	**Brain function***	**Clinical trials in mood disorders**
Manganese	• Manganese deficiency lowers the catecholaminergic content of the brain[156]	None.
Molybdenum	• Role in the breakdown of nucleotides (precursors to DNA and RNA) to form uric acid, which contributes to the plasma antioxidant capacity of the blood[157]	None.
Boron	• May maintain membrane integrity and signal transduction[158]	None.

*Brain function section of table adapted from Kaplan BJ, Crawford SG, Field CJ, Simpson JSA (2007). "Vitamins, Minerals and Mood." *Psychological Bulletin*, Volume 133, Number 5, 747-760.
Ham-D = Hamilton Depression Scale

Evidence of abnormal metabolism of calcium may also account for associations found between major depression and osteoporosis. A study by Michelson et al.[159] reported significantly decreased bone mineral density (at hip, spine, and radius) in 24 women with major depressive disorder or with a past history of depression, compared with 24 healthy women matched for age, body mass index, menopausal status, and race. The lower bone mineral density may reflect long-latency effects of impaired calcium absorption in people with mood disorders[8]; however, others have speculated that high levels of cortisone may be the underlying risk factor[160]. In a group of patients with chronic depression, mean diurnal serum calcium levels were significantly higher than those of depressed patients in remission and normal controls, and depressive symptoms were positively correlated with serum calcium level[161]. Hypocalcemia in depression has also been linked to magnesium deficiency and the differences between serum and plasma calcium levels have been attributed to a change in a calcium-binding factor[36].

Iron

Iron exists in the brain bound to heme- and nonheme-containing proteins. A number of enzyme functions exist in energy metabolism and neurotransmitter homeostasis. Iron provides electron carriers during the synthesis of ATP and is an important part of hemoglobin for providing sufficient oxygen in the brain for oxidative metabolism. This mineral is also involved in the production of serotonin, norepinephrine, epinephrine, and dopamine. For example, iron is a cofactor in the metabolism of tyrosine to dopamine[77]. It is also believed to increase the binding of dopamine and serotonin to serotonin binding proteins in the frontal cortex[162].

Mood disorders have been associated with both chronic iron deficiency[163], and iron overload[163]. Maes et al.[164] found significantly lower serum iron transferrin levels, lower red blood cell counts, lower hematocrit, and lower hemoglobin in 53 subjects with major depression compared with 15 normal controls. The role of excess iron in mood disorders may be due to the role of excess free iron levels in lipid peroxidation[151].

Lithium, Bromine and Rubidium

Low nutritional levels of lithium intake may be inversely related to the risk of serious mental illness[165]. Supplementation with lithium-rich and lithium-free brewer's yeast in two randomly selected groups of different drug users (mostly heroin and crystal

methamphetamine) showed significant differences in total mood test scores[166]. Supplementation with nutritional lithium in mood disorders has not been investigated, although lithium in pharmacological doses is standard treatment for bipolar disorder.

Whether bromine has a known essential role in human health is unknown; weak historical evidence suggests it is an essential ultra trace element[167]. Raised bromine levels have been found in patients during lithium treatment, and it is suggested that bromine may aid the therapeutic effect of lithium[147]. Dietary sources of bromine include fumigated grains and its products. However, animal studies appear to suggest that brain levels of bromine may be affected by iodine levels[168].

Rudidium is an alkali metal that is widespread in nature, found in plant, animal and human tissues. Soybeans are a particularly rich dietary source of this mineral. It has been suggested that rubidium may affect the response of bipolar patients to lithium as it may increase the availability of dopamine at receptor sites[148]. Several studies of rubidium were conducted from the 1970's to 1980's. Many of the ones concerning blood levels have numerous methodological problems. Two double-blind, placebo-controlled studies[169;170] investigated the effects of rubidium chloride in hospitalized chronic schizophrenic patients (18 and 24 subjects). Both showed improvement in negative symptoms; the first showed no effect on positive symptoms while the latter showed negative effect on positive symptoms.

Magnesium

Magnesium has a structural role in cell membranes, and is required for the active transport of ions like potassium and calcium, thereby affecting the conduction of nerve impulses. Magnesium deficiency is well known to produce neuropathologies. Only a small amount of the magnesium found in whole-wheat remains in refined flour, setting a stage for possible deficiency. Magnesium levels vary widely in psychiatric patients; however, those with levels either below or above normal ranges tend to have more disturbed behaviour[171]. Magnesium (as well as zinc and copper) plays an important role in controlling NMDA (N-methyl-D-aspartate) receptors and decreased levels of these minerals may cause abnormal behaviour[156;172;173].

Several systems in the pathophysiology of depression involve magnesium. Magnesium reduces the release of adrenocortico-trophic hormone (ACTH) and also

affects adrenocortical sensitivity to ACTH. The role of magnesium in the central nervous system could be mediated via the N-methyl-d-aspartate-antagonistic, γ-aminobutyric acid -agonistic or an angiotensin II-antagonistic property of this ion. A direct impact of magnesium on the function of the transport protein p-glycoprotein at the level of the blood-brain barrier has also been demonstrated, possibly influencing the access of corticosteroids to the brain. Furthermore, magnesium dampens the calcium ion-proteinkinase C–related neurotransmission and stimulates Na-K-ATPase.

Low magnesium levels were reported in 10 adult inpatients with depression in comparison with healthy controls but not in 6 additional patients who were manic[174]. Imada et al.[175] measured serum magnesium levels in 71 patients with mood disorders and in 30 healthy controls and investigated the relationships between those levels and clinical background factors. Serum levels were significantly higher in patients with mood disorders than in controls and showed no correlation with sex, age, disorder subtype, or disease phase in the mood disorder group. Serum levels did not differ between medicated and unmedicated patients with major depressive disorder. These results suggest that the high serum magnesium levels in patients with mood disorders are related to the underlying disorder itself and are not influenced by medication or clinical background factors. In a cross-sectional study of 66 hospitalized patients with major depression and 58 healthy controls, it was found that blood magnesium, rather than sodium, potassium, and calcium, was related to the intensity of clinical symptoms[176].

The research regarding magnesium and mood disorders has also included three small studies using different forms of magnesium supplementation. Heiden et al.[177] administered intravenous magnesium sulphate to 10 patients with severe treatment-resistant mania. Subjects received a continuous magnesium (Mg) flow of approximately 200 mg/h (4353+/-836 mg/day; daily monitored Mg plasma level: 2.44+/-0.34 mmol/l) for periods ranging from 7 to 23 days. Even though the patients were still being treated with lithium (n = 10), haloperidol (n = 5), and/or clonazepam (n = 10) for the duration of the study, it was possible to significantly reduce their medication dosages with the addition of magnesium sulphate to the treatment regimens. Of the 10 patients, 7 showed “marked improvement” in the Clinical Global Impression Scale[178].

In an earlier study, 9 female patients with severe rapid-cycling bipolar disorder were treated in an open trial with either a magnesium compound (mono-magnesium L-aspartate hydrochloride trihydrate, $C_4H_6ClNO_4Mg.3H_2O$), MW:245.9) four times daily in tablets containing 5 mEq of magnesium or lithium for up to 32 weeks[179]. The magnesium had clinical effects equivalent to those of lithium in more than half the patients; 7 of the 9 (77.8%) patients showed a significant positive response based on Hamilton Depression Scale, Global Impression of severity of illness (mania and depression), the Brief Psychiatric Rating Scale and the Brief Manic Scale.

In treating patients with mania, Giannini et al.[149] compared the effects of verapamil in combination with magnesium oxide to a verapamil-placebo combination in a randomized double-blind study. Subjects included 20 white male volunteers between the ages of 22 to 30 years seen in a private clinic and with DSM-IV prior manic episodes. Participants had been receiving verapamil maintenance therapy for at least 6 months. Based on measures of the Brief Psychiatric Rating Scale (BPRS) all subjects were considered by a psychologist to be either hypomanic or euthymic. Ten patients were each randomly assigned to one of two groups. The first group received verapamil 80 mg q.i.d. p.o. and magnesium oxide 375 mg (the V–M group). The second (V–P group) received verapamil 80 mg q.i.d. and glucose placebo. Manic symptoms decreased significantly in the experimental group while their serum magnesium levels rose significantly. Based on these results, a verapamil–magnesium combination may be a useful therapeutic modality in maintenance therapy of mania.

Molybdenum

In humans, molybdenum is known to function as a cofactor for three enzymes; only sulfite oxidase is know to be crucial for human health[151]. This enzyme catalyzes the transformation of sulfite to sulfate, a reaction that is necessary for the metabolism of sulfur-containing amino acids (methionine and cysteine). Molybdenum also has a role in the breakdown of nucleotides (precursors to DNA and RNA) to form uric acid, which contributes to the plasma antioxidant capacity of the blood. Molybdenum exists in both the cerebrum and cerebellum; however, it is not known whether all brain molybdenum is associated with know molybdoenzymes.

Potassium and Sodium

Sodium- and potassium-activated adenosine triphosphatase (Na+, K+-ATPase) and endogenous digitalis-like compounds (DLC) in the brain have been implicated in the pathogenesis of mood disorders. Na+, K+-ATPase activity in all cells including neurons and glia is a fundamental process that affects cell volume, electrical membrane potential, and various transport systems[180;181]. Mood disorders have traditionally been conceptualized as neurochemical disorders of certain neurotransmitter systems (such as serotonergic, adrenergic, and more recently, glutamatergic and GABAergic systems). However, recent brain imaging and postmortem studies have revealed regional changes in the central nervous system volume, as well as changes in the number and/or size of glia and neurons in discrete brain areas of patients with mood disorders. These recent data suggest neurotrophic function, cellular growth, death, and resilience as possible factors that contribute to these disorders[182]. Changes in the Na+, K+-ATPase/DLC system may contribute to such mechanisms and may be involved in the etiology of the disease. Bipolar disorder has consistently been associated with abnormalities in the Na+, K+-ATPase activity in erythrocytes[183;184]. Furthermore, an allelic association between bipolar disorder and a specific polymorphism within the gene encoding the α-3 isoform of the α subunit of Na+, K+-ATPase has been discovered[185]. Reduced Na+, K+-ATPase activity may result from a lower level of α2 isoform of Na+, K+-ATPase in the brain[186]. Furthermore, levels of endogenous digoxin-like immuno-reactive compound were found to be lower in manic patients compared to normal controls[180].

A study by Goldstein et al.[150] examined the determination of Na+, K+-ATPase / DLC systems in the parietal cortex of patients with mood disorders and two animal models of depression. Na(+), K(+)-ATPase concentrations in human brain synaptosomal fractions, from patients with mood disorders, schizophrenia, and normal individuals, were determined by (3)H-ouabain binding assay. Results indicated that (3)H-ouabain binding in bipolar patients was significantly lower than in patients with major depression or schizophrenia. Na(+), K(+)-ATPase alpha isoforms in synaptosomal fractions were not different among the groups. DLC levels in the parietal cortex of bipolar patients were significantly higher than in normal individuals and depressed patients. These results

support the possibility that Na(+), K(+)-ATPase and endogenous DLC participate in the pathogenesis of depressive disorders.

Among depressed patients, intracellular potassium levels may be low, while serum and plasma potassium levels may be normal[180;187;188]. It is possible that dietary electrolytes may alter cortisol secretion[189;190] which in turn can affect mood state. A recent study compared a low-sodium, high-potassium diet (LNAHK), high-calcium diet (HC) with the moderate-sodium, high-potassium, high-calcium Dietary Approaches to Stop Hypertension diet (OD) in 97 subjects greater than 25 years old who had a blood pressure (BP) measured in the high/normal range (≥ 120 mmHg systolic BP or ≥ 80 mmHg diastolic BP). In a crossover design, subjects were randomized to the two diets for four weeks, each preceded by a two-week control diet (CD). Dietary compliance was assessed by 24 hour urine collections. Mood was measured weekly by the Profile of Mood States (POMS). Saliva samples were collected to measure cortisol. The change in mood between the preceding CD and the test diet (LNAHK or HC) was compared with the change between the CD and OD. Of the 38 women and 56 men (mean age 56.3 (sem 9.8) years) that completed the OD, 43 completed the LNAHK and 48 the HC. There was a greater improvement in depression, tension, vigour and the POMS global score for the LNAHK diet compared to OD ($P < 0.05$). Higher cortisol levels were weakly associated with greater vigour, lower fatigue, and higher levels of urinary potassium and magnesium ($r = 0.1\text{-}0.2$, $p < 0.05$ for all). In conclusion, a LNAHK diet appeared to have a positive effect on overall mood[191].

Copper

Copper is an important modulator of NMDA-receptor activity and is part of the superoxide dismutase enzyme system that fights the damage caused by free radicals. As previously outlined, disturbances of glutaminergic transmission (especially via NMDA-receptors) are involved in pathogenesis of mood disorders. For example, there are some reports regarding blood levels of copper during a depressive episode. For instance, a study that estimated plasma copper and zinc in 35 depressed patients both before treatment and after recovery compared with 35 normal healthy individuals showed mean plasma copper levels were significantly higher compared to controls and after recovery from depression[192]. In another study[193], alterations in serum copper concentrations in

depressive patients before, during, and after antidepressant treatment were investigated and compared to concentrations in healthy volunteers. The serum copper concentration in depression was significantly higher (by 21%) than in the controls. However, effective antidepressant treatments (which reduced symptoms by about 50% measured by the Hamilton Depression Rating Scale) did not normalize this increased copper concentration. These latter findings suggest that serum copper remains stable regardless of treatment and may be considered a "trait marker" of depression. However, these results may be limited by the fact that zinc was not measured; copper/zinc ratios are considered prognostic indicators for monitoring disease activity.

Zinc

Zinc is found in high concentrations in the brain hippocampus and is the second most abundant transition metal in the brain next to iron[34]. It is a cofactor of many proteins distributed among all enzyme classes and thus has diverse functions in brain homeostasis. Its roles include synaptic transmission, DNA synthesis, and the function of many enzymes. Henkin et al.[194] discovered that severe zinc deficiency impaired neuromotor and cognitive performance of adults. They induced zinc deficiency by administration of large doses of histidine, which caused high urinary excretion of zinc. All subjects developed abnormal taste and smell acuity. Some were ataxic or depressed, or developed hallucinations and/or paranoia. Maes et al.[164] found significantly lower serum zinc levels in 31 patients with major depression in comparison to 15 healthy volunteers. Serum zinc levels were even lower for the 23 treatment-resistant patients; however, antidepressant therapy did not affect serum zinc levels. Analyses showed lower serum zinc levels to be both sensitive (79%) and specific (93%) as a marker for treatment-resistant depression.

Clinical studies have demonstrated that the efficacy of standard pharmacotherapy is enhanced by supplementation of zinc and magnesium. The hypothesized antidepressant mechanisms of these minerals include effects on glutamate, brain-derived neurotrophic factor, and glycogen synthase kinase-3[195]. Zinc may also be relevant in mechanisms of depression as copper and zinc levels are regulated by metallothionein, a family of cysteine-rich, low-molecular-weight proteins that have the capacity to bind physiological metals (e.g., zinc, copper, selenium) through the thiol group of its cysteine residues.

One double blind pilot study of zinc supplementation in antidepressant therapy was conducted in patients who fulfilled DSM IV criteria for major (unipolar) depression[153]. Patients received zinc supplementation (6 patients; 25 mg of Zn^{2+} once daily) or placebo (8 patients) and were treated with standard antidepressant therapy (tricyclic anti-depressants, selective serotonin reuptake inhibitors). Patients' status was evaluated before the treatment and 2, 6 and 12 weeks after its commencement. Antidepressant treatment significantly reduced Hamilton Depression Rating Scale scores by the second week of treatment in both groups, and lowered Beck Depression Inventory scores at the 6th week in the zinc-treated group. Zinc supplementation significantly reduced scores in both measures after 6- and 12-week supplementation when compared with placebo treatment.

Selenium

Selenium is found in over 30 selenoproteins and, in the form of selenocysteine, is the only trace element to be incorporated into the genetic code. The selenium-containing amino acid, selenocysteine (Sec), is coded by UGA, which functions as both a signal for termination and a codon for Sec. Selenium plays an important role in the antioxidative protection of membranes, lipoproteins, and nucleic acids and is a component of glutathione peroxidase[196;197]. In a double-blind crossover RCT[196], 50 healthy adults were given either 100 mcg of selenium or a placebo daily for 5 weeks. After a 6-month wash-out period, subjects crossed over to the other treatment for the same length of time. The level of selenium in each subject's diet at baseline was estimated from food frequency questionnaires. Selenium supplementation was associated with a significant improvement in self-reported mood on the Profile of Mood States questionnaire, most notably in those with selenium-deficient diets at baseline. The results suggest that low selenium intake may affect mood; the effects can be reversed with increased selenium intake.

Vanadium

As previously cited, Na+-K+-ATPase activity may be associated with both the manic and depressive phases of mood disorders and may explain the changes in sodium transport that occur in depressive psychosis[184;196;198;199]. Vanadium is a powerful inhibitor of this activity. Therapies based on decreasing vanadate levels in the body (e.g., psychiatric medications, ascorbic acid, EDTA) have been reported to be effective in both

depression and mania[94;200]. Studies of vanadium have also shown lower levels of cobalt and higher levels of aluminum in the blood of patients on lithium[184].

Chromium

Chromium is required for normal glucose and lipid homeostasis[154]. McLeod, Gaynes, and Golden[201;202] conducted a series of single-blind and open-label trials of chromium in treating antidepressant–refractory dysthymic disorder in 5 patients. The addition of chromium supplements led to remission of the symptoms in all 5 patients, and single-blind substitution of other supplements showed that the symptom remission was specific to the chromium supplements. In another study[155], 15 medication-free patients with atypical depression received either 600 mcg of chromium picolinate or a placebo over a 8-week RCT. Seventy percent of the patients who received chromium were responders (i.e., at least a 66% drop in Hamilton Depression Scale score plus improvement on the Clinical Global Impressions of Improvement Scale), versus 0% in the placebo group.

Manganese

Information about the neurochemical functions of manganese is limited. Deficiency of manganese might lead to alterations in neurotransmitter homeostasis via its influence on GABA and glutamate[151]. Low manganese containing enzymes in mitochondria could also lead to lipid peroxidation which might influence calcium flux and neurotransmitter release. There is some evidence that manganese deficiency can lower the catecholaminergic content of the brain[156]. While biological evidence suggests that depression is due to a disruption to neurotransmission of monoamine systems (e.g., norepinephrine) and manganese may have a role in these mechanisms, specific investigations are lacking. Excess manganese may influence monoamine homeostasis[151].

Boron

There is limited evidence to suggest that nutritional intakes of boron, an ultra trace element found in fruits, vegetables, nuts, and pulses has beneficial effects on central nervous function, possibly maintaining membrane integrity and signal transduction. Boron's actions appear to be influenced by magnesium and fatty acid levels[158]. For example, boron deprivation in older men and women altered electroencephalograms (EEG), prompting a shift towards more activity in the low frequencies and less activity in the high, dominant frequencies of the EEG spectrum[203]. While behavioural effects of low

boron (i.e., reduced behavioural activation and mental alertness, reduced psychomotor tasks, decreased attention and memory) have been investigated[158], no specific studies in mood disorders have been conducted to date.

1.3 Multi-Ingredient Formulas

As the literature review suggests, neurological function relies on many nutrients, which further suggests that ingesting multiple nutrients simultaneously as in food sources would optimize brain health. There has therefore been increased interest in using combined nutrient supplements in the treatment of mood disorders. The rationale underlying the use of multi-nutrient formulas is that if one nutrient is deficient in the body, a whole grouping of nutrients will be deficient as the level of one nutrient can affect the adequacy of others[8;204]. Furthermore, interventions of single ingredients may actually alter nutritional balances, creating deficiencies in other nutrients[205].

Several studies of multi-ingredient formulas (e.g., vitamins, minerals, fatty acids, amino acids, and botanicals) suggest they may be significantly more beneficial than single-ingredient interventions[206-209]. In each of these case studies, case series, and clinical trials, the intervention has consisted of three or more micronutrients. In two case studies, on-off control of mood and temper problems were demonstrated using ABAB reversal designs, with benefits sustained for four years. In an analysis of self-report data from 682 adults with bipolar disorder consuming a micronutrient formula (81% taking psychiatric medications), decreased symptom severity over 6 months was associated with increasing micronutrient dosage and with reducing medication[76;210]. The formula in the study consisted of 16 minerals, 14 vitamins, three amino acids, and three antioxidants (a full list of ingredients can be found on the manufacturer's website at www.truehope.com). A more recent 8-week open label trial with natural extension that examined the effects of the same supplement on behaviour and mood in medication-free adults with Attention-deficit/hyperactivity Disorder showed significant improvements on measures of inattention and hyperactivity/impulsivity, mood, quality of life, anxiety, and stress[211].

Studies of other multi-nutrient formulas have also shown positive results. Bell et al.[80] carried out a four-week RCT in 16 elderly inpatients with depression to evaluate the augmentation of tricyclic antidepressant medication with modest supplementation (10 mg

each) of three B vitamins: thiamin, riboflavin, and B_6. Nutrient-induced medication augmentation was observed. Patients taking nortriptyline who received the supplementation experienced higher serum nortriptyline levels, though they were only marginally better in terms of mood symptoms. In a small double-blind randomized controlled study of nursing home residents given selenium, vitamin C, and folate supplements for 8 weeks, the baseline measures showed that 67% of the patients had low serum concentrations of vitamin C, and no one was below the reference range for selenium. Depression was significantly associated with selenium levels, but not with folate or vitamin C levels. Following 8 weeks of micronutrient supplementation, there was a significant increase in selenium levels and improved symptoms of depression occurred in a subgroup[212]. These results suggest that the study of nutrition in mood disorders requires integrated approaches where several interacting substances are considered simultaneously.

A more recent study of multi-ingredient formulas utilized an individualized approach for patients with major depression[213]. Forty in-patients with DSM IV depression were investigated by a randomized double-blind placebo-controlled study. Plasma levels of 20 amino acids and measures of depression, suicidal behaviour and aggression were surveyed on admission and after 4 weeks therapy with Remeron® plus an individualized amino acid mixture or placebo. The preparation of the amino acid mixture was based on an aminogram and consisted of essential amino acids plus vitamins (β-carotene, C, E, B_1, B_2, B_6, B_{12}, folic acid, pantothenic acid, nicotinamide, biotin) and trace minerals (zinc, magnesium and selenium) dosed according to their Recommended Dietary Allowances to facilitate amino acid metabolism. The experimental group showed a significantly better improvement of depression (Ham-D scores) and a higher responder rate than those of the placebo group. The results suggest that oral application of a deficit oriented amino acid mixture can improve the therapeutic outcome of an anti-depressant.

1.4 Phytochemicals

While many nutrients may be implicated in the role of mood disorders, it is also important to note that phytochemicals, a ubiquitous class of plant secondary metabolites, may also have relevance. It is recommended that humans eat a high proportion of fruits and vegetables, therefore providing abundant phytochemicals that promote antioxidant activity and positive modulation, either directly or indirectly, of the cellular and tissue

redox balance. Recent research suggests that molecules with a chemical structure compatible with a putative antioxidant capacity can perform roles independent of such capacity, interacting with cellular functions at different levels, such as affecting enzyme activities, and binding to membrane or nuclear receptors as either an elective ligand or a ligand mimic. Inductive or signalling effects may occur at concentrations much lower than that required for effective antioxidant activity. Therefore, the "antioxidant hypothesis" may be biasing the understanding of the molecular mechanisms underlying the beneficial effects of various classes of food items[214;215].

1.5 Other Nutritional Compounds

Other nutritional factors examined in mood disorders include carnitine, S-adenosyl-L-methionine (SAMe), phosphatidylserine, caffeine, and betaine. Acetyl-L-carnitine, a derivative of L-carnitine, was shown to be effective for depression in the elderly[216;217]. A double-blind study of bovine cortex phosphatidylserine (acidic brain phospholipid) in geriatric patients with mood disorders showed improvement in depressive symptoms[218]. Supplementation with SAMe, an important methyl group donor related to folate metabolism, has shown significant effects in depression[219]. As in the study of multi-ingredient formulas, these results illustrate that using interacting substances generally brings more success than using single-ingredient treatments.

When evaluating the nutritional intake of individuals with mood disorders, non-nutrient consumption such as caffeine should also be considered. Caffeine is a naturally occurring substance in coffee beans, cocoa beans, and tea leaves and is often added to drinks such as colas and "pep-up" drinks. It is a psycho-stimulant, and caffeine deprivation is associated with a withdrawal syndrome[220] causing depressed mood[221]. Caffeine intake has been positively related to stress levels and proneness to hallucination — believed to be due to increased cortisol response[222] — in general samples.

Betaine is found in dietary sources such as seafood, wheat germ, bran, and spinach. The principal physiologic role of betaine is as an osmolyte and methyl donor (transmethylation) in the methionine cycle. Inadequate dietary intake of methyl groups leads to hypo-methylation in many important pathways, including 1) disturbed hepatic protein (methionine) metabolism as determined by elevated plasma homocysteine concentrations and decreased S-adenosylmethionine concentrations; and 2) inadequate

hepatic fat metabolism, which leads to fatty accumulation and subsequent plasma dyslipidemia. These alterations may contribute to various conditions including cerebral diseases[223]. The growing body of evidence shows that betaine is an important nutrient for the prevention of mental disease; however, effects of the betaine pathways may largely be due to inadequate folate intake[224].

In addition to nutritional compounds specifically examined in mood disorders, there are a number of other nutrition components that have known activity in the brain and nervous system that likely have relevance. These include anthocyanins, curcumin, gingko biloba, glutathione, grapeseed, green tea, mannitol, miso, quercetin, and resveratrol[225;226].

1.6 Nutritional Risk Factors Associated with Mood Disorders

Various risk factors may mediate the relationships between mood disorders and nutrition status. Table 1.0.3 summarizes some common mental health factors that affect nutrition status.

Table 1.0.3: Factors that may affect nutritional intake in psychiatric populations*

Factor	**Description of effect on nutritional intake**
Psychotic symptoms	Delusions about food and visual hallucinations.
Social withdrawal and exclusion	Avoid mealtimes, embarrassed to eat with others and not shopping. Lack of access to health support (e.g., dietitian, dentist).
Overactivity in mania, anxiety	Unable to sit long enough to eat, eating "on the go" and increased energy output. May turn to food to soothe.
Memory impairment	Forgetting to eat. Forgetting a meal has been taken and overeating. Impaired ability to retain new information from nutrition counselling.
Lack of motivation or poor energy levels	No desire to shop or prepare food and poor food hygiene. Comfort eating. Exacerbates a sedentary lifestyle associated with subsequent weight gain.
Hospital admission	Usual preferences may not be adequately catered for. Alternatively, patients eat healthy and have a social mealtime so that diet improves.

*Table adapted from Abayomi J and Hacket A. (2004). "Assessment of Malnutrition in Mental Health Clients: Nurses' Judgement vs a Nutrition Risk Tool." *Journal of Advanced Nursing,* Vol. 45, No. 4, 430-437

Table 1.0.3: Factors that may affect nutritional intake in psychiatric populations* /continued	
Factor	**Description of effect on nutritional intake**
Physical changes	Possible swallowing difficulties, problems feeding self, and conditions requiring therapeutic diets.
Medications	Nutrient-drug interactions or nutrition-related side effects such as changed appetite, weight gain, gastrointestinal disturbances, and dry mouth. May be prone to use of alternative therapies (e.g., herbs).
Food insecurity	Limited income and resources making access to food challenging.
Mania	Associated with treatment non-adherence. Patients with bipolar disorder are also less likely to report that their provider discussed diet habits with them.
Weight changes	May lead to eating disorder. May be prone to food fads or use of alternative therapies.

*Table adapted from Abayomi J and Hacket A. (2004). "Assessment of Malnutrition in Mental Health Clients: Nurses' Judgement vs a Nutrition Risk Tool." *Journal of Advanced Nursing,* Vol. 45, No. 4, 430-437

Malnutrition refers to overnutrition (intake of nutrients in excess of requirements) and undernutrition (intake of insufficient nutrients to meet requirements)[227]. Under-nutrition is a well-documented consequence of entering a care setting. There is little evidence of the situation in mental health care, but one study[228] that compared mental health nurses' judgments of nutritional risk with the risk identified on a screening tool found that nurses failed to identify, or wrongly identified, a significant group of people who were at risk of malnutrition[228]. Risk of malnutrition was commonly associated with psychosis, and was underestimated in those with depression and who were middle-aged.

People with mental health needs are more likely than the general population to become obese for many reasons[229], including reduced motivation to address weight gain, impaired access to primary care, side effects of psychotropic medications, a diet that is high in fat, reliance on convenience foods, and a lack of regular exercise. The causes of obesity are multi-factorial but at the basis of the condition is the intake of energy beyond one's needs. Those with bipolar disorder may have unique challenges because of its

cyclical nature (i.e., alternating episodes of mania and depression). Mood lability can lead to periods of minimal contact between the patient and health care providers[230;231] which contributes to increased risk of medical comorbidity. Moreover, depressive episodes can lead to increased risk of cardiovascular disease through the effects of a sedentary lifestyle[232;233]. Previous research has demonstrated that compared to those without serious mental illness, people with bipolar disorder are more likely to report poor exercise habits, suboptimal eating behaviours — including having fewer than two daily meals — and having difficulty obtaining or cooking food[234].

Many of the health care costs of treating patients with mood disorders have been attributed to general medical comorbidities[235;236]. In particular, dyslipidemia, hypertension, and diabetes are thought to occur more frequently among patients with mood disorders than among the general population[237-239]. Furthermore, there is consensus that antipsychotic medications are associated with various degrees of weight gain and metabolic disturbance[240]. Evidence suggests that preclinical risk factors for obesity and cardiovascular disease are not consistently addressed by providers, largely because of time constraints limiting their capacity to provide preventive services.

Risk factors associated with nutrition that have been implicated in mood disorders include low income, substance use, social isolation, and drug-nutrient interactions. Low income is associated with food insecurity, broadly defined as limited, inadequate access to sufficient, safe, nutritious, personally acceptable food that meets dietary requirements for a healthy and productive life[241]. The impacts of food insecurity include poorer overall health due to higher rates of multiple chronic conditions (e.g., heart disease, obesity, high blood pressure, and diabetes)[242;243]. Large-scale epidemiological studies illustrate the frequent co-occurrence of substance use with psychiatric disorders[244;244]. The use of any substances can have significant effects on one's nutritional status. For example, smokers use more vitamin C and vitamin E due to oxidative stress which then contributes to low plasma antioxidant concentrations[245;246].

Another area of nutritional risk factors in mental health relates to the possible effects of some foods on neurodegeneration. Some research appears to suggest that serum-advanced glycation end products (AGEs) increase serotonin-induced platelet aggregation[247]. AGEs are a group of endogenous proinflammatory sugar-protein

compounds. Exogenous AGE, found in high sugar and fried foods as well as foods subjected to high and dry heat, are thought to contribute to the body's pool of AGEs[248].

1.7 Drug and Nutrient Interactions

The interactions of drugs and nutrients may be classified in four categories: 1) drug-nutrient interactions where the prescribed medication alters the bioavailability of the nutrient; 2) nutrient-drug interactions where the nutrient alters the effectiveness of the medication; 3) drug-nutrition interaction where nutrient intakes are altered because of the types and amounts of food consumed; and 4) nutrient-nutrient interaction where one's nutritional status alters the bioavailability of the nutrient.

Many of the drugs used in the treatment of mood disorders have significant drug-nutrient interactions. Anticonvulsants and antipsychotics can increase the metabolism of vitamin K, vitamin D, calcium, biotin, and folate, and can alter blood glucose and lipids[249;250]. Valproic acid may affect ammonia metabolism[251]. Individuals taking monoamine oxidase inhibitors may develop hypertensive crisis if they consume foods high in tyramine[252]. Some antidepressants may cause weight gain and fluid retention; others may cause weight loss[253]. Phenothiazines may cause riboflavin depletion[254], neuroleptics may cause xerostomia, and benzodiazepines may alter taste. Lithium may alter gastrointestinal, electrolyte, and glucose metabolism functions[255;256].

Given the various nutrient-related side effects of psychiatric medications, it is possible that many patients taking these as treatment are not receiving sufficient micronutrients for optimal function. For example, the triage theory posits that some functions of micronutrients (approximately 40 essential vitamins, minerals, fatty acids, and amino acids) are restricted during shortage and that the functions required for short-term survival take precedence over "secondary" functions[257]. Micronutrient deficiencies (even at the subclinical level) induced by psychiatric medication therapy may therefore have long-term health implications. There is evidence that different nutrients may enhance or augment the clinical effect of psychiatric medications. Most of these include the combination of anti-depressants with B vitamins, folic acid, or SAMe[34] and, as previously cited, smaller studies have suggested that magnesium supplementation combined with lithium, haloperidol, clonazepam, or verapamil may help reduce manic symptoms[149;255].

1.6 Nutrient Intakes and Mood Disorders Research

To date, two studies have assessed nutrient intakes in mood disorders; specifically in individuals experiencing depressive episodes. Dietary data from a small sample of depressed women and men revealed that many consumed a diet that supplied less than the Recommended Dietary Allowance (RDA) for four of the essential nutrients, due to intake of insufficient calories[258]. The other study compared depressed and non-depressed individuals and found that nutrient intakes were dissimilar for protein and carbohydrates[259]. Current data based on the *Dietary Reference Intakes* (*DRIs*) are needed to estimate group prevalence of nutrient inadequacy. Appendix A summarizes the *DRI*s and their use in nutrient intakes for a group. Current nutrition guidelines for those with mood disorders cover only those patients taking antipsychotics[260].

A research initiative of the author included the nutrient analysis of 15 individuals with verified bipolar disorder. Participants completed 7-day food records, and macro- and micronutrients were analyzed using Food Processor nutrient analysis software at the University of Alberta. At least 40% of the 15 individuals studied did not meet their daily requirements (based on the *DRIs*) for 17 micronutrients. Furthermore, 7% (95% Binomial Exact CI 0.002 to 0.319) exceeded the Tolerable Upper Limits for niacin.

1.7 Determinants of Food Intake

Previous discussion in this chapter has focused on the nutrition-related causes of mood disorders. Another important consideration in linking nutrition and psychiatric illnesses are the leading determinants of morbidity and mortality in this population that are rooted in behavioural choices related to eating habits. In this section, intermediate factors related to nutrient intakes in individuals with mood disorders are considered. There are complex interactions among the determinants of eating that include individual and collective (social and environmental) determinants of personal food choice. A synthesis of the literature[261] revealed that individual determinants of personal food choice (physiological state, food preferences, nutrition knowledge, perceptions of healthy eating, and psychological factors) help explain eating behaviour. Collective determinants that include contextual factors, such as the interpersonal environment created by family and peers; the physical environment, which determines food availability and accessibility; the economic environment, in which food is a commodity to be marketed for profit; and the

social environment, in which social status (income, education and gender) and cultural milieu are also determinants of eating[261]. How these factors particularly influence individuals with mood disorders is not yet known.

Many hypotheses have been advanced to account for the apparent relationship between mood state and food intake. Eating may be a learned response to certain mood states because it has become associated with a lessening of the unpleasant mood. Or one may be so preoccupied with the unpleasantness of the mood state that eating is no longer of importance. There also appears to be strong evidence for a biochemical link between mood and appetite. Some research has focused on the similarities between the diagnostic categories of anorexia and major depressive illness[262]. Disturbance of appetite is so often associated with a major depressive episode that it is included in the diagnostic criteria for this affective syndrome. This implies that there are common biochemical pathways underlying mood, appetite, and disordered eating behaviour. It is clearly understood that variations in mental health contribute to eating. Furthermore, the relation between eating and mental health is bi-directional: one's mood or psychological state can affect what and how much one eats, and eating affects one's mood and psychological well-being[263].

Preliminary work by the author regarding food choice behaviour in depression was undertaken using public use data from the 1996-97 Government of Canada's National Population Health Survey. A detailed description of that survey's methodology is available in a Government of Canada publication[264]. Participants in the survey were randomly selected within geographical clusters across Canada. This dataset allowed for the comparison of various nutrition-related factors between samples with and without depression based on scores of the Composite International Diagnostic Interview Short Form for Major Depression where a score of 0.9 indicated a 90% likelihood of a positive diagnosis of a major depressive episode (MDE)[265]. The total number of respondents was 17, 244, of whom 669 reported depression, 16,058 reported no depression, 487 reported no depression but were taking anti-depressants, and 30 provided insufficient information for coding. Comparisons of the depressed and non-depressed samples indicated that those who were depressed were more likely to have food insecurity issues (OR = 2.54, 95% CI 2.02 to 3.19), need help preparing meals (OR = 2.88, 95% CI 2.43 to 3.42), have self-reported food allergies (OR = 1.76, 95% CI 1.57 to 1.97), be of insufficient weight (OR =

1.65, 95% CI 1.45 to 1.88), and be more likely to indicate that the most important thing they could do to follow a healthy diet included taking vitamins (OR = 2.43, 95% CI 1.85 to 3.16). The results of this data analysis suggest that those with depression were at heightened nutritional risk based on health-related and sociodemographic factors.

1.8 Chapter Summary

This chapter examined the research literature related to nutrition and mood disorders. Many of the investigations conducted to date have focused on clinical and case-control studies. Much of the research also has many methodological limitations related to sample selection and sizes, heterogeneity in both psychiatric measures and diagnostic criteria, and inadequate control of confounders, thereby limiting generalizations that can be drawn about links between nutrition and psychiatric function.

Of the macronutrients, carbohydrates are believed to have the most profound impact on brain function due to their multiple roles in CNS function. Specific amino acids (i.e., tryptophan and tyrosine) may have relevance in mood disorders, but more studies are needed to evaluate their efficacy and safety as treatments. Research suggests that the essential fats, particularly the omega-3 fatty acids, are important in mental function and show promise as treatment for mood disorders. Similar to the amino acids, more studies are required to determine which essential fats and dosages are relevant.

Of the micronutrients examined to date, most of the evidence suggests that folate, vitamin B_{12}, magnesium, zinc and chromium have important roles in mood disorders; while evidence suggests that SAMe has significant effects in unipolar depression. It is important to note, however, that few human studies have been conducted with many of the nutrients. The micronutrient research also has an apparent lack of investigations regarding the role of specific foods (providing several co-occuring nutrients) in mood disorders. This suggests a need for research into the individual and combined roles of nutrients in mood disorders. Finally, to further the understanding about how nutrition and mood disorders may be linked, the underlying frameworks and determinants require further investigation.

The dietetics and mood disorders research has largely been dominated by intervention studies using a variety of single vitamins, minerals, dietary neurotransmitter precursors, and other nutrient factors as treatments. However, these only partially explain the

relationships between nutrition and psychiatric function. Evidence suggests that those with mood disorders are at heightened nutritional risk due to issues related to food inaccessibility and susceptibility to drug-nutrient interactions. Some preliminary data also suggests that those with mood disorders have a high prevalence of inadequate intakes for various nutrients. Thus, there is an apparent need for research that details the actual dietary habits of those with mood disorders and how they may differ from general populations. This will help determine strategies to improve overall health outcomes in this vulnerable group.

The purpose of this study is to help bridge the gap between knowledge of the nutritional issues of this population and how that may be applied in nutrition interventions. In particular, the study is intended to provide detailed descriptive information about the nutritional status of people within this population as well as the factors that may be associated with food intake and psychiatric function.

Due to the limitations of current formats of nutrient analysis software systems and nutrition standards, only specific nutrients were examined in this study. These included intakes of the macronutrients, vitamins B_1, B_2, B_3, B_6, folate, B_{12}, C, pantothenic acid, calcium, iron, magnesium, phosphorus, sodium, potassium, and zinc. As this literature review suggests, there are other nutrients of interest (e.g., dietary neurotransmitter precursors, phytochemicals). However, they were not considered because they cannot be adequately measured with the systems currently available. In the following chapter, the methodology of the study is detailed.

CHAPTER TWO: METHODOLOGY

The research literature reviewed in Chapter One identified that many nutrients are important to mental health; however, there is little known about the actual nutrition-related behaviours of individuals with mood disorders and how these may relate to psychiatric function. This gap suggested the importance of an investigation involving observation of a subset of individuals with mood disorders, their actual food intake and psychiatric function. Thus, a cross-sectional research design was selected. The data were collected systematically to define nutrient intake and food-choice behaviour in adults with mood disorders, and the design allowed for control of relevant variables to identify nutritional patterns and psychiatric function in this study population.

2.1 Research Question, Objectives, and Hypotheses

Research Question

The intent of this study was to evaluate nutrient intakes and their relationship with mental status in community-based adults with bipolar or major depressive disorder. This investigation sought to answer the overall question "When examining nutrient intakes, sociodemographic and health-related factors, and biochemical indicators, are adults (≥ 18 years) with mood disorders at nutritional risk?" To address this overall question, a subset of inquiries was developed:

1) Compared to the *Dietary Reference Intakes* (*DRI*s), is there evidence of inadequate (i.e., less than the Estimated Average Requirement or Adequate Macronutrient Distribution Ranges) and/or excess (i.e., greater than the Tolerable Upper Intake Levels or Adequate Macronutrient Distribution Ranges) nutrient intakes in individuals with mood disorders?
2) Do nutrient intakes of individuals with mood disorders differ from those in a general population sample?
3) Do blood levels of selected nutrients in individuals with mood disorders occur outside the reference ranges?
4) Do nutrient intake levels in individuals with mood disorders differ significantly according to specific sociodemographic, mental health status and health-related variables?

5) What sociodemographic, mental health status and health-related variables are associated with energy and macronutrient intakes in individuals with mood disorders?
6) What nutrients, sociodemographic, and health-related variables are associated with mental health status in individuals with mood disorders?
7) What nutrient intake, sociodemographic, mental health status and health-related variables are associated with blood levels of selected nutrients in individuals with mood disorders?

Objectives

The proposed study's primary, secondary, and tertiary objectives were:

1) *Primary*: to compare carbohydrate intakes of adults with mood disorders to nutrition standards and provincial nutrition survey data;
2) *Secondary*: to compare other nutrient intakes (i.e., protein, fat, fibre, cholesterol, vitamins B_1, B_2, B_3, B_6, folate, B_{12}, C, pantothenic acid, calcium, iron, magnesium, phosphorus, sodium, potassium, zinc) and non-nutritive substances to accepted nutrition standards and provincial nutrition survey data, and to determine if there is biochemical evidence of nutritional deficiencies in adults with mood disorders;
3) *Tertiary*: to determine whether macronutrient intakes as well as sociodemographic and health-related influences on food consumption in adults with mood disorders differ according to mood disorder subtype, psychiatric symptoms and functioning; and to examine relationships between nutrient intakes, sociodemographic factors, health-related factors, and symptom severity in adults with mood disorders.

It was hypothesized that 1) carbohydrate intakes will not meet all the nutrition standards (e.g., *DRI*s); 2) other nutrient intakes (i.e., protein, fat, fibre, vitamins B_1, B_2, B_3, B_6, folate, B_{12}, C, pantothenic acid, calcium, iron, magnesium, phosphorus, sodium, potassium zinc) and non-nutritive substances will differ from the nutrition standards; 3) there will be biochemical evidence of nutritional deficiencies; 4) macronutrient intakes and sociodemographic and health-related influences on food consumption will differ according to mood disorder subtypes and symptoms; and 5) a unique set of nutrient intake and sociodemographic and health-related factors will be associated with psychiatric status.

2.2 Outcomes

The primary, secondary, and tertiary measurements of this investigation are described in the following:

1) *Primary*: Mean carbohydrate intake measures derived as average values from the three-day food record (actual nutrient intakes). This macronutrient was considered to have the greatest impact on neurological function. Natural carbohydrates contain many essential nutrients and specialized chemicals (e.g., phytochemicals) needed for brain metabolism and they are one of the most widely distributed macronutrients in the food supply. If intakes of carbohydrates in the sample differ significantly from the general population, it is likely that many micronutrients (e.g., folate) will also differ.
2) *Secondary*: These included i) intakes of the two other macronutrients (protein and fat) derived as average values from a three-day food record (actual nutrient intakes); ii) intakes of the micronutrients derived as average values from a three-day food record (actual nutrient intakes); iii) intakes of non-nutritive substances derived as average values from the three-day food record (actual nutrient intakes) compared to the accepted nutrition standards (i.e., *DRI*); iv) blood indicators of nutrient status (i.e., red blood cell folate, vitamin B_{12}, vitamin B_6, vitamin E, and albumin). In order to control for confounding of blood lipid concentration between vitamin E level and serum cholesterol, total cholesterol was also measured. Originally selenium was included as a measure as well as the selenium-dependent enzyme glutathione perioxidase, a potential confounder of selenium status. However, the laboratories did not have adequate facilities to perform these tests. It should be noted that hereafter blood indicators of nutrient status will be referred to as biochemical indicators;
3) *Tertiary:* These included i) Macronutrient intakes (carbohydrates, protein, and fat), sociodemographic characteristics (age, gender, marital status, income, education), and health-related influences (nutrition education, dietary restraint, food security, smoking status, alcohol intake, physical activity, social contexts, and nutrition-related side effects of psychiatric medications) according to mood disorder subtypes as well as depressive symptoms (as measured by the Hamilton Depression Scale; Ham-D score > 7) and manic symptoms (as measured by the Young Mania Rating Scale; YMRS score > 12) in adults with mood disorders; and ii) The consumption of

nutrients, biochemical indicators, sociodemographic characteristics (age, gender, marital status, income, education as measured on the food selection questionnaire), health-related influences (nutrition education, dietary restraint, food security, smoking status, alcohol intake, social contexts, and nutrition-related side effects of psychiatric medications), mood disorder subtype, and mood symptoms.

2.3 Overview of Method

2.3.1 Sample

2.3.1.1 Ethics Approval

The study proposal, introductory letter, questionnaires, and consent form were approved by the University of Calgary's Conjoint Health Research Ethics Board.

2.3.1.2 Target Population

Non-institutionalized adults with mood disorders were recruited through the Mood Disorders Association of British Columbia (MDABC). Where appropriate, comparisons were made with the data from the British Columbia Nutrition Survey (BCNS). The target population for the BCNS was adults aged 19 or older residing in the province of British Columbia. Persons living in care or correctional facilities were excluded from the sample because their diets would be influenced by their institutional situation.

Exclusion Criteria

The following exclusion criteria were applied:

1) Investigators and their immediate families (i.e., spouse, parent, sibling);
2) those younger than 19;
3) those who did not have a sufficient level of understanding to complete all the questionnaires required by the study;
4) those with a current or lifetime diagnosis of any of the following according to DSM-IV criteria: schizophrenia, schizophreniform disorder, schizoaffective disorder, delusional disorder, psychotic disorder not otherwise specified, delirium of any type, dementia of any type, amnestic disorder; or any psychotic disorder due to a general medical condition, defined on the basis of self-report;

5) those with uncorrected hypo- or hyperthyroidism or with any degenerative neurological disorder, or who have not fully recovered from a cerebrovascular accident; and
6) women who were pregnant or breastfeeding as they have different nutrition requirements. People with other chronic physical conditions requiring dietary interventions were not excluded as comorbid physical conditions are prevalent in this particular group.

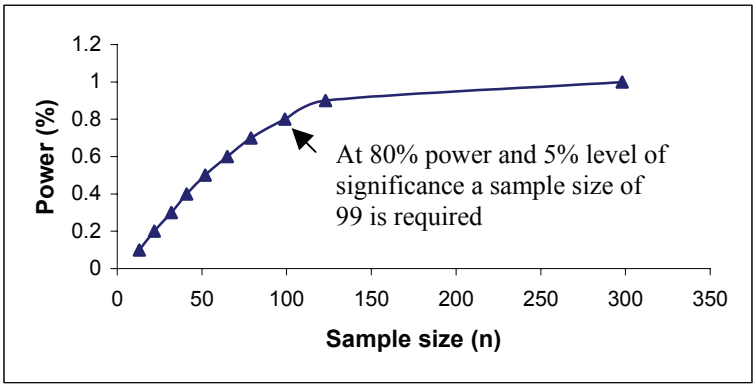
2.3.1.3 Sampling Frame and Size

The sampling frame for this study included members of the MDABC who resided in the lower mainland of British Columbia. Based in Vancouver, British Columbia, the MDABC is a non-profit network that provides support and education for people with a mood disorder and their supporters. The ages of members range from 16 to seniors and they have variable incomes (people on low income can have their fees waived).
Sample size requirements were based on the following criteria:

1) The study's primary research question that focused on the determination of nutrition risk in this population. Nutrition risk in this context refers to under nutrition as determined by suboptimal nutrient intakes.
2) The primary measure was carbohydrate intake (grams/day), a key macronutrient associated with neurological function[266]. Foods that are a major source of this macronutrient also contain most of the micronutrients associated with neurological function (e.g., folate, vitamin B_6). Carbohydrates are the most widely distributed macronutrient in the food supply. If intakes are suboptimal, it is highly likely that several micronutrients will also be compromised;
3) When the sample size is 99, a single group t-test with a 0.05 significance level has 80% power to detect the difference between a null hypothesis mean of 285 g/day of carbohydrate intakes and an alternative mean of 245 g/day of carbohydrate intakes, assuming the standard deviation is 140[258]. This represents a 15% relative difference between the expected level of carbohydrate intake in the population and in people with mood disorders; and
4) Rate of drop-outs or loss to follow-up was estimated at 10%[44].

The estimated rate of loss to follow-up brought the number of participants required to 109. The sample size provided for at least 80% power to detect differences in intakes of the other nutrients (Figure 2.0.1). Using a sample size formula applicable to the estimation of inadequate levels of the biochemical indicators, within a maximum 0.2% error rate (based on the formula where the error rate = 1.96 ÷ [(n ÷ p (1-p)] * 0.5), 50 participants providing the indicated biochemical measurements were required. Based on the preliminary data from the investigator's prior research, using a two-sample t-test with mean (± SD) carbohydrate intakes between those who were symptomatic (148 ± 36) and asymptomatic (256 ± 22) for manic mood state and the same assumptions as for the primary measurements, a minimum of 80% power is provided to detect differences. Finally, it was estimated that for the proposed regression analyses the number of independent variables would typically be 5. Following Kleinbaum's[267] guideline of a variable to subject ratio of 1:10 for linear regression modeling, a minimum sample size of 50 would be sufficient for the proposed statistical analyses. The membership of the MDABC is 1400 people, thus it was likely that at least 7% (n = 99) would agree to participate in the study.

Figure 2.0.1: Power vs. sample size



2.4 Recruitment

The MDABC helped develop procedures to determine summer versus fall/winter data collection. Participant lists were based on postal codes.

A letter from the University of Calgary was sent to a total of 146 (73 in summer and 73 in the fall/winter) randomly selected MDABC members, inviting them to participate in a study of food habits and factors that influence those habits (Appendix B). The letter described the importance of the study in terms of enhancing the understanding of eating habits of those with mood disorders, but potential participants were clearly advised that their participation was voluntary.

The letter was followed by a telephone call from the MDABC office, requesting permission to release potential participants' names and numbers to the study's research coordinator. All attempted contacts with a potential respondent to arrange an appointment were recorded on the Record of Calls and Appointments Form, which collected information about reasons for the "non-contact" (e.g., no answer, telephone line busy, not at home, or the telephone not in service), the date and time for the scheduled interview appointment, or negative outcomes. This record helped to ensure that all follow-up attempts were made at different times of the day and week.

A minimum of 6 attempts was made to contact a respondent. No messages were left as the potential respondent was to be the first person informed about the request to participate in the study. If a close family member (i.e., spouse or parent) insisted on taking a message, the caller left a general message about having called on behalf of the University of Calgary but offered no information about the study. If the caller repeatedly reached an answering machine, he or she left a message on the third attempt. The message asked the respondent to contact the caller about a "Nutrition Project." After leaving a message, the caller continued to telephone the person at different times of the day and days of the week until a minimum of 6 attempts at contact had been made. A maximum of 10 attempts were made.

If the first call and a check of the local address did not locate the potential respondent (e.g., not known at this number), the caller attempted to find out where he/she now lived. The interviewer tried a minimum of three resources such as criss-cross and online directories, and personal contacts. Even after a reasonable effort and investigation, some potential respondents could not be located or contacted. These cases were recorded as "Not Resolved" on the Interview Status Form.

If the caller did not make contact, the file was closed and coded accordingly. If the individual refused to participate in the study, the interviewer attempted to complete a non-response questionnaire (Appendix B) and the file was closed and coded accordingly. The non-response questionnaire was used to determine if those who refused to participate in the study differed systematically from those who did participate in terms of habits and lifestyle, for example. The questions dealt with types of bread and milk eaten, vitamin/mineral supplements taken, current smoking behaviour, marital status, and education level because studies have found that these are significant factors in the intakes of many nutrients[268]. Having completed this short questionnaire, the potential participant was asked if he/she would like to reconsider his/her decision to not participate. Some "converted" at that point but the actual number was not monitored.

If the individual agreed to participate and proved eligible for the study (e.g., not pregnant), the interviewer scheduled an appointment. In some cases, the individual was located but could not participate due to language difficulties, illness, or extended absence (e.g., working at a logging camp). These files were closed and coded accordingly.
The research coordinator called the individuals who agreed to participate and outlined the study to them, explaining that a consent form and three-day food record would be mailed out (Appendix B). She trained the respondent by telephone to complete the food record (reporting two weekdays and one weekend day that were non-consecutive). To ensure accurate estimates of food amounts, food pictures and a handy serving sizer were also mailed out and their use reviewed with the participant[269]. The dietitian contacted the respondent a few days after the food records had been sent to confirm that they had arrived and to remind the participant to complete the records. Participants were told to start their food records within 7 days prior to their scheduled interview.

During the first telephone call, participants were asked to provide a blood sample for various nutrient indicators. If they agreed, a lab requisition with instructions that the bloodwork must be based on a fasting state was sent to them with the consent form and three-day food records. The request for blood samples continued until blood had been collected from 50 people.

2.5 Study Components

2.5.1 Confirmation of Diagnosis and Instruments to Measure Mental Health

Mood disorder diagnosis of each potential subject was verified with the Structured Clinical Interview for DSM-IV Axis I Disorders – Patient Edition (SCID-P including the Global Assessment of Functioning[270] Scale)[271], administered by a trained interviewer. Reliability studies of the SCID indicate a kappa index of 0.85 for bipolar disorder and 0.64 for major depression[272].

2.5.1.1 Global Assessment of Functioning (GAF) Scale

The GAF scale considers psychological, social, and occupational functioning on a hypothetical continuum of mental health-illness. It ranges from a score of 1 (i.e., "persistent danger of severely hurting self or others (e.g., recurrent violence) or persistent inability to maintain minimal personal hygiene or serious suicidal act with clear expectation of death") to 100 (i.e., "superior functioning in a wide range of activities, life's problems never seem to get out of hand, is sought out by others because of his/her many positive qualities. No symptoms."). The scale does not include impairment in functioning due to physical (or environmental) limitations. The rating of overall psychological functioning on a scale of 0–100 was operationalized by Luborsky in the Health-Sickness Rating Scale[273]. Spitzer and colleagues developed a revision of the Health-Sickness Rating Scale called the Global Assessment Scale (GAS)[274]. A modified version of the GAS was included in the DSM-III as the GAF Scale. The GAF is believed to be a reliable (ICC = 0.81) and valid measure of psychiatric disturbance in mentally ill samples[270;275].

2.5.1.2 Hamilton Depression and Young Mania Rating Scales

The trained interviewer also administered two measures to assess the severity of current mood symptoms: the Hamilton Depression Scale (Ham-D; to assess depression), and the Young Mania Rating Scale (YMRS; to assess mania). The Ham-D[276] is a 17-item measure that evaluates depression symptoms. Inter-rater reliability has been shown to be quite high ($r = 0.90$)[277;278] and the cut-off score normally used to define remission is 7[279]. The YMRS comprises 11 items[280], four of which are rated on a scale from 0 (asymptomatic) to 8 (severe symptoms). The remaining items are rated on a scale from 0 (symptom not present) to 4 (symptoms extremely severe). Items such as irritability,

speech, content of what they said (e.g., normal, grandiose ideas, hallucinations, etc.), and disruptive-aggressive behaviour are given twice the weight of others in order to compensate for the condition of severely ill subjects. Acceptable validity and reliability were demonstrated in adults rated during their first week in hospital, with a correlation of 0.93 between raters[280]. A frequently employed cut-off score used to define remission in adults is 12. Individuals experiencing mixed episodes would have a score indicative of symptoms on both the Ham-D and YMRS.

2.5.1.3 Drug Abuse Screening Test

To identify alcohol and drug use among the study population, the psychiatric interviewer administered the short (10-item) version of the Drug Abuse Screening Test (DAST-10)[281], a "yes-no" questionnaire that assesses the extent of problems related to drug and alcohol as reported by the respondent. The total score is based on the total number of negative consequence items endorsed (range 0-10). According to previous studies, the DAST demonstrated adequate internal consistency and temporal stability. Furthermore, criterion-related, concurrent, and discriminant validity have been evaluated; research suggests that the scale has good psychometric properties when used with psychiatric outpatients[280].

2.5.2 Instruments to Measure Food Intake and Nutrition Factors

The nutrition measurement had three main sections: the three-day food record, the Food Frequency Questionnaire, and the Food Selection Questionnaire (Appendix B). The Food Selection Questionnaire also contained demographic and anthropometric questions.

2.5.2.1 Three-Day Food Records

Dietary intake was assessed in a personal interview by the three-day food record (i.e., two weekdays and one weekend day that were not consecutive). Details affecting nutrient composition — such as added condiments, spreads, salt, and cooking methods — were recorded, and whenever possible, recipe ingredients, product brand names, and grocery store or restaurant sources were included. The accuracy of the three-day food record method depends on the memory and honesty of the respondents. Because of social desirability bias, respondents tend to over-report consumption of foods perceived to be

healthy and under-report consumption of foods perceived to be unhealthy[282]. There is a tendency to under-report energy intake, especially by overweight individuals[283;284]. In an attempt to obtain accurate intake data, the study used a standardized "multi-pass" system of questioning with unbiased, supportive, and non-judgmental probes. This involved the interviewer and respondent reviewing the record three times to obtain successively more detailed and complete information. Portion sizes were estimated using a 25-item kit with three-dimensional food models and household measures to improve participants' recall accuracy. The advantages of this method were that it was easily administered and had a low respondent burden.

2.5.2.2 Food Frequency Questionnaire (FFQ)

A semi-quantitative food frequency questionnaire was used to obtain detailed information on the usual consumption of various foods of interest. The Food Frequency Questionnaire (Appendix B) focused on fat intake and some foods that are eaten less often or seasonally (e.g., broccoli). Participants were asked about their frequency of consumption during a specific time period (past day/week/month) and their average portion size of specific food items. The FFQ also included questions about the consumption of added fat and reasons for choosing foods. Studies on the reliability of FFQs have found correlations ranging from 0.5 to 0.7 for nutrient intakes and 0.6 to 0.7 for frequently eaten foods and beverages[285]. The simultaneous use of food records and FFQ tends to obtain a true representation of individual consumption[286;287].

Information on the type, amount, and frequency (e.g., yesterday, monthly) of nutritional and non-nutritional supplements was also collected as part of the FFQ (Appendix B). Whenever possible, information was recorded directly from the labels, such as brand names and DINs (drug identification numbers). For the study, the definition of non-nutritional supplements covered everything that was not vitamin and/or mineral (including herbal, botanical, and homeopathic preparations).

2.5.2.3 Food Selection Questionnaire (FSQ)

The FSQ's content was chosen to examine food security, physical activity, weight, body image, and other nutrition-related variables (Appendix B). It was based on the BCNS's Nutrition and Physical Activity questionnaire. Where possible, the questions selected were based on previously validated questions.

The questions derived from the BCNS were originally tested on 20 healthy adults by University of British Columbia researchers to ensure that the questions were clear and specific. In addition, questions that were not part of standardized questionnaires were revised in accordance with the recommendations of a plain language review.

Healthy Weight and Body Image

The healthy weight and body image questions focused on perceived weight status, weight change efforts, body image and eating behaviour[288;289], and bodily comfort. Parts of the Three-Factor Eating Questionnaire (TFEQ)[288] were included to assess eating behaviour (question 17). The measurement tool assesses three dimensions of eating perceptions/behaviour: cognitive restraint of eating, which is the perception that one is attempting to limit the amounts consumed in an effort to control weight; disinhibition of eating, which is the perception of losing control over food intake; and perceived hunger. The original scale contains 21 items that assess restraint, 16 items that assess disinhibition, and 14 items that assess hunger. Because the TFEQ was too long to be included in its entirety, items with the highest item-total correlations with the total restraint and disinhibition subscale scores were selected for inclusion[290], resulting in 5 items from the restraint subscale and 4 items from the disinhibition subscale. The selection of these questions was also consistent with what was used in the British Columbia Nutrition Survey to allow for direct comparisons to this provincial survey data.

Food Security

Food security means always having dignified access to sufficient food that is safe, nutritious, and personally appropriate and that has been produced in a manner that is environmentally and socially sustainable. A screening question from the Statistics Canada National Health Population Survey was used to identify participants who experience food insecurity[291]. A question from the Human Resources Development Canada Food Insecurity Supplement was used to determine the prevalence and severity of food insecurity among identified participants[291]. The third question dealt with participation in food action programs (e.g., community kitchens).

Demographic Questions

Date of birth, gender, marital status, family composition, education, and income were collected to characterize the sample and to allow for comparisons to various demographic groups. Information on cigarette use was also collected.

Anthropometric Measurements

Height and weight were needed to calculate body mass index (BMI). Measurements were taken according to protocols established by Health Canada and with the awareness of potential personal sensitivities around body size and shape. The weight scale was calibrated weekly, and a setsquare and measuring tape were used to measure height. Participants were asked to remove their shoes, hats, any heavy clothes, and any heavy items from their pockets for this final phase of the interview. The weight measurement was not adjusted for clothing. Weight and height were measured and recorded once. Interpretation of BMI was in accordance with accepted standards[292].

Other Health-Related Information

To assess knowledge and attitudes related to diet, questions that measure the pursuit of health, identify coexisting nutrition-related disease conditions, and assess the impact of nutrition education programs on food-choice behaviour were included. For FSQ questions with many response categories, laminated cards were used to show the categories to facilitate accurate responses.

2.6 Biochemical Measurements

The assessment of nutrient intakes and dietary risk factors is an incomplete determinant of nutrient status as levels of a nutrient in blood or tissues can be affected by, for example, genetic or disease factors. The choice of biochemical indicators was based on reliability figures, nutrients most likely to be related to neurological function, and feasibility of measurement. These measurements included RBC folate, vitamin B_{12}, vitamin B_6, vitamin E, iron, and albumin. Information on the representative values of validity from the literature for each of the measures include the following correlations of estimated dietary exposure and biochemical levels: $r = 0.51$ for RBC folate[293], $r = 0.61$ for vitamin B_6[294], $r = 0.65$ for vitamin E[295], and $r = 0.22$ for iron[296]. Furthermore, in NHANES I[294], albumin was a significant predictor of protein intake.

In addition to the above noted biochemical indicators, total cholesterol was measured to control for confounding with vitamin E level. All specimen collection and analyses were done by BC Biomedical Laboratories and BC Children's Hospital (vitamin E). All analyses were in accordance with accepted standards[297].

2.7 Study Staff, Training, and Pilot Work

The study staff consisted of two registered dietitians (one of whom was the research coordinator), four clinical interviewers, and support staff persons. The clinical interviewers were graduate students from the clinical psychology program at the University of British Columbia. They were trained to use the SCID and other psychiatric measures by a trained SCID interviewer from the University of Calgary. All the interviewers remained with the study for the full 7 months of data collection. The research coordinator was responsible for overseeing all aspects of data collection, including tracking the progress of interviewers, verifying the completed questionnaires prior to data entry, preparing the training manual, and organizing the training sessions. The support persons were MDABC office staff. They helped with booking appointments and in directing potential participants' questions about the study. They also facilitated the disbursement of honoraria.

All staff completed full training prior to the study. The research coordinator developed and conducted the training sessions of the study protocol with the interviewers and support staff persons. The training session focused on administering the questionnaires and locating and contacting potential participants. It also covered the completion of administrative, control, and tracking forms, and offered many opportunities for practice.

Pilot work consisting of several practice sessions using all measurement instruments was carried out with 22 healthy volunteers as well as with individuals with mood disorders. The pilot sessions were conducted at the MDABC.

2.8 Data Collection

This study adapted the protocol and standard procedures established in the BCNS. The data collection was divided into two seasons to account for seasonal variations in eating habits, with the summer season running from August 1 to September 20 and the fall/winter season running from October 15 to January 30.

2.8.1 Interviewing Participants

The interviews took place in the MDABC office and typically lasted for 90 minutes. The interviewer first asked the individual to read and sign the consent form (Appendix B) documenting that all information would be held in strict confidence and that no personal identifying information would appear in any reports. Then one of the trained psychiatric interviewers administered the SCID-P (including the GAF), the Ham-D, the YMRS, and the DAST-10. If participants were eligible for the study based on the outcomes of the SCID-P and GAF (i.e., confirmed to have a mood disorder), they then followed this interview with a session with the registered dietitian.

The dietitian reviewed the three-day food records first to ensure that the information was complete. Participants were asked about the source of the item (home, fast food, etc.) and whether they considered the food item as part of a snack or meal. At the end of each reviewed day, participants were also asked if they had prepared most of the food they ate on that day. The dietitian then administered the FFQ and FSQ and measured height and weight. At the end of the interview, the respondent received a thank-you letter and an honorarium to cover the cost of coming to the office.

2.8.2 Quality Control of Data

Several checkpoints ensured the accuracy and completeness of the data. The interviewer reviewed the forms twice, once at the conclusion of the interview with the participant present and then again within 24 hours. The research coordinator then checked the original forms and added any new information. All the data were double-entered to ensure accuracy and the research coordinator did a final check of the data entry by reviewing the forms of each participant and the data entered. Custom-designed displays were used for the data entry for the questionnaires, with some automated validations being built in to ensure a certain degree of consistency and data quality. For example, data in the food selection questionnaire indicating that a participant took supplements was cross-referenced with the data collected on actual supplement intake.

2.9 Data Entry and Analysis

2.9.1 Data Entry

2.9.1.1 Psychiatric, Food Frequency, and Food Selection Questionnaires

All the completed forms were provided to the research coordinator. Data from the SCID-P, DAST, YMRS, Ham-D, FFQ, and FSQ were entered by a research assistant.

2.9.1.2 Three-Day Food Records

An undergraduate nutrition student, trained by the research coordinator, used the software program ESHA — which contains the Canadian Nutrient File[298] (CNF) and a recipe database of nearly 3000 recipes — to record the data from the food records. The CNF is a food composition database containing average values for up to 115 nutrients in 4668 basic foods available in Canada.

Issues related to the recorded data that were identified during data entry were relayed to the research coordinator and research staff for clarification or correction. The research coordinator manually checked the data input for each record by comparing a printed copy of the computer-recorded data with the information on the original questionnaire or form. Input of food record data began with the entry of a key name for a food or mixed dish, followed by the selection of the exact or best match to the food or mixed dish from the options displayed. Existing computerized recipes were used for mixed dishes, with ingredient substitutions or the addition of new recipes entered as required. Amounts consumed were entered as weights or volumes.

2.9.1.3 Nutrient Supplement Data

Nutrient supplement data were entered by name and nutrient content. In the event of insufficient information, the strength of the supplement was selected by default that was determined by the research coordinator. Standard conversions were used to translate nutrients into one type of measure for each as outlined in Table 2.0.1.

Table 2.0.1: Conversions for nutrient contents of supplements		
Folate	1 mcg dietary folate equivalent (DFE) = 0.5 mcg folate as a supplement (taken on empty stomach) or 0.6 mcg folate as a supplement consumed with meal	
Vitamin A	Many supplements report vitamin A content in IU or retinol equivalents (RE)	
	1 (RE) =	1 mcg Retinol or 6 mcg beta carotene or 12 mcg other provitamin A carotenoids or 3.33 IU from animal food (Retinol) or 5 IU from mixed foods or 10 IU from plant foods (carotenoids)
	1 Retinol Activity Equivalent (RAE) =	1 mcg Retinol or 12 mcg beta carotene or 24 mcg other provitamin A carotenoids
		RE, if RE = Retinol or RE x 0.5, if RE = carotenoids
	IU Vitamin A =	0.3 mcg Retinol or 3.6 mcg beta carotene
	For vitamin A supplements and foods fortified with vitamin A: 1 RE = 1 RAE	
Vitamin D	40 IU vitamin D = 1 mcg of cholecalciferol (D_3) or ergocalciferol (D_2)	
Vitamin E	Vitamin E is expressed in IUs, mg or in mg of alpha-tocopherol equivalents (αTE). One mg of vitamin E in αTE includes the various components of vitamin E and their activity levels.	
	1 IU vitamin E = 0.909 mg all-rac-alpha-tocopherol (synthetic)	
	1 mg alpha-tocopherol = 1 alpha-tocopherol equivalent	

2.9.2 Data Analysis

The data were analyzed by the statistical software programs Stata 7.0[299], SPSS[300], and SUDAAN[301]. This section briefly describes the method of estimating the distribution of usual nutrient intakes from food sources and supplements, the estimation of the proportion of nutrient inadequacy in the study sample, and additional analyses conducted by the researchers.

2.9.2.1 Assessing Normality of Variable Distributions

Variables were examined to determine whether they satisfied the assumptions of normality and symmetry using dot plots, box plots, quantile normal plots, and values for

skewness. When skewness occurred, variables were transformed (e.g., by using a square root or a log transformation). If normality was attained or skewness reduced to an acceptable value, analyses were conducted using the transformed variable; however, the results were presented using the units of the original variable. If a non-skewed distribution was not possible, analyses were conducted using non-parametric tests.

2.9.2.2 Estimating Response Rates and Differences

The method for estimating response rates included the calculation of four rates (see Chapter Three). Group comparisons were also made between responders and non-responders for the variables contained in the non-response questionnaire (Appendix B) using Student t-tests and Fischer Exact statistics.

2.9.2.3 Estimating Distribution of Food Intakes

Prior to examining nutrient intakes, food intakes were compared to nutritional standards to help understand patterns of nutrient inadequacies and excesses. A standardized approach that included portion assignments and food grouping information on a wide range of foods included in the CNF was used to subdivide diets into *Eating Well with Canada's Food Guide* (Canada's Food Guide) servings[302].

Food Group Assignments

Foods and recipes were assigned to the major food groups (i.e., grain products, vegetables and fruit, milk and alternatives, and meat and alternatives) or to "other foods," foods with excessive fat and sugar content. The grain products group includes cereals, grains, pasta, rice, bagels, bannock, pita, crackers, muffins, pancakes, and tortillas, but not cakes, cookies, Danish pastries, or doughnuts. All kinds and forms of vegetables and fruit are included in the vegetables and fruit group, including french-fried potatoes, pickled cucumbers, tomato ketchup, and dried cranberries. The milk and alternatives group includes fluid milk, cheese, and yoghurt. Cream, butter, and ice cream were considered "other foods." The meat and alternatives group contains all meat (including processed), poultry, and fish, plus eggs, legumes, nuts, nut butters, and tofu. "Other foods" includes those foods that are mostly fat (non-nut butter, margarine, oils, and cream), mostly sugar (jam, jellies, and candy), or mostly sugar and fat (cookies, cakes, chocolate bars, and ice cream); and high-calorie beverages (pop, lemonade, beverage mixes), high-calorie snack foods (chips, cheesies and crisps), alcohol, condiments, coffee,

tea, water, fruit drinks, and diet soft drinks. In addition, a system of 53 subgroups was used to classify foods according to Canada's Food Guide guidance statements.

Reported foods from the food records were coded either as basic foods or as recipes. For classification purposes, recipes were either treated as a single food (e.g., reconstituted dry milk, breaded pork chop) and assigned to one food group, or broken down into their individual ingredients with each major component then being assigned to a food group.

Portion Assignments

For all food groups except meat and alternatives, the number of portions was calculated directly by dividing the grams consumed by the portion size recommended by Canada's Food Guide. For meal replacements and the "other foods" group, the grams consumed were recorded and no portions were assigned. An additional step for meat and alternatives calculated "equivalent 50 grams" before portions were determined. For foods with fixed portion sizes, one portion would be considered as "equivalent 50 grams" of cooked meat, poultry, and fish. Then the "equivalent 50 grams" for those foods, plus the actual grams of cooked meat, poultry, and fish were totalled over the day with raw foods scaled to reflect cooked amounts. This total "equivalent 50 grams" amount was then compared to a "sliding window" scale based on 50–100 g meat/poultry/fish as one serving. The "sliding window" scale used was: 1) 0–25 grams = 0 serving; 2) 25–49 grams = 1/2 serving; 3) 50–99 grams = 1 serving; 4) 100–300 grams = 2–3 servings, and 5) 301–600 grams = 4–6 servings.

2.9.2.4 Estimating and Comparing Distribution of Usual Nutrient Intakes

The distributions of usual nutrient intakes from food sources were determined by averaging the nutrient intakes of the three-day food records. To determine nutrient intake from food plus supplements, the monthly nutritional supplement use recorded for each participant was expressed as a daily amount and was added to the usual intake obtained from food sources alone. For example, if someone took a multinutrient supplement containing 50 mg of vitamin C each day and a 500 mg vitamin C supplement four times in the past month, the monthly intake from supplements would be 3500 mg (50 mg x 30 + 500 mg x 4). This amount would be averaged over the month (3500 mg/30 = 116.7 mg) and added to the individual's usual vitamin C intake from food. The nutrient intake data were reported according to age (19 to 30 years, 31 to 50 years, 51 to 70 years) and gender

categories set out by the *DRI*s. These groups were then tested using Analysis of Variance (ANOVA) or Kruskal-Wallis tests to discover where significant differences existed.

2.9.2.5 Determining Inadequate and Excess Nutrient Intakes

To estimate the inadequacy of nutrient intakes, the Estimated Average Requirement (EAR) cutpoint method, developed by the Institute of Medicine[303], was used for all nutrients except iron. The EAR is a nutrient intake value that is estimated to meet the requirements of half the healthy individuals in a group. If an EAR was not available, Adequate Intakes (AI) were used instead. The AI is a recommended daily intake level based on observed or experimentally determined approximations by a group (or groups) of healthy people[304].

For iron, the assumption that the requirement distribution be symmetric is not met, particularly for menstruating women. In this case, "The full probability approach" where the full distributions of requirement and intake are used to estimate the probability that individuals with a particular intake would not meet their requirement[305-308], was used. The analysis in this study uses the full probability approach and incorporates a number of uncertainty factors, specifically, uncertainty around the fraction of oral contraceptive (OC) users[307] and uncertainty around the distribution of iron intakes. In these calculations, the proportions of self-reported OC users are estimated from the respondents aged 19–30 and 31–49, respectively, who were healthy and not pregnant. These two samples are extracted from the 1998-99 National Population Health Survey (NPHS) conducted by Statistics Canada. Uncertainty in these proportions is characterized by the 95th percentile confidence interval calculated taking the design of the NPHS into effect, using the specialized variance estimation software SUDAAN. The distribution of iron intakes is estimated from respondents' usual food consumption. The expanded probability method uses a two-dimensional Monte Carlo simulation to propagate the uncertainties through the probability approach.

To examine excess nutrient intakes, the Tolerable Upper Intake Levels (UL) of the *DRI*s were used. The ULs are a recommended daily nutrient intake that is likely to pose no risks of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the risk of adverse effects increases[304]. The adverse effects can vary from minor to critical.

2.9.2.6 Influence of Income, Education, and Marital Status on Nutrient Intakes

To determine whether specific demographic factors may have biased the nutrient intake results, it was necessary to determine whether education, income, and marital status were associated with nutrient intakes. Before analyzing the effects of these variables, each of them was reclassified. Income levels (n=10) obtained from the demographic information were reclassified as "low income" or "not low income." This assessment took into account the household income level, the number of occupants in the household, and the breakdown of children and adults in the household. The "low income" cut-offs were based on the 1999 Statistics Canada low-income measures by family type[308]. The 10 education levels were collapsed into three groups: high school or less, trade school or some university, and completion of university. Marital status was reclassified into two groups: not married (i.e., single, widowed, separated, divorced, and never married) and married (i.e., married or living common-law).

Nutrient intakes were assessed according to education level, income level, and marital status. For the analyses, the unadjusted nutrient intakes were initially transformed logarithmically, and the possible effects of education, income, and marital status were then examined using a General Linear Model procedure in SPSS. The dependent variable was the transformed nutrient of interest. The study design elements, including gender and age, were entered as fixed effects, as were the reclassified education and income variables. Unadjusted energy intake was entered as a covariate in each model. Both the main effects and two-way interactions of these variables were determined.

2.9.2.7 Comparison to the British Columbia Nutrition Survey

The British Columbia Nutrition Survey (BCNS), conducted in 1999 by the British Columbia Ministry of Health Services (Ministry), Health Canada, and the University of British Columbia (UBC), examined 1823 British Columbians aged from 19 to 84 years. The province-wide survey involved in-home interviews by trained health professionals utilizing a 24-hour recall, a food frequency questionnaire, and a general nutrition questionnaire focusing on physical activity, healthy weight and body image, and food security. Sociodemographic information and body measures were also collected. All the food, nutrient, and other nutrition-related data available from the BCNS were compared with the results of this study to identify any important differences. Full details of the

BCNS are located in selected reports[309;310]. Nutrient intake data was compared to the BCNS using Student's t-tests and the Mann-Whitney two-sample statistics.

Data from other national nutrition survey sources were considered as comparisons. For example, the BC subsample of Canadian Community Health Survey (CCHS) conducted in 2004 could have been utilized, however at the time of this study's nutrient analyses, data on nutrient intakes from vitamin and mineral supplements in the CCHS were still being validated. The most recent and complete data available at the time of the study implementation and analysis was the BCNS.

2.10 Other Statistical Analyses

2.10.1 Psychiatric Measures

Scores on the Drug Abuse and Screen Test (DAST-10), Young Mania Rating Scale (YMRS), Hamilton Depression Scale (Ham-D), and Global Assessment of Functioning (GAF) measurement tool were reported as mean values (± standard deviation) or as the median with 25th and 75th percentile, depending on the distribution. The proportion of those experiencing manic or depressive symptoms, or not, was also reported using standard cut-offs of the respective psychiatric scales.

2.10.2 Food Frequency Questionnaire (FFQ)

The FFQ-based food servings of each subject was computed by multiplying the relative frequency with which each food item was consumed (e.g., once a day equal to one) by the specified serving size. They were also correlated with the food group data derived from the three-day food records to examine how well the food record data represented usual intakes. The last section of the FFQ contained the supplement information. This data was reported in frequency distributions according to supplement type and compared to the BCNS data using Fischer Exact statistics.

2.10.3 Food Selection Questionnaire (FSQ)

For dietary risk factors measured on the FSQ, either the aforementioned descriptive statistics for continuous data or frequency tables for categorical data were derived and compared. Descriptions of BMI (weight {kg}/height{m^2}) status were in accordance with accepted age-appropriate standards[311].

2.10.4 Biochemical Indicators

For the biochemical indicators measured, means (± standard deviation), medians (25^{th} and 75^{th} percentiles), proportions lower than the reference standards[296], frequency histograms, box plots, and dot plots were analyzed.

2.10.5 Nutrients by Disorder, Psychiatric Symptoms and Functioning

To assess the hypothesis that nutrient intakes as well as sociodemographic and health-related influences on food consumption differ according to mood disorder subtypes and psychiatric symptoms and functioning, group comparisons were made between nutrient intakes, mood disorder subtype, and symptoms using ANOVA. To minimize Type I error rates for any multiple comparisons, Bonferroni's Correction was applied where necessary. The bivariate analyses also included examining correlations among nutrients and mental health status variables.

This stage of the analysis also included a contingency analysis that determined which diet factors were related to mood disorder symptoms. This included analysis for the presence of any confounders or effect modifiers by comparing crude to stratified odds ratios for meaningful differences and, where required, using the Mantel-Haenzel test to assess for the appropriateness of combining odds ratios and the test of homogeneity to assess for effect modification. Potential effect modifiers or confounders included age, gender, type of mood disorder, the presence of a health condition, special, diets, dietary restraint, food security, substance use, education, and income[309]. The outcomes of this analysis helped determine which variables to include in the multivariate analyses.

For parametric analysis, ANOVA was conducted. When significant main effects were observed, post hoc analysis, using Bonferroni's Correction, determined which groups differed significantly from one another. When significant interactions with sex were detected, one-way ANOVA was conducted for each sex. Cross-tabulations were conducted to examine the distributions of categorical data and Fischer Exact statistics were used to determine whether distributions differed from expected values.

Nonparametric analyses included the Mann-Whitney U test for comparison of two unrelated samples (e.g., men and women) and Kruskal-Wallis Analysis of Variance for

comparison of three or more unrelated samples (e.g., different age groups). Analyses were two-tailed and were considered significant at $p < 0.05$.

2.10.6 Prediction Models of Nutritional Status

To assess whether a unique set of nutrient intake, sociodemographic, and health-related factors were associated with disease status, prediction models to describe nutritional status, symptoms, and functioning were analyzed. Linear regression models were used to assess the effect of sociodemographic and health-related variables on energy and macronutrient consumption (protein, fat, and carbohydrates). The effect of each variable on macronutrient consumption was measured by the appropriate regression coefficient, interpreted, for example, as a change in protein consumption in grams per unit of change in the explanatory variable (e.g., income).

To further assess predictors of nutritional status, linear regression models as described above were analyzed with biochemical indicators (e.g., iron measurement) as the dependent measures and relevant nutrient intakes as well as the sociodemographic and health-related factors as the independent measures.

To examine nutrition-related factors that predict mood disorder symptoms and functioning, multiple regression models were also used. Three models were analyzed. One assessed dietary predictors (actual macronutrient intakes, and sociodemographic and health-related variables) and Ham-D scores as the dependent variable. Another assessed the same independent variables but the dependent variable was YMRS scores. The final model used GAF scores as the dependent variable. In these multivariate analyses, the effect of each variable on mood disorder symptoms was measured by the appropriate regression coefficient, interpreted, for example, as a change in the depression score for one unit increase in the independent variable (e.g., magnesium intake). Significance of the estimates was tested by individual t-tests. The adjusted R^2, an estimate of the proportion of dependent variable (e.g., Ham-D scores) that is explained by the regression model, was also reported.

To assess the plausibility of the multiple linear regression assumptions and overall model fit, a variety of diagnostics were assessed. To assess for linearity, the plot of dependent variable to predicted dependent variable was examined. To assess for

homoskesdasticity, linearity, and the presence of influential observations, plots of residuals versus predicted dependent variable were examined. Finally, to assess for the assumption that the residuals are symmetrically distributed, quantile normal plots of the residuals were assessed. Any influential data points were assessed using Cook's Distance values and plots. If outliers or influential data points were detected, separate analyses of the data were conducted to assess how they may have affected results.

2.11 Chapter Summary

In this chapter, the methodology of the study was detailed, including sampling strategy, survey instruments, staff training, data entry, and analysis procedures. In the following chapter, the methodology for reporting response rates, including the results, of the study is detailed. In addition, the sample's demographic characteristics are presented.

CHAPTER THREE: DESCRIPTION OF SAMPLE

The next four chapters outline the results of this investigation. This chapter provides descriptive analyses of the respondents while Chapter Four outlines details of food and nutrient intakes. Chapter Five provides descriptive analyses of other food- and health-related variables and Chapter Six outlines the multivariate analyses examining relationships among nutrient intakes and psychiatric functioning.

3.1 Response Rates

The calculation of the response rates was adapted from a Statistics Canada Methods and Standards Committee document[312] (Table 3.0.1). Overall, 97% of the names drawn for the study were resolved; that is, located and contacted to participate. Of those, 124 (88%) were in-scope or eligible. Individuals were considered out-of-scope or not eligible if they were dead, had moved out of the area, or exhibited at least one of the exclusion criteria (e.g., pregnant, lactating, living in an institution). Of those contacted and eligible, 3% refused to participate in the study and 2% did not show up for their appointment.

Upper and lower bounds of the response rate were calculated to account for the proportion of unresolved individuals who were likely to be in-scope. Rates that represent the upper bounds are calculated on the assumption that all unresolved cases are counted as out-of-scope or ineligible individuals. Alternatively, lower bounds are calculated on the assumption that all unresolved cases represent in-scope individuals. The true rate lies somewhere between these values.

As described in the previous chapter, the targeted sample size was 99 participants; however, as outlined in Table 3.0.2, only 97 responses were usable. Originally there were 99 participants with completed files, but one month after the study's completion, one participant contacted the Mood Disorders Association of British Columbia (MDABC) office to say that she had been pregnant at the time of the study. Three months after the study's completion, another respondent contacted the research coordinator to say he had intentionally fabricated most of the information provided in the interviews. By then, the trained clinical interviewers and other study staff were no longer available, and so only the 97 complete files were analyzed. This sample size allowed for 80% power to detect the difference between the null and alternative mean carbohydrate intake discussed in the previous chapter.

Table 3.0.1: Definitions of rates involved in the calculation of response rates

Rate	Definition	Equation
Resolved rate	Number of potential participants contacted to participate from the total number of contacts first attempted.	$\frac{\text{Number resolved}}{\text{Total attempted}}$
In-scope rate (eligible)	Number of potential eligible participants contacted. Out-of-scope means that these individuals were not eligible to participate (e.g., had moved or were deceased).	$\frac{\text{Number in-scope}}{\text{Number resolved}}$
Refusal rate	Number of potential participants who refused to participate over the number that were successfully contacted and met the inclusion criteria. This rate only includes those individuals who simply refused.	$\frac{\text{Number refusals}}{\text{Number in-scope}}$
Response rate	The response rate was based on the number of usable first interviews provided by the in-scope individuals and was dependent on if the unresolved individuals were eligible or ineligible to participate. The lower bound of the response rate is calculated assuming that all the unresolved cases were in-scope; upper bound assumes that all unresolved cases are out-of-scope.	Lower bound = $\frac{\text{\# responding - \# unusable}}{\text{\# in-scope + \# unresolved}}$ Upper bound = $\frac{\text{\# responding - \# unusable}}{\text{\# in-scope}}$

Table 3.0.2: Response status by season						
Summary status	**Summer**		**Fall/Winter**		**Total**	
Drawn	73		73		146	
Attempted	73		73		146	
Resolved	71		70		141	
In-scope (eligible)	68		56		124	
Interviewed	45		54		99	
Response usable	44		53		97	
	LB[1]	**UB[2]**	**LB[1]**	**UB[2]**	**LB[1]**	**UB[2]**
Response Rate	63%	65%	90%	95%	75%	78%

[1]LB is the lower bound of the response rate that is calculated assuming that all the unresolved cases were in-scope (eligible)
[2]UB is the upper bound of the response rate that is calculated assuming that all unresolved cases are out-of-scope (ineligible)

The overall response rate for the study was 75%–78%, considerably higher than the response rate of the British Columbia Nutrition Survey (BCNS) (42%–52%). Some reasons for non-response included the time commitment, confidentiality concerns, and inaccuracies in the sampling frame.

3.2 Comparison of Respondents to Non-respondents

Of the non-respondents, 44% (11/25) were willing to answer the short non-response survey (Appendix B). Statistical comparisons using Fisher's exact test showed no significant differences between the respondents and non-respondents for any of the variables measured (Table 3.0.3). The over-representation of university-educated participants in this study may introduce significant bias towards nutrition recommendations. It has been reported that groups with higher education will have lower alcohol and meat consumption and higher fruit, vegetable, and supplement intakes[312]. The effects of education and income on energy and nutrient intakes were therefore examined.

3.3 Demographics and Psychiatric Diagnoses

Demographic and psychiatric diagnoses information was also provided to characterize the study sample. The characteristics presented here were from information in the Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID), Global

Assessment of Functioning (GAF), Drug Abuse Screening Test-10 (DAST-10), the Food Selection Questionnaire, and the Food Frequency Questionnaire.

The agreement among the four clinical interviewers for diagnosis of condition and GAF, Young Mania Rating Scale (YMRS), and Hamilton Depression Scale (Ham-D) scores was very high (Table 3.0.4).

Table 3.0.3: Comparison of study respondents and non-respondents

Characteristic	Respondents (n=97)		Non-respondents (n=11)	
	Sample	%[1]	Sample	%[1]
Daily smokers:				
All	20	20.6	3	27.3
Males	8	40.0	3	100
Females	12	60.0	0	0
Bread consumers:				
All	83	85.6	10	90.9
Whole wheat, multigrain, rye, or pumpernickel	63	64.9	5	50
White	20	20.6	5	50
Milk consumers:				
All	97	100	11	100
Whole	4	4.1	0	0
2%, 1%, or skim	90	76.4	10	90.9
Other	3	3.1	1	9.1
Vitamin/mineral supplement usage:				
All	61	62.9	6	54.5
Males	12	19.7	2	33.3
Females	49	80.3	4	66.6

[1]For each characteristic, percentages by subgroups are based on the total respondents for that characteristic

Table 3.0.3: Comparison of study respondents and non-respondents /continued					
Characteristic	**Respondents (n=97)**			**Non-respondents (n=11)**	
	Sample	**%[1]**		**Sample**	**%[1]**
Education (bachelor's degree and above)					
All	30	30.9		2	18.2
Males	6	20.0		2	100
Females	24	80.0		0	0
Marital status:					
Married:					
All	37	38.1		6	54.5
Males	14	37.8		3	27.3
Females	23	62.2		3	27.3
Single, widowed, divorced, never married or separated:					
All	60	61.9		5	55.5
Males	14	14.4		3	27.3
Females	46	47.4		2	18.2

Table 3.0.4: Measures of agreement among clinical interviewers		
Measure	**Kappa**	**Agreement**
Diagnosis of condition	--	1.0
Global Assessment of Functioning	0.433 (SE = 0.334, 95% CI 0.22 to 1)	0.882
Young Mania Rating Scale	0.638 (SE = 0.327, 95% CI -0.003 to 1)	0.941
Hamilton Depression Scale	0.638 (SE = 0.327, 95% CI -0.003 to 1)	0.941
Drug Abuse Screening Test	--	1.0

Most participants had bipolar disorder (Table 3.0.5) and had been diagnosed between 1 and 37 years previously (median = 9.5 years; 25th percentile = 5; 75th percentile = 19). The average GAF score suggested that most were generally functioning well. Fewer than 11% reported a substance abuse problem (Table 3.0.6; questions 6 to 11).

Table 3.0.5: Diagnosis and Global Assessment of Functioning of study sample (n=97)

	Total (% of total sample)	**Males (% of subgroup total)**	**Females (% of subgroup total)**
Bipolar I or II (N (%))	58 (59.8)	19 (32.8)	39 (67.2)
Depressive Disorder (N (%))	39 (40.2)	11 (28.2)	28 (71.8)
Global Assessment of Functioning Scores (GAF)			
GAF (mean ± standard deviation)	62.7 ± 14.7	65.1 ± 2.9	61.1 ± 1.8

Table 3.0.6: Drug and Alcohol Screening Test (DAST-10) results (n=97)

Question	**Yes - Y (%)**	**No - N (%)**
1. Have you used drugs or alcohol other than those required for medical reasons?	52 (53.6)	45 (46.4)
2. Have you used more than one at a time?	10 (10.3)	87 (89.7)
3. Have you always been able to stop using them when you wanted to?	43 (44.3)	54 (55.7)
4. Have you had “blackouts” or “flashbacks” as a result of their use?	1 (1.0)	96 (99.0)
5. Have you ever felt bad or guilty about your use of them?	12 (12.4)	85 (87.6)
6. Has your spouse (or have your parents) complained about your involvement with them?	7 (7.2)	90 (92.8%)

Table 3.0.6: Drug and Alcohol Screening Test (DAST-10) results (n=97) /continued		
Question	**Yes - Y (%)**	**No - N (%)**
7. Have you used drugs or alcohol other than those required for medical reasons?	52 (53.6)	45 (46.4)
8. Have you used more than one at a time?	10 (10.3)	87 (89.7)
9. Have you always been able to stop using them when you wanted to?	43 (44.3)	54 (55.7)
10. Have you had "blackouts" or "flashbacks" as a result of their use?	1 (1.0)	96 (99.0)
11. Have you ever felt bad or guilty about your use of them?	12 (12.4)	85 (87.6)
12. Has your spouse (or have your parents) complained about your involvement with them?	7 (7.2)	90 (92.8)
13. Have you neglected your family because of your use of them?	3 (3.1)	94 (96.9)
14. Have you engaged in illegal activities in order to obtain them?	4 (4.1)	93 (95.9)
15. Have you experienced withdrawal symptoms as a result of heavy intake?	4 (4.1)	93 (95.9)
16. Have you had medical problems as a result of their use? (e.g., memory loss, hepatitis, convulsions, bleeding)	1 (1.0)	96 (99.0)
17. Have you neglected your responsibilities at work because of your use of them?	3 (3.1)	94 (96.9)

Based on YMRS scores, 2.6%, all females (Table 3.0.7), were experiencing symptoms of mania. While Hamilton did not specify cut-off points for his scale, it is generally agreed that scores lower than 7 indicate an absence of depression; scores between 7 to 17 represent mild depression; 18 to 24, moderate depression; and 25 or above, severe depression[313]. Taking 17 as the cut-off point, 14.4% were experiencing

moderate depressive symptoms at the time of participation. Most participants were experiencing mild depression (n = 44; 45.4%) and four (4.1%) were severely depressed.

Table 3.0.7: Young Mania Rating Scale (YMRS) and Hamilton Depression (Ham-D) Scale scores (n=97)

Rating scale	Total	Males	Females	n > cut-off
YMRS (≥ 20 indicates manic episode)	Median = 3 (25^{th} %ile = 1; 75^{th} %ile = 5) Range = 1 to 26	Median = 2 (25^{th} %ile = 1; 75^{th} %ile = 4) Range = 1 to 16	Median = 3 (25^{th} %ile = 1; 75^{th} %ile = 5) Range = 1 to 26	2 (Females = 2)
Ham-D (≥17 indicates depressed phase)	Mean = 9.9 (± 7.5) Range = 1 to 31	Mean = 7.2 (± 7.4) Range = 1 to 28	Mean = 10.8 (± 7.4) Range = 2 to 31	14 (Females = 9, Males =5)

Most participants were taking some type of psychiatric medication and about 25% combined this with herbal or natural remedies (Table 3.0.8). Other self-reported treatments included acupuncture, massage, reiki, biofeedback, and marijuana.

Table 3.0.8: Type of treatment used for mood disorders (n=97)

Treatment type	n (%)
Prescription medication	85 (87.6)
Herbal or natural remedies with prescription medications	24 (24.7)
Herbal or natural remedies without prescription medications	2 (2.1)
Other treatments (with or without prescription medications)	24 (24.7)

Eighty-five participants were taking a variety of psychotropic agents (Table 3.0.9), with the majority taking antidepressants (72.9%) and mood stabilizers (52.9%). Participants were also asked about adverse events. Only one person reported weight loss, while 39.2% reported an average weight gain of 30.6 lbs (± 24.8 lbs) within the first year

of treatment. Other commonly reported side effects included nausea (n = 24; 40.7%), dry mouth (n = 45; 76.3%), constipation (n = 24; 40.7%), and altered taste (n = 22; 37.3%).

Table 3.0.9: Psychotropic agents by medication group (n=85)*		
Group		**n (%)**
Anticonvulsants		
Toperimate, Lamotrogine, Gabapentin		16 (18.8)
Antipsychotics Typical		
Phenothiazines	Aliphatics (Chlorpromazine, Methotrimeprazine)	3 (3.5)
	Piperazines (Fluphenazine, Perphenazine, Thioproperazine, Trifluoperazine)	2 (2.4)
Dibenzoxazepine (Loxapine)		2 (2.4)
Antipsychotics Atypical/Novel		
Dibenzodiazepine, Benzisoxazole, Thienobenzodiazepine, Indolone, Quetiapine (Clozapine, Risperidone, Olanzapine, Molindone, Seroquel)		29 (34.1)
Antiparkinsonians		
Anticholinergics (Benztropine, Biperiden, Procyclidine, Trihexphenidyl, Orphenadrine)		3 (3.5)
Antihistaminergic, Dopamine agonist, β-Adrenergic or α-Adrenergic antagonists, Benzodiazepines		29 (34.1)
Mood Stabilizers (Antimanics)		
Carbamazepine, Lithium Carbonate, Divalproex Sodium and Valproic Acid		45 (52.9)
Antidepressants		
Tricyclics	Dibenzapine, Dibenzocyclogeptene, Dibenzoxazepine	1 (1.2)
Atypical	Dibenzoxazepine, Tetracyclic, Triazolopyridine, Monocyclic	19 (22.4)

*Numbers do not equal total as many individuals were taking more than one type of medication

Table 3.0.9: Psychotropic agents by medication group (n=85)* /continued		
Group		**n (%)**
Antidepressants continued..		
Selective Serotonin Reuptake Inhibitors (SSRI), Fluoxetine, Fluvoxamine, Sertraline and Paroxetine		25 (29.4)
Serotonin and Norepinephrine Reuptake Inhibitor (SNRI) and Venlafaxine		12 (14.1)
Monoamine Oxidase Inhibitors (MAOI). Includes phenelzine and tranylcypromine		1 (1.2)
Noradrenergic and Specific Serotonergic	Mirtazapine	4 (4.7)
Adjunctive Psychotropics		
Includes L-tryptophan and Clonazepam previously indicated in Antiparkinsonian group		1 (1.2)
Antianxiety/Sedative Hypnotics		
Benzodiazepines	Alprazolam, Bromazepam, Chlordiazepoxide, Clorazeptate, Diazepam, Flurazepam, Nitrazepam, Oxazepam, Temazepam and Triazolam. Lorazepam is included in Antiparkinsonian group	3 (3.5)
Non-sedating, anti-anxiety, miscellaneous	Buspirone, chloral hydrate, chlormezanone, hydroxyzine, meprobamate, promethazine, propranolol and zopiclone	9 (10.6)

*Numbers do not equal total as many individuals were taking more than one type of medication

There were more females than males (Table 3.0.10). About one-third of participants were married and/or had completed a university degree. About 20%, mainly females, were smokers. Many participants (45%) were moderately active, and living alone (n = 34, 35.1%) or with one other person (n = 30, 30.9%).

Table 3.0.10: Demographic characteristics of study participants (n=97)			
Characteristic	**Total n (% of total)**	**Males n (% of total)**	**Females n (% of total)**
Gender	97 (100)	28 (28.9)	69 (71.1)
Education level:			
Completed high school or less	21 (21.6)	16 (18.6)	5 (45.5)
Technical school/some university	46 (47.4)	40 (46.5)	6 (54.5)
University degree	30 (30.9)	30 (34.9)	0 (0.0)
Marital status:			
Married or common law	37 (38.1)	4 (10.8)	33 (89.2)
Divorced/separated/never married/widowed	27 (27.8)	1 (3.8)	26 (97.2)
Single	33 (34.0)	3 (9.1)	30 (90.9)
Smokers	20 (20.6)	2 (10.0)	18 (90.0)
Self-reported activity level:			
Active	22 (23)	7 (32)	15 (68%)
Moderately active	44 (45)	13 (30)	31 (70)
Inactive	25 (26)	9 (36)	16 (64)

3.4 Income Status and Food Security Issues

Since lower socio-economic status may influence nutrient intakes, income levels were recalculated to determine the proportions of the population who would be classified as low income or not. Of the 97 participants who answered the income question, 48.5% were classified as low income (Table 3.0.11). This proportion is considerably more than that in the BCNS (25.2%) and the BC census (19.6%).

Participants were asked three questions associated with food insecurity (Table 3.0.11). The result for the question on quality and variety of food was similar to that found for BC in the 2001/02 Canadian Community Health Survey (CCHS). The study sample had a significantly higher proportion of people reporting food insecurity based on all three screening questions ($p < 0.0001$). The CCHS targeted British Columbians aged

12 years and up, and respondents were instructed to answer often, sometimes, and never to the questions. Answering "often" or "sometimes" constituted a positive response. In the BCNS and this study, participants were instructed to answer simply "yes" or "no." When income levels were examined, slightly more than half of those who responded "yes" to the "worried" questions (66%) and the majority of those who said "yes" to the "quantity" question (73.8%) were low income.

Table 3.0.11: Food security indicators compared to the British Columbia Nutrition Survey (BCNS)

Characteristic	Study (%)	BCNS (%)	B.C. (%)
Low-income status	40.2*	25.2	19.6[1]
Worried that there would not be enough to eat because of lack of money	36.1**	7.3	11.6[2]
Did not have enough food to eat because of lack of money	19.6**	3.4	8.2[2]
In the past 12 months did you access the services of any food assistance programs (i.e., food bank, soup kitchen, or other charitable agency because there was not enough money for food)	19.6**	5.6	--

*Significant difference between study sample and BCNS at $p < 0.05$
**Significant difference between study sample and BCNS at $p < 0.0001$
[1]Statstics Canada, Census
[2]BC statistics calculated from positive responses in the Canadian Community Heath Survey, 2002

3.5 Body Mass Indices

Body mass index (BMI; kg/m^2) was calculated by dividing body weight in kilograms by height in metres squared and was categorized using the Health Canada[314] weight classifications. These standards indicate that a BMI < 18.5 is "underweight", 18.50–24.99 is a "normal weight", 25.0–29.99 is "overweight," and ≥ 30 is "obese."

The distribution of BMI was highly skewed (skewness = 2.876 ± 0.078 SE), so the $\log_{10}$ transformation was used in an effort to improve its symmetry. However, the distribution remained significantly skewed (skewness = 0.976 ± 0.088 SE). Statistical

analyses were conducted on the transformed data, although untransformed data are displayed for clarity. Nonparametric analyses were also conducted.

About 32% of participants were overweight and almost 35% were classified as obese (Table 3.0.12). The BCNS had more overweight participants ($p < 0.05$) whereas the study sample revealed a significantly higher proportion ($p < 0.05$) that was obese. Five percent were extremely obese.

The distributions of BMI category within the sample were also examined by a variety of demographic and psychiatric variables. There were no significant differences in BMI according to the following subgroups: gender, age (19–30, 31–50, 51–70 years), income (low vs not low income) subdivided by gender, education (secondary school or less, some university or technical school, and completion of university degree), whether taking psychiatric medication or not or by type of diagnosis (major depressive vs. bipolar disorder). The category of participants taking at least one type of medication tended to have a higher proportion of obese or extremely obese; participants with bipolar disorder tended to include a higher proportion of people that were overweight; and those with depression tended to include a higher proportion of people who were obese.

Compared with the BCNS sample, this study had significantly more participants in the obese category (n = 18; 34.6%) who were not low income (n = 181; 20.1%); significantly fewer men with some university or technical school (n = 13; 32.5%) who were overweight (n = 152; 48.9%); and significantly fewer men with a university degree (n = 7; 23.3%) who were overweight (n = 30; 44.3%). However, there were more females with some university or technical school (n = 3; 50.0%) who were obese than the BCNS (n = 106; 31.5%). Correlations of BMI and psychiatric symptom measures were also analyzed. Although the correlations between the Ham-D and GAF and BMI were positive, and those between the YMRS and BMI were negative, none were significant.

Mean BMI by age group for men, women, and both sexes combined showed that the study sample tended to have higher BMIs (Table 3.0.13). Statistical analysis revealed significant differences in females between the ages of 31 and 50 years ($p < 0.001$), males and females combined aged 31 to 50 ($p < 0.50$), and males and females combined for all age groups ($p < 0.001$). There were no significant differences between the gender groups.

Table 3.0.12: Percent distribution of Body Mass Index (BMI)[1] by age and gender between the study sample and British Columbia Nutrition Survey (BCNS)

BMI	Study Sample [n (%)]			BCNS [n (%)]		
	Total (n=91)	Males (n=17)	Females (n=62)	Total (n=1320)	Males (n=596)	Females (n=724)
Underweight (BMI < 18.5)	1 (1.1)	0 (0)	1 (1.1)	2 (1.3)	5 (0.8)	9 (1.3)
						316 (43.7)
Overweight (BMI 25 to 29.9)	29 (31.9)*	7 (7.7)	21 (23.1)	487 (36.9)	265 (44.5)	267 (36.9)
Obesity (BMI 30 to 39.9)	27 (29.7)*	7 (7.7)*	19 (20.9)*	240 (18.2)	114 (19.2)	132 (18.2)
Extreme obesity (BMI ≥ 40)	5 (5.5)	2 (2.2)	3 (3.3)	0 (0)	0 (0)	0 (0)

[1]92.3% of the sample participants were measured for height and weight. BMI is calculated as weight (kg)/height (m^2)
The shaded area represents the healthy weight range
*Significant difference between study sample and BCNS at $p < 0.05$

Table 3.0.13: Mean BMI by age group for men, women, and both sexes combined and compared to British Columbia Nutrition Survey (BCNS)

Age Group	Study Sample (Mean ± SD[1])			BCNS (Mean ± SD[1])		
	All	Men	Women	All	Men	Women
19-30	28.8 ± 9.9	28.8 ± 5.6	29.4 ± 10.9	25.0 ± 5.6	25.2 ± 4.1	24.9 ± 7.1
31-50	29.3 ± 6.0*	30.0 ± 6.6	29.0 ± 5.8**	26.6 ± 5.8	27.7 ± 5.6	25.5 ± 5.9
51-70	27.2 ± 5.1	26.8 ± 4.2	27.3 ± 5.4	26.7 ± 5.7	26.8 ± 4.6	26.7 ± 6.6
All	28.5 ± 6.3**	28.7 ± 5.7	28.5 ± 6.5	26.2 ± 5.7	26.7 ± 5.0	25.6 ± 6.3

[1]Standard Deviation
*Significant difference compared to BCNS at $p < 0.05$
**Significant difference compared to BCNS at $p < 0.001$

3.6 Dietary Patterns

The interviewer asked participants about the sources of the foods they ate. The definitions for each code/source are outlined in the following table.

Table 3.0.14: Definitions of codes for sources of foods outside the home

Code	Description
Restaurant	A place where table service is provided or food eaten on an airplane.
Cafeteria	A place where tray service is provided, no table service offered.
Fast food	A restaurant where, in most cases, no table service is provided; it has a fixed menu with a limited selection of foods.
Take-out/deli	A place where meals or main dish food items can be bought or ordered but have to be consumed elsewhere.
Vending machine/snack bar	Small food items or snack foods obtained from tuck shops, vending machines, stands, chip wagons, mobile canteens, convenience stores, or grocery store sample displays, or food bought at the movies.

Baked goods bought at a bakery (e.g., breads, bagels) and eaten at home, foods obtained in a private home (respondent's, friend's or relative's home) but eaten elsewhere, foods consumed at a community hall function where food is prepared by "locals," and coffee consumed at the office coffee station were excluded. The proportion of males eating foods from restaurants ($p < 0.05$) and vending machines or snack bars ($p < 0.0001$) was significantly higher than females. When examining specific age groups, significantly more males aged 19 to 34 years ($p < 0.05$) and 35 to 49 years ($p < 0.0001$) ate foods from vending machines or snack bars when compared to females.

Table 3.0.15: Frequency of food intake from sources outside the home based on three days' intake

	Total [n (%)]		**Males [n (%)]**			**Females [n (%)]**		
Food Source[1]	**Males (n = 504)**	**Females (n = 1242)**	**18-34 yrs (n = 90)**	**35-49 yrs (n = 252)**	**50+ yrs (n = 162)**	**18-34 yrs (n = 288)**	**35-49 yrs (n = 468)**	**50+ yrs (n = 486)**
Restau-rant	58 (11.5)*	103 (8.3)	13 (14.4)	31 (12.3)	14 (8.6)	34 (11.8)	45 (9.6)	24 (4.9)
Cafeteria	12 (2.4)	27 (2.2)	3 (3.3)	6 (2.4)	3 (1.9)	6 (2.1)	15 (3.2)	6 (1.2)
Fast food	55 (10.9)	137 (11.0)	8 (8.9)	31 (12.3)	16 (9.9)	32 (11.1)	60 (12.8)	45 (9.3)
Take-out / deli	49 (9.7)	111 (8.9)	6 (6.7)	28 (11.1)	15 (9.3)	12 (4.2)	57 (12.2)	42 (8.6)
Vending machines / snack bar	76 (15.1)**	98 (7.9)	16 (17.8)*	38 (15.1)**	32 (19.8)	25 (8.7)	52 (11.1)	21 (4.3)

[1]See Table 3.0.14 for definitions of each
*Significant differences between males and females at $p < 0.05$
**Significant differences between males and females at $p < 0.0001$

Respondents were also asked whether the food item they ate was considered as part of a meal (breakfast, brunch, lunch, dinner, or other meal) or as a snack (defined as any foods eaten in between meals). The proportion of males eating lunch ($p < 0.0001$), dinner ($p < 0.05$), or snacks ($p < 0.05$) was significantly higher than females. Males aged 18 to 34 years ate significantly more dinners ($p < 0.05$), males aged 35 to 49 years ate significantly more snacks ($p < 0.05$), and males over 50 years ate significantly more lunches ($p < 0.05$) when compared to females within the same age groups.

Table 3.0.16: Frequency of food intake from sources outside the home based on three days' intake

Eating event[1]	**Gender**	**Totals**[3]	**Age group**[2] **[n (%)]**		
			18-34 yrs	**35-49 yrs**	**50+ yrs**
Breakfast (n = 291 total meals)	Males (n = 84)	60 (71.4)	**n = 15**	**n = 42**	**n = 27**
			15 (100.0)	30 (71.4)	15 (53.6)
	Females (n = 207)	141 (68.1)	**n = 48**	**n = 78**	**n = 81**
			21 (43.8)	66 (84.6)	54 (66.7)
Lunch (n = 291 total meals)	Males (n = 84)	81 (96.4)***	**n = 15**	**n = 42**	**n = 27**
			15 (100.0)	42 (100.0)	24 (88.9)*
	Females (n = 207)	151 (72.9)	**n = 48**	**n = 78**	**n = 81**
			21 (43.8)	76 (97.4)	54 (66.7)
Dinner (n = 291 total meals)	Males (n = 84)	79 (94.0)*	**n = 15**	**n = 42**	**n = 27**
			13 (86.7) *	39 (92.9)	27 (100.0)
	Females (n = 207)	167 (80.7)	**n = 48**	**n = 78**	**n = 81**
			27 (56.3)	71 (91.0)	69 (85.2)
Brunch (n = 291 total meals)	Males (n = 84)	4 (4.7)	**n = 15**	**n = 42**	**n = 27**
			0 (0.0)	3 (7.1)	1 (0.0)
	Females (n = 207)	12 (5.8)	**n = 48**	**n = 78**	**n = 81**
			2 (4.2)	5 (6.4)	5 (6.2)
			67 (46.5)	171 (73.1)	169 (69.5)

[1]Based on the respondent's perception of the eating event
[2]Percents of each age group based on total for that gender as the denominator
[3]Based on total possible meals or snacks possible over three days. Meals totals are based on one meal type per day; snacks are based on three snacks per day
*Significant differences between males and females at $p < 0.05$ for the total sample or within the age group specified

Table 3.0.16: Frequency of food intake from sources outside the home based on three days' intake /continued

Eating event[1]	Gender	Totals[3]	Age group[2] [n (%)]		
			18-34 yrs	**35-49 yrs**	**50+ yrs**
			2 (4.2)	5 (6.4)	5 (6.2)
Snacks (n = 873 total snacks)	Males (n = 252)	186 (73.8)*	**n = 45**	**n = 126**	**n = 81**
			24 (53.3)	105 (83.3)*	57 (70.4)
	Females (n = 621)	407 (65.5)	**n = 144**	**n = 234**	**n = 243**
			67 (46.5)	171 (73.1)	169 (69.5)

[1]Based on the respondent's perception of the eating event
[2]Percents of each age group based on total for that gender as the denominator
[3]Based on total possible meals or snacks possible over three days. Meals totals are based on one meal type per day; snacks are based on three snacks per day
*Significant differences between males and females at $p < 0.05$ for the total sample or within the age group specified

A final question was whether participants prepared most of the foods they ate that day. Out of the 291 (3 days*97 respondents) total possible days of intake, 224 (77.0%) days were indicated as having included foods prepared by the respondent. Males reported significantly fewer food preparation days (n = 25/84; 29.8%) than females (199/207; 96.1%) ($p < 0.0001$).

3.7 Supplement Use

Participants were asked if they had consumed supplements the previous day and how often during the past month. Supplements were classified as nutritional (containing recognized vitamins and minerals) and non-nutritional (not containing recognized vitamins and minerals). The total proportion of study participants taking supplements in most groups was typically higher than the BCNS sample (Table 3.0.17). In particular, over two-thirds of study participants were taking vitamin B complex (64%), folic acid (42%), vitamin C (74%), vitamin D (60%), multivitamins (90%), minerals (84%), other nutrients (84%) — including glucosamine, amino acids, evening primrose oil, coenzyme Q10, flax seed oil, or lactic acid bacteria — and herbs and natural products (61%).

Using Fisher Exact tests to compare the study and BCNS sample, males 31 to 50 years were found to have significantly higher intakes of vitamin B complex ($p < 0.05$),

vitamin D ($p < 0.0001$), minerals ($p < 0.05$), and homeopathic preparations ($p < 0.05$). Males aged 51 to 70 years had significantly higher intakes of vitamin D ($p < 0.05$). For females, some significant differences were also found between the study and the BCNS sample. Those females aged 31 to 50 years had higher intakes of iron ($p < 0.05$), replacement preparations ($p < 0.05$), gastrointestinal products ($p < 0.05$), vitamin A ($p < 0.05$), vitamin A and D combinations ($p < 0.05$), vitamin B complex ($p < 0.0001$), vitamin C ($p < 0.0001$), vitamin E ($p < 0.001$), and multivitamins ($p < 0.0001$). There were also a few significant differences among females aged 51 to 70 years; they had higher intakes of enzymes ($p < 0.0001$), gastrointestinal products ($p < 0.001$), vitamin B complex ($p < 0.0001$), vitamin D ($p < 0.0001$), and vitamin E ($p < 0.05$). An absence of numbers in one or more cells needed to calculate the Fisher Exact statistic meant that not all data could be compared.

Overall, those in the study had higher intakes of iron preparations ($p < 0.0001$), enzymes ($p < 0.0001$), gastrointestinal products ($p < 0.0001$), vitamin A ($p < 0.0001$), vitamin A and D combinations ($p < 0.05$), vitamin B complex ($p < 0.0001$), vitamin C ($p < 0.0001$), vitamin D ($p < 0.0001$), vitamin E ($p < 0.0001$), multivitamins ($p < 0.0001$), minerals ($p < 0.0001$), other nutrients ($p < 0.0001$), herbs and natural products ($p < 0.0001$), and homeopathic preparations ($p < 0.0001$).

3.8 Chapter Summary

This chapter compared respondents and non-respondents of the study as well as detailed demographic information, psychiatric features, smoking status, anthropometric measurements, dietary patterns, and supplement use data.

The overall response rate was high and non-respondents did not differ significantly from participants. Examination of psychiatric and general characteristics indicated that most participants met the criteria for depression and more females than males exhibited depressive symptoms. About one-third of the participants were married and had completed a university degree. About 20% were smokers, and of these the majority were females. A high proportion of participants were low income and overweight or obese.

Table 3.0.17: Proportion of participants taking supplements in previous month by supplement groups, age, and gender and compared to the British Columbia Nutrition Survey (BCNS)

		Males						Females						
Supplement group	**Sample**	**Total n**	**19-30 % of total n**	**Total n**	**31-50 % of total n**	**Total n**	**51-70 % of total n**	**Total n**	**19-30 % of total n**	**Total n**	**31-50 % of total n**	**Total n**	**51-70 % of total n**	**Total (%)**
Iron preparations	Study	5	0	16	6	7	0	9	9	39	5*	21	0	21***
	BCNS	142	0	205	0	249	<1	176	2	266	3	282	<1	1
Replacement preparations[1]	Study	5	0	16	6	7	0	9	0	39	3*	21	0	9
	BCNS	142	0	205	1	249	1	176	<1	266	1	282	5	2
Enzymes	Study	5	0	16	6	7	0	9	0	39	6	21	5***	18***
	BCNS	142	0	205	0	249	1	176	<1	266	0	282	<1	<1
Gastrointestinal products[2]	Study	5	0	16	6	7	14	9	0	39	5*	21	5**	24***
	BCNS	142	2	205	2	249	3	176	4	266	8	282	8	5
Vitamin A	Study	5	0	16	6	7	14	9	0	39	3*	21	0	17***
	BCNS	142	2	205	2	249	4	176	1	266	<1	282	3	2
Vitamins A and D	Study	5	0	16	6	7	0	9	0	39	3*	21	0	9*
	BCNS	142	0	205	1	249	2	176	2	266	3	282	7	3

[1]Contains mostly calcium and potassium
[2]Includes antacids, adsorbents, laxatives, digestants
*Significant differences found at $p < 0.05$ between the study sample and BCNS
**Significant differences found at $p < 0.001$ between the study sample and BCNS
***Significant differences found at $p < 0.0001$ between the study sample and BCNS

Table 3.0.17: Proportion of participants taking supplements in previous month by supplement groups, age, and gender and compared to the British Columbia Nutrition Survey (BCNS) /continued

		Males						Females						
Supplement group	**Sample**	**Total n**	**19-30 % of total n**	**Total n**	**31-50 % of total n**	**Total n**	**51-70 % of total n**	**Total n**	**19-30 % of total n**	**Total n**	**31-50 % of total n**	**Total n**	**51-70 % of total n**	**Total (%)**
Vitamin B complex	Study	5	20*	16	16*	7	7	9	9	39	15***	21	10***	64***
	BCNS	142	2	205	5	249	8	176	9	266	12	282	16	9
Folic acid	Study	5	0	16	6	7	14	9	11	39	10	21	0	42
	BCNS	142	0	205	0	249	0	176	0	266	0	282	0	0
Niacin	Study	5	0	16	6	7	14	9	0	39	0	21	0	21
	BCNS	142	0	205	0	249	0	176	0	266	0	282	0	0
Pantothenic acid	Study	5	0	16	0	7	14	9	0	39	0	21	0	14
	BCNS	142	0	205	0	249	0	176	0	266	0	282	0	0
Vitamin B_6	Study	5	0	16	6	7	14	9	0	39	0	21	10	31
	BCNS	142	0	205	0	249	0	176	0	266	0	282	0	0
Vitamin C	Study	5	20	16	12***	7	14	9	9	39	18***	21	21	74***
	BCNS	142	19	205	22	249	16	176	25	266	28	282	30	24
Vitamin D	Study	5	0	16	16***	7	14*	9	9	39	8	21	19***	60***
	BCNS	142	0	205	<1	249	2	176	0	266	0	282	3	1

*Significant differences found at $p < 0.05$ between the study sample and BCNS
**Significant differences found at $p < 0.001$ between the study sample and BCNS
***Significant differences found at $p < 0.0001$ between the study sample and BCNS

Table 3.0.17: Proportion of participants taking supplements in previous month by supplement groups, age, and gender and compared to the British Columbia Nutrition Survey BCNS /continued

		Males						Females						
Supplement group	**Sample**	**Total n**	**19-30 % of total n**	**Total n**	**31-50 % of total n**	**Total n**	**51-70 % of total n**	**Total n**	**19-30 % of total n**	**Total n**	**31-50 % of total n**	**Total n**	**51-70 % of total n**	**Total (%)**
Vitamin E	Study	5	0	17	12	7	14	9	9	39	10**	21	14*	54***
	BCNS	142	4	205	9	249	22	176	30	266	42	282	37	17
Multivitamins	Study	5	5	16	12	7	7	9	9	39	33***	21	21	90***
	BCNS	142	7	205	11	249	4	176	12	266	8	282	14	9
Minerals	Study	5	5	16	16*	7	7	9	9	39	23	21	21	84***
	BCNS	142	4	205	6	249	12	176	8	266	18	282	28	13
Other nutrients[3]	Study	5	0	16	16	7	7	9	9	39	28	21	21	84***
	BCNS	142	11	205	17	249	18	176	8	266	27	282	33	20
Herbs and natural products	Study	5	0	16	16	7	7	9	0	39	15	21	21	61***
	BCNS	142	10	205	11	249	19	176	21	266	25	282	25	19
Homeopathic preparations	Study	5	0	16	6*	7	7	9	0	39	8	21	10	32***
	BCNS	142	<1	205	<1	249	2	176	5	266	3	282	4	3

[3]Includes glucosamine, amino acids, evening primrose oil, coenzyme Q10, flax seed oil, lactic acid bacteria
*Significant differences found at $p < 0.05$ between the study sample and BCNS
**Significant differences found at $p < 0.001$ between the study sample and BCNS
***Significant differences found at $p < 0.0001$ between the study sample and BCNS

Study participants included a significantly higher proportion of people who were obese when compared to the BCNS, and income and education level were both associated with BMI. Income seemed to affect males more as the proportion of males in the underweight category who were not low income and the obese category that were low income was significantly higher than in the BCNS. The proportion of females in the obese category who were not low income was significantly higher than in the BCNS. The highest prevalence of overweight and obesity was in the group who had attained some university or technical school education.

Dietary patterns indicated that males, particularly those between the ages of 19 and 49 years, tended to eat out more often. The proportion of males eating lunch, dinner, or snacks was significantly higher than in the BCNS.

Of particular interest was the proportion of study participants taking supplements. Participants had higher proportions of intake of each supplement compared to those in the BCNS. Among those aged 31 and 50 years, and to a lesser extent among those aged 51 to 70 years, several significant differences emerged. The number of participants taking single vitamin and mineral preparations is concerning because of the increased risk of exceeding the Tolerable Upper Intake Levels and because other evidence also suggests that supplemental use of some nutrients can contribute to all-cause mortality[315].

In Chapter Four, food and nutrient intake data will be examined in more detail, including a review of intake from food sources and the contribution that selected nutrient supplements make to the overall nutrient intake of study participants.

CHAPTER FOUR: RESULTS – FOOD AND NUTRIENT INTAKES

This chapter documents results of the sample's food and nutrient intakes and addresses the following research inquiries outlined in Chapter Two: 1) Compared to the *Dietary Reference Intakes (DRIs)*, is there evidence of inadequate (i.e., less than the Estimated Average Requirement or Adequate Macronutrient Distribution Ranges) and/or excess (i.e., greater than the Tolerable Upper Intake Levels or Adequate Macronutrient Distribution Ranges) nutrient intakes in individuals with mood disorders?; 2) Do nutrient intakes of individuals with mood disorders differ from those in a general population sample?; and 3) Do blood levels of selected nutrients in individuals with mood disorders occur outside the reference ranges?

Energy and nutrient intakes are examined according to the gender and age groups of the *DRIs* (19–30, 31–50, and 51–70 years), with 70 being the maximum age for participants. Nutrient intake data was also compared with the British Columbia Nutrition Survey (BCNS), and data on selected biochemical indicators of nutrient status was reported on a subsample of participants.

4.1 Food Group Use

Food group use analyses complement detailed nutrient analyses as they allow one to recognize why specific nutrients may be lacking and to develop dietary strategies to address nutrient inadequacies and excesses. The food group results included the sample's usual intakes, some general diet characteristics, the proportion of the sample meeting the minimum recommendations as suggested by *Eating Well with Canada's Food Guide*[316], and comparison to the BCNS where feasible. Since the data are presented as either usual intakes (study sample) or one-day intakes (BCNS), it is important to note that one-day nutrient intakes tend to be less representative of usual food intakes. Food group use was reported according to gender and the following age groups: 18–34, 35–49, 50–64, and 65–74 years[1]. The data as outlined in Table 4.0.1 are based on the minimum recommendations of the 2007 *Eating Well with Canada's Food Guide*[316].

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[1]Unlike the results on energy and nutrient intakes in Chapter Five, the age groups do not correspond to the *Dietary Reference Intakes* (DRI) age categories.

Table 4.0.1: Proportion of participants consuming selected food groups relative to *Eating Well with Canada's Food Guide* by age and gender and compared to the British Columbia Nutrition Survey (BCNS)

Grains	**Total**		**< 5 servings/day (%)**		**Recommended 5-12 servings/day (%)**		**> 12 servings/day (%)**	
	Study	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**
All participants	97	1823	58.8**	40.5	37.1**	56.7	5.2	2.3
All females	69	955	63.8	61.3	34.8	38.9	1.4	0
F 18-34	16	228	62.5	53.2	37.5	46.8	0	0
F 35-49	26	193	76.9	59.4	19.2*	40.6	3.8	0
F 50+	27	534	51.9	69.3	48.2	31.4	0	0
All males	28	868	46.4**	19.3	42.9**	75.0	10.7	5.6
M 18-34	5	187	60.0*	13.9	40.0	76.1	0	10.1
M 35-49	14	150	42.9*	15.9	42.9**	79.6	14.3	4.5
M 50+	9	531	44.4	29.1	44.4	66.5	11.1	3.1

*Significant differences between study and BCNS samples at $p < 0.05$
**Significant differences between study and BCNS samples at $p < 0.001$
***Significant differences between study and BCNS samples at $p < 0.0001$

Table 4.0.1: Proportion of participants consuming selected food groups relative to *Eating Well with Canada's Food Guide* by age and gender and compared to the British Columbia Nutrition Survey (BCNS) /continued

Vegetables and fruit	**Total**		**< 5 servings/day (%)**		**Recommended 5-10 servings/day (%)**		**> 10 servings/day (%)**	
	Study	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**
All participants	97	1823	75.3*	64.6	22.7*	32.3	2.1	3.1
All females	69	955	72.5	72.7	24.6	25.3	2.9	2.0
F 18-34	16	228	68.8	83.7	31.3	16.3	0	0
F 35-49	26	193	69.2	77.8	23.1	19	7.8	3.2
F 50+	27	534	77.8	59.9	22.2	36.7	0	2.2
All males	28	868	82.1*	56.3	17.8*	39.4	0	4.3
M 18-34	5	187	80.0	62.3	20.0	33.9	0	3.8
M 35-49	14	150	85.7*	54.3	14.2	40.6	0	5.1
M 50+	9	531	77.8	49.8	22.2	44.4	0	3.9

*Significant differences between study and BCNS samples at $p < 0.05$
**Significant differences between study and BCNS samples at $p < 0.001$
***Significant differences between study and BCNS samples at $p < 0.0001$

Table 4.0.1: Proportion of participants consuming selected food groups relative to *Eating Well with Canada's Food Guide* by age and gender and compared to the British Columbia Nutrition Survey (BCNS) /continued

Meat and alternatives	**Total**		**Less Than 100 grams/day (%)**		**Recommended 100-300 grams/day (%)**		**More Than 300 grams/day (%)**	
	Study	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**
All participants	97	1823	40.2*	26.4	37.1***	65.8	21.6***	7.8
All females	69	955	36.2	39.8	40.6*	58.2	7.2***	2.0
F 18-34	16	228	43.8	48.7	31.3	50.6	25.0***	0.7
F 35-49	26	193	26.9	33	46.2	62.9	26.9***	4.1
F 50+	27	534	40.7	35.7	40.7	53.8	18.5***	0.8
All males	28	868	50.0***	12.7	28.6***	73.5	57.1	13.7
M 18-34	5	187	60.0**	10.5	20.0*	67.9	20.0	21.6
M 35-49	14	150	46.2*	15.2	23.1**	70.9	30.8	13.9
M 50+	9	531	55.6**	12.6	44.4*	82.6	0	4.7

*Significant differences between study and BCNS samples at $p < 0.05$
**Significant differences between study and BCNS samples at $p < 0.001$
***Significant differences between study and BCNS samples at $p < 0.0001$

Table 4.0.1: Proportion of participants consuming selected food groups relative to *Eating Well with Canada's Food Guide* by age and gender and compared to the British Columbia Nutrition Survey (BCNS) /continued

Milk and milk alternatives	**Total Sample (n)**		**Less Than 2 servings/day (%)**		**Recommended 2-4 servings/day (%)**		**More Than 4 servings/day (%)**	
	Study	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**	**Study**	**BCNS**
All participants	97	1823	50.5***	77.4	39.2***	19.9	10.3***	2.7
All females	69	955	53.6***	84.6	34.8***	14.7	11.6***	0.7
F 18-34	16	228	31.3***	80.5	37.5	18.7	31.3***	0.8
F 35-49	26	193	57.7**	86.8	34.6*	12.9	7.7*	0.2
F 50+	27	534	63.0*	85.4	33.3*	13.8	3.7	2.1
All males	28	868	42.9*	69.9	50.0*	25.3	7.1	4.8
M 18-34	5	187	60.0	61.4	40.0	29.8	0	8.8
M 35-49	14	150	30.8*	68.7	53.8*	25.7	15.4	5.5
M 50+	9	531	55.6	80.3	44.4*	15.6	0	0.7

*Significant differences between study and BCNS samples at $p < 0.05$
**Significant differences between study and BCNS samples at $p < 0.001$
***Significant differences between study and BCNS samples at $p < 0.0001$

About 59% did not meet the minimum recommendations of 5 grain servings a day; significantly lower ($p < 0.001$) than the BCNS (about 41%). Fewer participants met the suggested amount compared to the BCNS for females aged 35 to 49 years ($p < 0.05$) and all males ($p < 0.001$). A higher proportion of males 18 to 49 years had grain intakes below the recommended levels compared to the BCNS ($p < 0.05$). In general, about 15% of the sample would need to consume two extra grain servings daily to meet the minimum recommendations and about 30% would need to add one more serving.

About three-quarters of the participants did not consume the minimum 5 daily servings recommended for vegetables and fruit; significantly higher than the BCNS ($p < 0.05$). The proportion of males 35 to 49 years that did not meet the minimum levels was significantly higher than the BCNS ($p < 0.05$). About 45%-50% of the sample would need to add two to three more servings of vegetables and fruit to their daily intake to meet the minimum recommended servings.

For all subgroups, more participants consumed the suggested amounts of milk and milk alternatives compared to those in the BCNS (Table 4.0.3). All female groups plus men 35 to 49 years had intakes that exceeded the suggested four servings/day. Significant differences were found for those 35–49 and > 50 years ($p < 0.05$). Interestingly, comparisons of females 18–34 years revealed more participants exceeding the suggested amount compared to the BCNS ($p < 0.0001$). To meet the minimum recommended two servings per day, about half of the sample would need to add one serving per day.

The proportion of participants who consumed the daily recommended two servings of meat and alternatives was less than in the BCNS. Male groups were significantly lower.

4.2 Meeting the Guidance Statements of *Eating Well with Canada's Food Guide*

The food intake data was also analyzed to examine compliance with the guidance statements in *Eating Well with Canada's Food Guide*[316].

Choose Lower-Fat Foods More Often

Five guidance statements focus on choosing lower-fat foods. They include choosing vegetables and fruit as well as grain products that are lower in fat, sugar, or salt, drinking skim, 1%, or 2% milk each day, as well as selecting lower-fat milk alternatives and lean meat and alternatives prepared with little or no added salt or fat. Participants reported consuming more high-fat grain products, fish/shellfish, and processed meats ($p < 0.0001$).

Intakes of higher-fat foods were lower than in the BCNS for milk and fortified plant beverages, milk products, meat, poultry, processed meats, and "other" foods ($p < 0.0001$).

Make at least half of your grain products whole grain each day

Non-whole grain products were the most (88.8%) prominent choices and whole grain intake was lower (15.6%; $p < 0.001$) than in the BCNS.

Eat at least one dark green and one orange vegetable each day

Some fruits, including mangoes, papayas, and peaches, are also included in this guidance statement. Study participants had lower intakes of dark green and deep yellow/orange vegetables ($p < 0.05$) and higher intakes of white potatoes ($p < 0.001$).

Have meat alternatives such as beans, lentils, and tofu often

Participants ate fewer high-fat legumes ($p < 0.001$) and nuts and seeds ($p < 0.0001$), and had higher intakes of low-fat legumes ($p < 0.0001$) and eggs ($p < 0.05$).

4.3 Meeting Nutrient Intakes

Based on a review of *Eating Well with Canada's Food Guide* by Health Canada, for diets that met the minimum of each food group**,** the observed proportion of diet below the nutrient specific EAR was less than 10% for most groups. There were a higher proportion of females 19 to 34 years in the study who did not meet all ($p < 0.0001$) of the recommended food group servings compared to the BCNS. Males 19 to 49 years also had a higher proportion that only met 1 of the food group serving minimums when compared to the BCNS ($p < 0.0001$). Fewer females ($p < 0.001$) and more males ($p < 0.0001$) in the study over 50 years only met two of the four food group servings when compared to the BCNS. Significantly less females 19 to 34 years ($p < 0.05$) and males 35 to 49 years ($p < 0.001$) met three of the four food group servings when compared to the BCNS (Table 4.0.2). For women, only 0%–6.3% met the minimum suggested servings for all groups on a given day; men did slightly better (0%–15.4%). A diet that does not meet requirements for all food groups on a given day is not necessarily nutritionally inadequate. Diets that consistently lack a food group, though, would be an issue.

4.4 Relationship of Food Group Use and Nutrient Intakes

The food group intake data gathered from the three-day food records and food frequency questionnaire correlated well; fruits and vegetables were 0.72 ($p < 0.001$), grains 0.67 ($p < 0.001$), meat and alternatives 0.63 ($p < 0.05$) and milk and milk alter-natives

0.65 ($p < 0.05$). These figures correspond with cited correlations ranging from 0.6 to 0.7 suggesting that the reported food intake was quite representative of usual consumption.

Table 4.0.2: Proportion of the British Columbia Nutrition Survey (BCNS) and study sample who met *Eating Well with Canada's Food Guide* guidelines for either 0, 1, 2, 3, or 4 food groups according to the reported food intakes

Age and gender	Sample	Proportion that met the specified number of food groups (%)				
		0	**1**	**2**	**3**	**4**
Females						
19-34	BCNS	10.8	38.8	32.1	15.1	3.2
	Study	25.0***	31.3	31.3	6.3*	6.3
35-49	BCNS	13.2	29.0	42.3	15.0	0.7
	Study	7.7	19.2	57.1	11.5	0.0
50+	BCNS	14.6	30.5	42.4	14.9	2.0
	Study	21.4	42.9	25.0**	7.1	3.6
Males						
19-34	BCNS	2.2	22.2	31.4	30.1	14.2
	Study	0.0	60.0***	0.0	40.0	0.0
35-49	BCNS	2.7	13.7	39.5	32.2	11.9
	Study	7.7	30.8***	30.8	15.4**	15.4
50+	BCNS	2.5	24.5	35.2	30.3	7.8
	Study	0.0	25.0	62.5***	0.0	12.5

*Significant differences between study and BCNS samples at $p < 0.05$
**Significant differences between study and BCNS samples at $p < 0.001$
***Significant differences between study and BCNS samples at $p < 0.0001$

Food group data helped explain nutrient intakes reported in the following section. Table 4.0.3 lists nutrients provided by foods within food groups, provides a useful overview of how food and nutrient intake data are linked.

Table 4.0.3: Food sources of selected nutrients					
Nutrient	**Meat and alternatives**	**Milk and milk alternatives**	**Vegetables and fruit**	**Grain products**	**Other foods**
Vitamin B_6 (pyridoxine)	Meats, eggs, fish, legumes	--	Green leafy vegetables, bananas, carrots, potatoes	Whole grains	Nutritional yeast, wheat germ
Vitamin B_{12}	Meats, eggs, fish, shellfish	Cheese, yoghurt	--	--	Margarine, meat fats and broths, cream cheese, malted milk (unprepared)
Vitamin C	--	--	Fruits (especially citrus), strawberries, cabbage, tomatoes, potatoes	--	--
Folate/folic acid	Organ meats, nuts, legumes	--	Green leafy vegetables, asparagus, bananas, strawberries	Whole grains, forti-fied flour-based products	Nutritional yeast
Calcium	Oysters, scallops, salmon and sardines with bones, tofu	All	Green leafy vegetables, broccoli, dates, fortified orange juice	--	Blackstrap molasses
Iron	Meats, eggs, shell-fish, nuts, sardines, legumes	--	Broccoli, peas, spinach, prunes, raisins	Bread, enriched cereals and breads	Blackstrap molasses, wheat germ
Magnesium	Nuts, legumes	--	Peas	Whole grains	--
Zinc	Meat, liver, eggs, shellfish	Cheese	Green leafy vegetables, oranges, prunes, strawberries	Whole grains	Chocolate syrup

4.5 Nutrient Intakes

The Canadian Nutrient File (CNF) was used to determine the nutrient content of foods consumed. This database is at least 90% complete for all nutrients presented in this chapter unless otherwise indicated. The tables and figures include the adjusted mean and distributions of nutrient intakes from food sources only and from food and supplements combined where applicable. Intakes were assessed against the Estimated Average Requirement (EAR), Adequate Intake (AI), and Tolerable Upper Intake Level (UL), when possible, to determine inadequacies or excesses. For nutrients with an EAR, the percentage below the EAR reflects the prevalence of inadequacy. For nutrients with an AI, the prevalence of nutrient inadequacy cannot be quantitatively determined. However, when median usual intake of a group meets or exceeds the AI, the expected prevalence of inadequacy is low. The *Dietary Reference Intakes (DRI)* criteria are listed in Appendix A. Data presented on energy, macronutrients, and selected micronutrients were generally presented in the same format for ease of use:

1. The medians for a given nutrient are presented as bar graphs or in tables and compared with the BCNS data. Median values are presented to allow for direct comparison to the EAR, the median population value for a given nutrient.
2. The distribution of the data for the 6 age and gender groupings of the sample were presented as dot plots (Appendix C). These figures provide a crude estimate of the density and each symbol (dot) represents a single observation. Results of a one-way ANOVA (or non-parametric Kruskal-Wallis test) testing the null hypothesis that the underlying mean nutrient levels are the same for the 6 groups were also presented. Where applicable, Bonferroni's post-hoc test is used to detect where the specific significant differences exist among the groups.

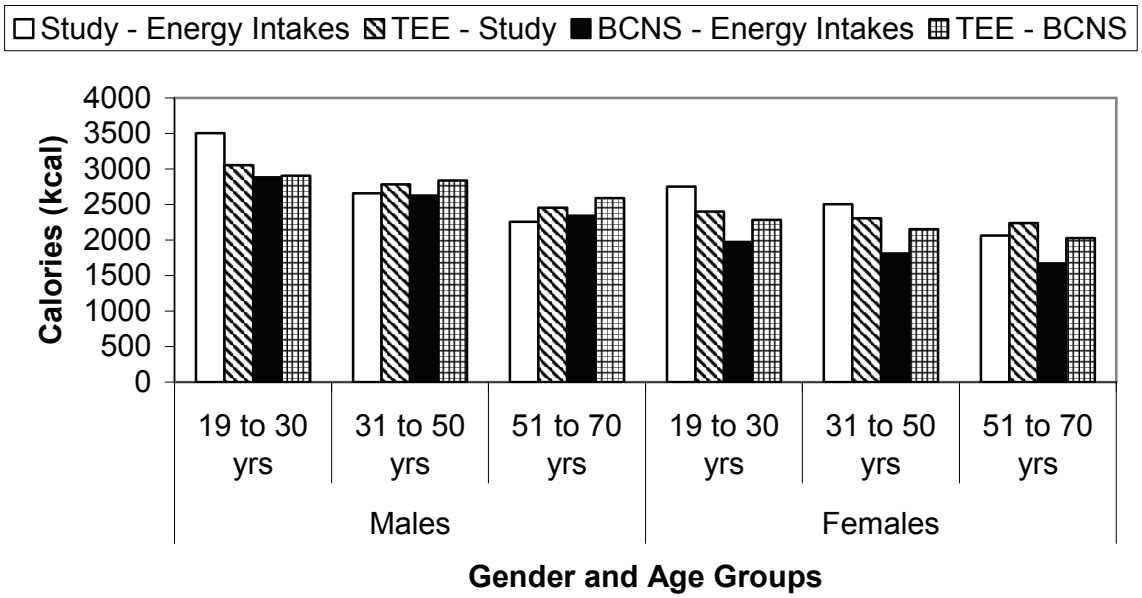
   Appendix C provides full results for nutrients (e.g., descriptive statistics, dotplots of each group) and compares them with the EARs and the ULs.

4.6 Energy and Macronutrients

Figure 4.0.1 illustrates the mean energy intakes compared to the total energy expenditure (TEE) for the study and BCNS sample. In Appendix C, a table of energy intakes (kcal) by gender and age compared to the BCNS (Table C.0.1) is outlined. Energy intakes decreased with age, as one would expect. TEE formulas are presented in the

footnotes of Figure 4.0.1. Basal Energy Expenditure (BEE) was based on formulas derived from using the doubly labelled water technique and use of measured heights and weights (where provided) and a physical activity coefficient (PA) that assumed participants were sedentary or engaged in low levels of activity[303]. These selected PAs were based on documentation that the majority of Canadians are relatively inactive[317].

Figure 4.0.1: Mean energy intakes (kcal) and total energy expenditure by age and gender and compared to British Columbia Nutrition Survey (BCNS)



Notes:
Total Energy Expenditure (TEE) low activity for men = TEE for those whose activity is low (defined as TEE/Basal Energy Expenditure (BEE) of 1.4-<1.6), estimated using the following formula = 662 - 9.53 x Age (yr) + PA x (15.91 x Weight [kg] + 539.6 x Height [m]), where PA is the physical activity coefficient and equal to 1.11
Total Energy Expenditure (TEE) low activity for women = Total Energy Expenditure for those whose activity is low, estimated using the following formula = 354 – 6.91 x Age (yr) + PA x (9.36 x Weight [kg] + 726 x Height [m]), where PA is the physical activity coefficient and equal to 1.12

When comparing the intakes of participants to their estimated requirements, mean energy intakes seem to be slightly under-reported for three of the age groups and in particular for men. Research has shown that respondents with low food consumption tend to overestimate their intake and those with high consumption underestimate their intake. Food records tend to underestimate intake by 10%–20% compared to observed intake[318]. There were no significant differences in energy intakes between the study sample and

BCNS. Figure C.0.1 (Appendix C) shows dot plots of each subgroup; underlying mean caloric levels of the 6 groups were significantly different [$F(5, 91) = 3.27$, $p < 0.05$] between males 19 to 30 years and females 51 to 70 years ($p < 0.05$).

Overall mean values for the proportion of energy derived from alcohol and macronutrients are presented in Table 4.0.4. Distributions of energy derived from the macronutrients are outlined in Table 4.0.5. Shaded areas represent the Acceptable Macronutrient Distribution Ranges (AMDR) that are based on chronic disease prevention and ensuring adequate nutrient intakes. These are 45%–65% of energy from carbohydrates, 20%–35% from fat, and 10%–35% from protein. Table 4.0.7 presents the distribution of energy derived from saturated fat, linoleic acid, and α-linolenic acid.

The percentage of energy derived from carbohydrates for females 19 to 30 years was slightly higher than in the AMDR and almost 15% higher than in the BCNS (Table 4.0.5); the percentage of energy derived from fat for females aged 51 to 70 years was higher than the AMDR (Table 4.0.6). In addition, a high proportion of females aged 51 to 70 years were not meeting the AMDR for carbohydrates, while 8% of the study sample exceeded it. Half (50%) the participants exceeded the AMDR for fat. Among participants aged 31 to 50 years, 6%–8% were not meeting the AMDR for protein. Over 90% of the study group consumed less than 5% of their energy from linoleic acid and more than 50% of the sample were consuming excess saturated fats (Table 4.0.6). Over 90% were consuming less than 0.6% of the calories from α-linolenic acid. The health implications of high total and saturated fat intakes include high blood lipid levels.

4.7.1 Carbohydrates

The EAR is set at 100 grams/day and is based on the amount of carbohydrate needed to produce enough glucose for brain function[319]. Only two participants (females 51 to 70 years) were below this amount. Overall median carbohydrate intake was higher compared to the BCNS but did not significantly differ (Figure 4.0.2). The proportion of the study sample consuming carbohydrates within the AMDR was significantly less than the BCNS (range of $p < 0.05$ to 0.001); the proportion of females 51 to 70 was also significantly less than the BCNS ($p < 0.05$). Females aged 19–50 years reported consuming significantly more carbohydrates than those in same age group of the BCNS ($p < 0.05$) (Table C.0.2).

Table 4.0.4: Percentage of energy derived from alcohol, carbohydrates, fat, and protein by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (mean values)

Nutrient	Males (%)						Females (%)					
	19-30 yr		31-50 yr		51-70 yr		19-30 yr		31-50 yr		51-70 yr	
	Study (n=4)	BCNS (n=142)	Study (n=17)	BCNS (n=205)	Study (n=7)	BCNS (n=249)	Study (n=9)	BCNS (n=176)	Study (n=39)	BCNS (n=266)	Study (n=21)	BCNS (n=282)
Alcohol	0	2.7	2.8	2.6	1.4	3.4	3.8	3.3	2.0	2.5	2.0	2.0
Carbohydrate	47.2	50.0	48.9	48.5	50.9	49.1	66.2	52.8	51.7	48.9	48.6	50.0
Fat	33.4	30.9	32.4	32.5	25.1	31.8	27.3	30.9	29.3	33.5	37	31.5
Protein	13.5	15.6	14.0	16.5	15.4	15.9	14.0	13.8	15.2	15.5	17	16.3

Table 4.0.5: Distribution of energy derived from carbohydrates, total fat, and protein by age and gender (expressed as a percentage of energy) and compared to the British Columbia Nutrition Survey (BCNS)

	Total		Males						Females					
			19-30 yr		31-50 yr		51-70 yr		19-30 yr		31-50 yr		51-70 yr	
	Study (n=97)	BCNS (n=1320)	Study (n=4)	BCNS (n=142)	Study (n=17)	BCNS (n=205)	Study (n=7)	BCNS (n=249)	Study (n=9)	BCNS (n=176)	Study (n=39)	BCNS (n=266)	Study (n=21)	BCNS (n=282)
Carbohydrate (% of total energy intake)														
< 45	27	22	25	18	35	34	14	23	<1	9	18	23	38	18
														57
														24
≥ 65	8	2	0	2	6	4	0	4	<1	3	8	0	5	1

Note: The shaded areas represent the Acceptable Macronutrient Distribution Ranges (AMDR) where appropriate and the proportion of the samples meeting these recommendations
*Significant differences between the study sample and BCNS at $p < 0.05$ **Significant differences between the study sample and BCNS at $p < 0.001$

Table 4.0.5: Distribution of energy derived from carbohydrates, total fat, and protein by age and gender (expressed as a percentage of energy) and compared to compared to the British Columbia Nutrition Survey (BCNS) /continued

	Total		Males						Females					
			19-30 yr		31-50 yr		51-70 yr		19-30 yr		31-50 yr		51-70 yr	
	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS
Fat (% of total energy intake)														
< 20	3	1	0	0	0	1	0	1	<1	0	5	2	5	2
														16
														26
														34
≥35 to < 40	45***	19	25	12	35	23	14	16	<1	14	26	25	14	16
≥ 40	5	6	25	3	29	4	14	5	<1	5	23	10	33	7
Protein (% of total energy intake)														
< 10	4	2	0	3	6	2	0	1	0	7	8	1	0	0
														98
														2
														0
Saturated Fat[1] (% of total energy intake)														
< 5%	5	2	0	0	0	1	0	0	<1	1	3	1	14	2
≥ 5 to < 10	38*	51	25	55	53	48	43	49	57	61	37	44	33	50
≥ 10 to < 20	56*	47	75	45	41	51	57	51	<1	37	60	55	53	47
≥ 20	1	0	0	0	6	0	0	0	0	0	0	0	0	1

Note: The shaded areas represent the Acceptable Macronutrient Distribution Ranges (AMDR) where appropriate and the proportion of the samples meeting these recommendations

[1]No AMDR is set for saturated fats

*Significant differences between the study sample and BCNS at $p < 0.05$

**Significant differences between the study sample and BCNS at $p < 0.001$

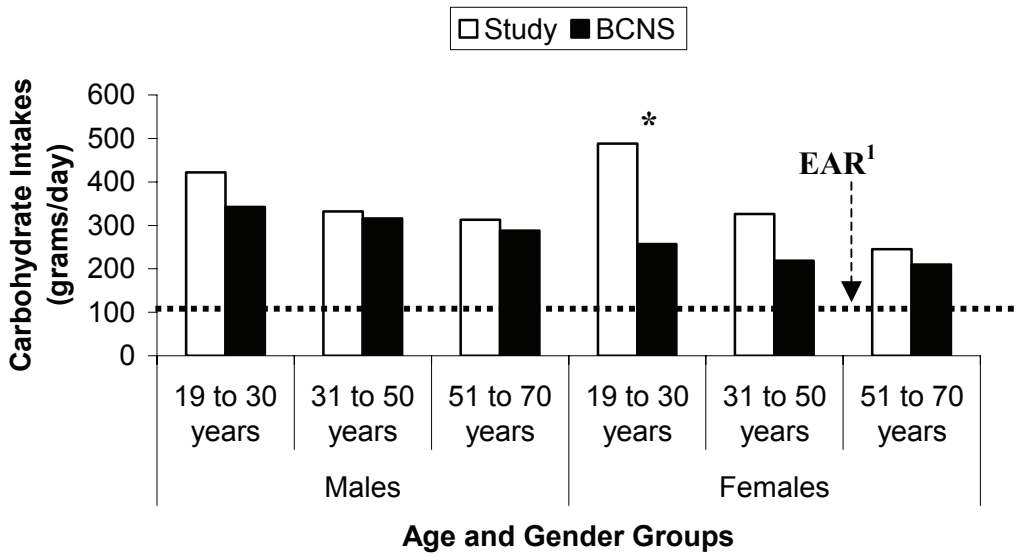
***Significant differences between the study sample and BCNS at $p < 0.0001$

Table 4.0.6: Distribution of energy derived from saturated fat, linoleic acid, and alpha-linolenic acid by age and gender (expressed as a percentage of energy) and compared to the British Columbia Nutrition Survey (BCNS)

	Total		Males						Females					
			19-30 yr		31-50 yr		51-70 yr		19-30 yr		31-50 yr		51-70 yr	
	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS	Study	BCNS
Linoleic acid (% of total energy intake)														
< 5	96**	76	50	81	100	67	71	61	100	84	95*	77**	86	85
														16
≥ 10	1	<1	25	0	0	0	14	1	0	0	0	0	4	0
α-Linolenic acid (% of total energy intake)														
< 0.6	91**	23	100	40	94**	23	86*	33	100	32	97**	13	81**	10
														79**
≥ 1.2	1*	7	0	10	0	6	0	14	0	1	0	4	5	11

Note: The shaded areas represent the Acceptable Macronutrient Distribution Ranges (AMDR) where appropriate and the proportion of the sample meeting these recommendations
*Significant differences between the study sample and BCNS at $p < 0.05$
**Significant differences between the study sample and BCNS at $p < 0.001$

Figure 4.0.2: Median carbohydrate intakes (grams/day) by age and gender and compared to the British Columbia Nutrition Survey (BCNS)



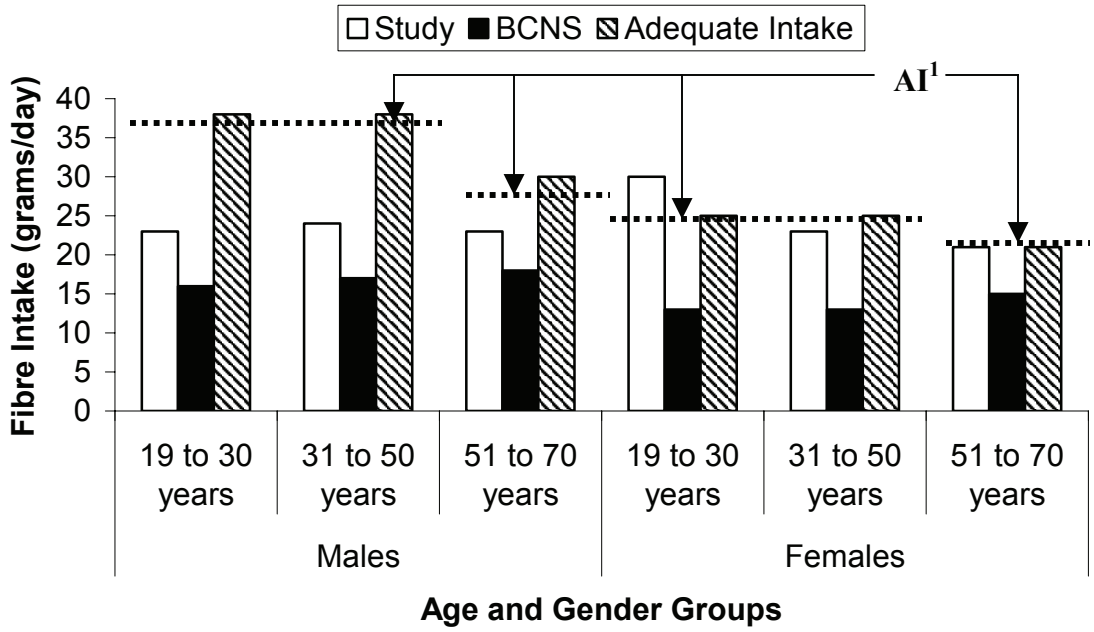
[1]EAR = Estimated Average Requirement (100 grams/day)
*Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.05$

The underlying mean carbohydrate levels of the 6 study groups were significantly higher [$F(5, 91) = 3.89$, $p < 0.05$]. Males aged 19 to 30 years were significantly different from females aged 51 to 70 years ($p < 0.05$) (Figure C.0.2).

4.7.2 Dietary Fibre

The AIs for dietary fibre are based on the amounts needed to help protect against coronary heart disease[319]. The median intakes for fibre were higher for all groups compared to the BCNS (Figure 4.0.3). Overall intakes, however, were not significantly different. Only females 19 to 30 years met the AI for fibre. Fibre intakes may be underestimated by about 5 grams/day, since the database for fibre does not include the contributions of inulin and fructo-oligosaccharides[319]. Mean fibre levels for the 6 groups differed [$F(5, 91) = 2.44$, $p < 0.05$], which was attributable to the higher intakes of females 19 to 30 years compared to females 51 to 70 years ($p < 0.05$) (Figure C.0.2).

Figure 4.0.3: Fibre intakes by age and gender compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams/day)

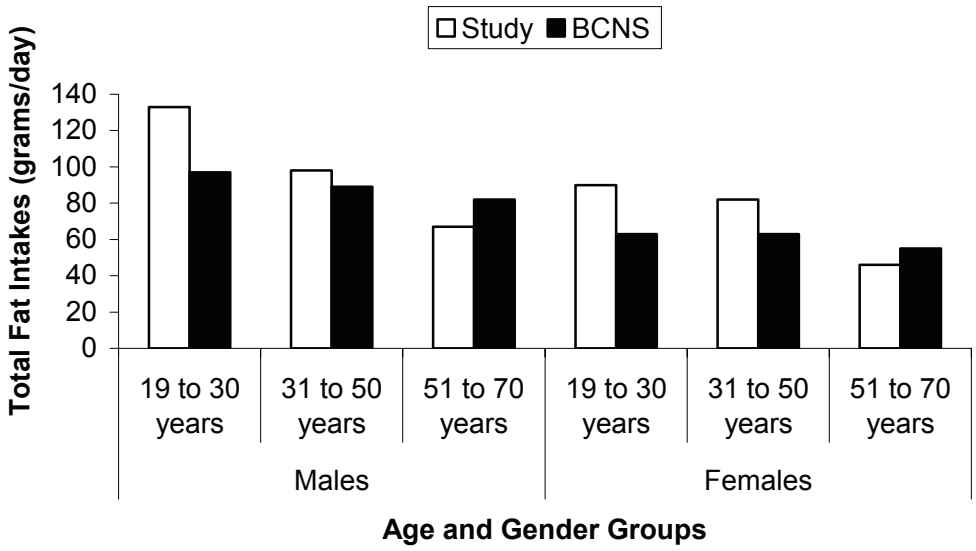


Adequate Intakes for fibre are 38 grams/day for men 19 to 50 years, 25 grams/day for women 19 to 50 years, 30 grams/day for men over 50 years and 21 grams/day for women over 50 years of age

4.7.3 Dietary Fat

In general, median intakes were higher (but not significantly) overall or in all subgroups compared to the BCNS, with the exception of males and females aged 51 to 70 years (Figure 4.0.4). Figure C.0.3 (Appendix C) presents the dot plots of total fat intake for specific age and gender groups. One-way ANOVA results indicate no significant differences among mean fat levels of the 6 groups [F (5, 91) = 1.75, ns].

Figure 4.0.4: Median intakes of total fat by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams/day)



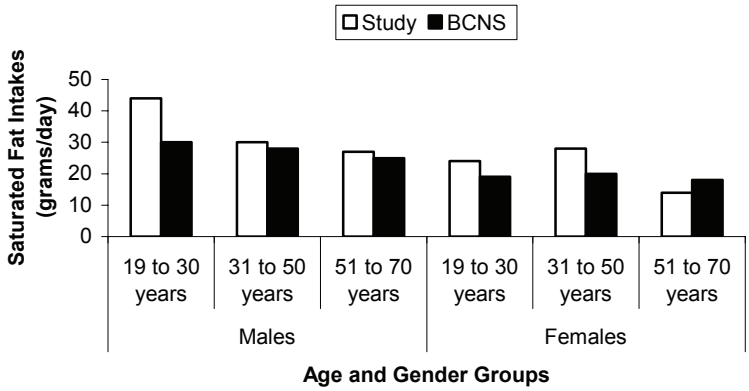
Note: There are no *DRI*s set for total dietary fat

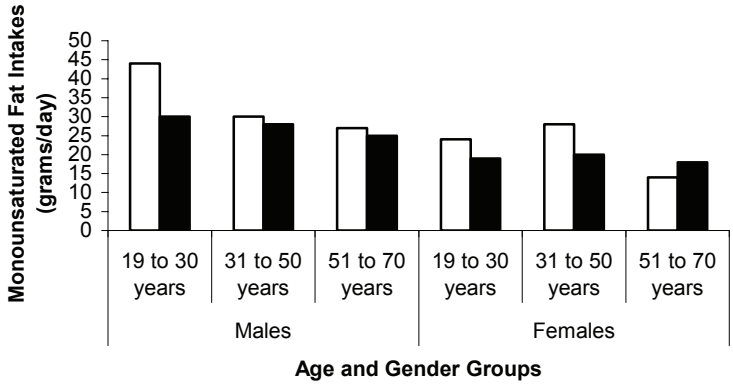
4.7.3.1 Fats by Subtype

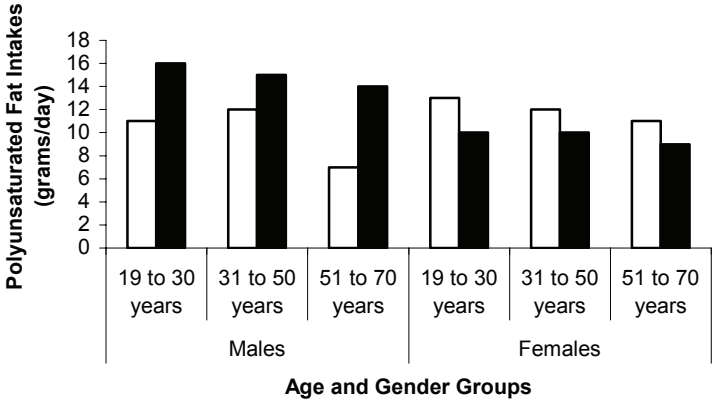
Figure 4.0.5 outlines the intakes of saturated, polyunsaturated, and monounsaturated fats. It is recommended that saturated fats consumption be kept to a minimum without compromising the nutritional benefits of foods that contain them. Almost half of the sample was consuming 10% or more of their calories as saturated fats, so intakes were inconsistent with this guideline. Median saturated intakes tended to be higher than the BCNS but none of the trends were significant (Table C.0.3). The underlying mean saturated fat levels of the 6 groups did not differ [F (5, 91) = 1.37, ns] (Figure C.0.3).

Generally, median monounsaturated intakes were higher in the study but not significantly (Figure 4.0.6). Also, the underlying mean monounsaturated fat levels of the 6 groups did not differ [F (5, 91) = 0.98, ns] (Figure C.0.4). Mean polyunsaturated fat levels of the subgroups were not significantly different [F (5, 91) = 0.97, ns] (Figure C.0.4).

Figure 4.0.5: Intakes of saturated, monounsaturated, and polyunsaturated fat by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams/day)







Note: There are no *DRI*s set for saturated, monounsaturated, and polyunsaturated fat

Linoleic and α-Linolenic Acids

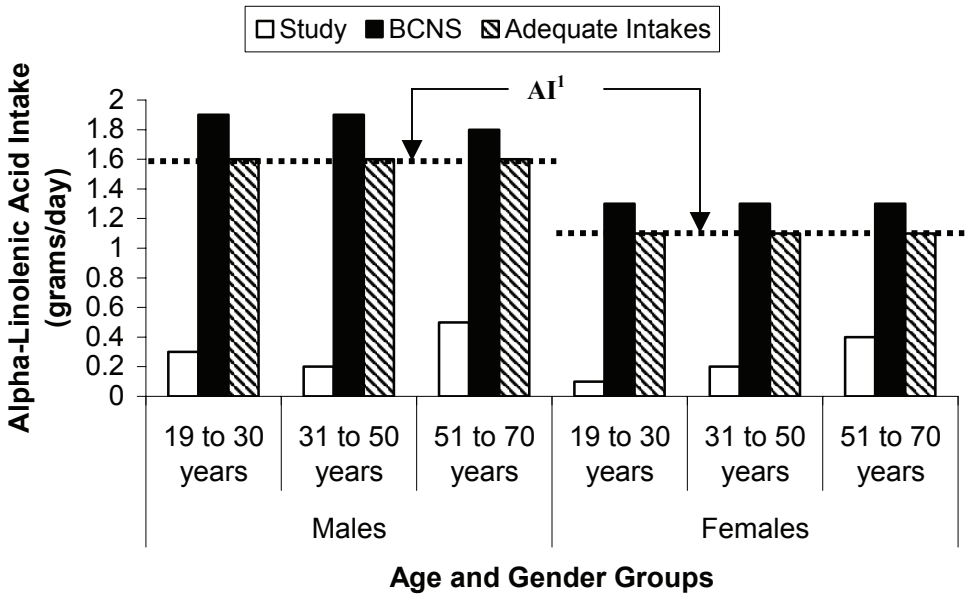
Two families of polyunsaturated fatty acids are considered essential: linoleic acid and α-linolenic acid. AIs for these fatty acids are based on median intakes in the U.S. α-linolenic acid has distinct nutritional functions and serves as the precursor for two other fatty acids: eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). The differences between α-linolenic and linoleic acid occur in their chemical structure. They are not interchangeable. The body needs about 10 times as much linoleic acid as linolenic acid to balance eicosanoid effects. Median α-linolenic intakes did not meet the AI of 1.6 grams/day for males and 1.1 grams/day for females (Figure 4.0.6) and linoleic acid intakes were consistently lower than the BCNS and AIs (Figure 4.0.5). There were no significant differences for overall intakes of α-linolenic and linoleic acid between the study sample and BCNS.

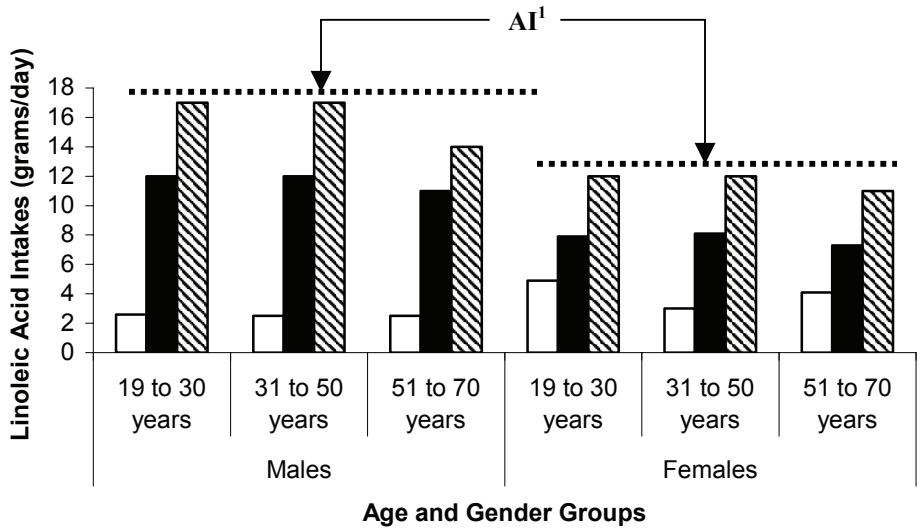
Dot plots of α-linolenic and linoleic fat intake (Figure C.0.5) indicated that three females over 31 years had very high intakes of α-linolenic acid due to a high intake of vegetable oils and fortified products. For all groups, the distribution range was small, though a number of outliers were present in the older female groups. There were no significant differences among α-linolenic fat levels of the 6 groups [$F(5, 91) = 0.41$, ns]. Compared to α-linolenic intakes, there was a wider distribution and more outliers in the older female groups for linoleic acid. The unusually high intakes were due to the consumption of food products fortified with linoleic acid. There was insufficient evidence to reject the null hypothesis that the underlying mean linoleic acid levels of the 6 groups were the same [$F(5, 91) = 0.30$, ns].

4.7.4 Cholesterol

The median intakes for dietary cholesterol ranged from 147 mg for the youngest female group to almost 455 mg in the youngest male group (Figure 4.0.7). When compared to the BCNS, intakes did not differ significantly. According to Table C.0.4, the majority of participants consumed less than 300 mg of cholesterol per day. No significant differences were found among the study groups [$F(5, 91) = 2.09$, ns] (Figure C.0.6).

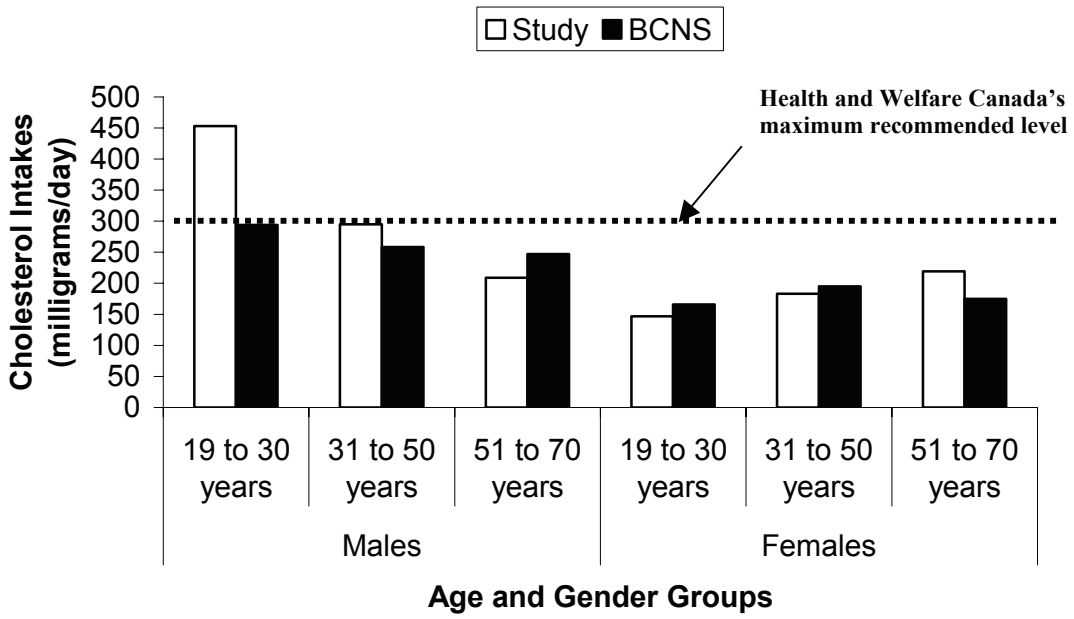
Figure 4.0.6: Median alpha-linolenic and linoleic acid intakes by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams/day)





[1]AI = Adequate intakes and are 1.6 grams/day for males and 1.1 grams /day for females (α-linolenic acid) and 17 grams/day and 12 grams/day for males (linoleic acid)

Figure 4.0.7: Median cholesterol intakes by gender and age and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

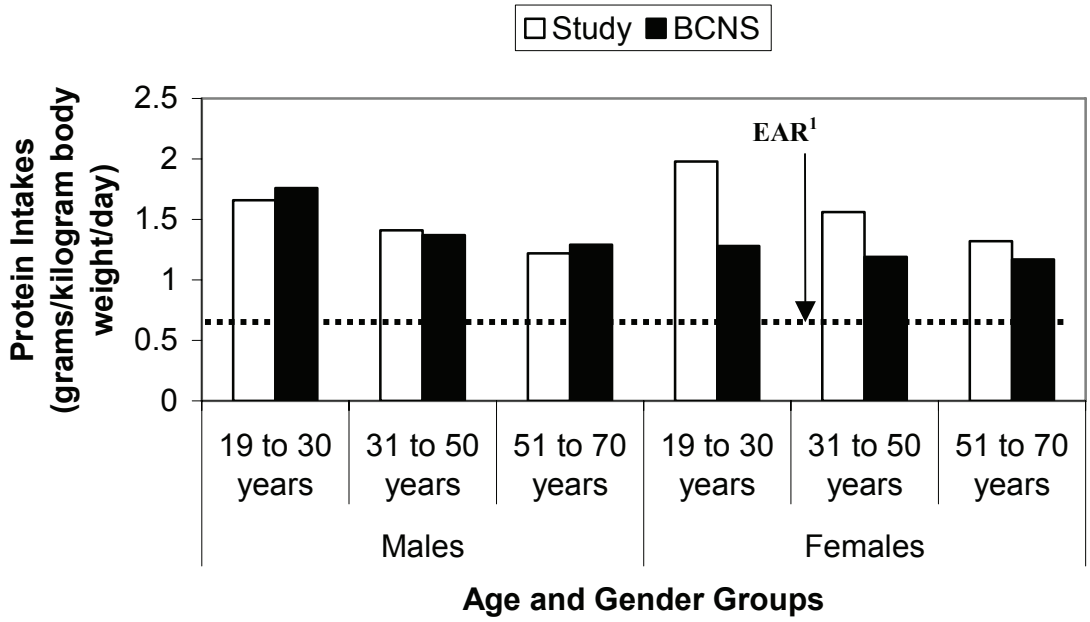


Note: Health and Welfare Canada recommends that Canadians not exceed 300 mg of dietary cholesterol per day

4.7.5 Protein

As presented previously, 96% of participants were consuming 10% or more of their calories from protein. This is well within the acceptable range; however, a high proportion of participants were below the EAR (Table C.0.5), which is based on the lowest continuing intake of dietary protein required for body nitrogen equilibrium[319]. The health implications of not meeting the EAR for protein are unknown in mental health populations as they are the only dietary source of many neurotransmitter precursors. No differences were found between the study sample and BCNS or among the study sample groups [F (5, 82) = 2.20, ns] (Figures 4.0.8 and C.0.7).

Figure 4.0.8: Median protein intakes by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams/kilogram body weight/day)



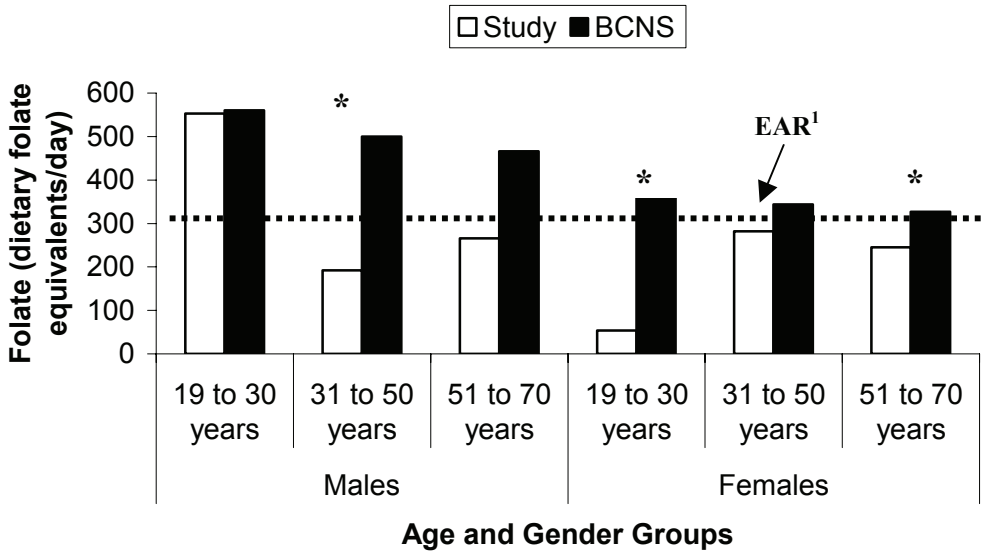
[1]EAR = Estimated Average Requirement (0.66 grams/kilogram body weight/day)

4.8 Vitamins

4.8.1 Folate

Low blood folate is associated with elevated serum homocysteine[320;321]. Folate intakes were expressed as dietary folate equivalents (DFE) which adjust for the lower bioavailability of naturally occurring folate in foods compared to the synthetic form (i.e., folic acid)[321] (Figure 4.0.9). The EAR for folate (320 DFE/d) and is based on the maintenance of red blood cell folate levels at 305 nmol/L and plasma homocysteine levels at or below 14 μmol/L[322]. The sample had a significantly larger proportion that were below the EAR (n = 62; 64% vs n = 358; 27%; $p < 0.0001$) (Figure 4.0.9 and Table C.0.6) compared to the BCNS. The study sample also had significantly lower intakes for males aged 31 to 50 years ($t = -2.46$, $p < 0.05$, 95% CI -585.15 to -44.85), and females aged 19 to 30 ($t = -2.83$, $p < 0.05$, 95% CI -434.49 to -47.51) and 51 to 70 years ($t = -2.41$, $p < 0.05$, 95% CI -155.06 to -12.94). No significant differences existed among the study groups [$F (5, 91) = 0.39$, ns] (Figure C.0.8).

Figure 4.0.9: Median folate intakes from food by age and gender and compared to the British Columiba Nutrition Survey (BCNS) (expressed as Dietary Folate Equivalents/day)

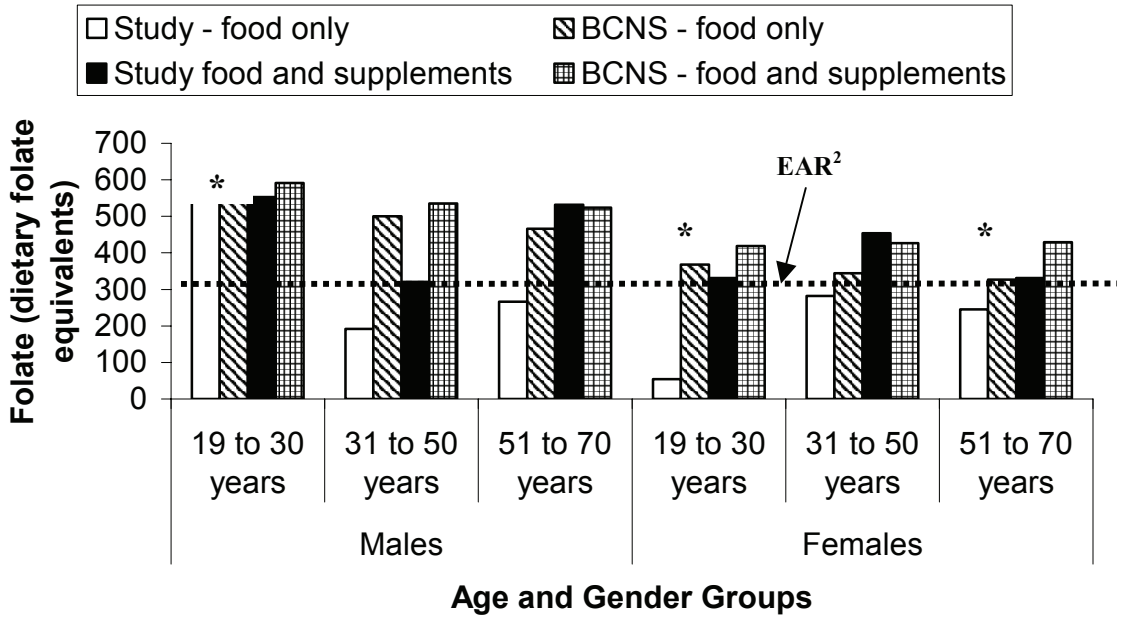


Dietary Folate Equivalents (DFE) = values that adjust for the differences in absorption of food folate and synthetic folic acid.1 mcg of DFE = 0.6 mcg of folic acid from fortified food or as a supplement taken with a meal = 1 mcg food folate = 0.5 mcg of folic acid from a supplement taken on an empty stomach
[1]EAR = Estimated Average Requirement (320 micrograms of DFE/day)
[*]Significant differences for that age and gender group between the sample and BCNS at $p < 0.05$

When fortified foods were included in the estimation (Figure 4.0.10), significant differences between the study and BCNS remained (n = 38; 37% vs n = 225; 17% for the BCNS; $p < 0.0001$) and between average intakes for males 31 to 50 years ($t = -3.23$, $p < 0.05$, 95% CI -588.54 to -133.46) and 19 to 30 years ($t = -2.28$, $p < 0.05$, 95% CI -377.05 to -4.95), and 51 to 70 years ($t = -2.36$, $p < 0.05$, 95% CI -372.30 to -27.70). When foods (including fortified) and supplements were considered, fewer people had intakes below the EAR; when compared to the UL (mcg/day; Table C.0.7), a sizable proportion had excess intakes. No significant differences were found among the groups [$F (5, 91) = 0.51$, ns] (Figure C.0.8). Flours, grains and pastas of the Canadian food supply are fortified with enough folic acid to increase the average intake by about 100 mcg/d[321]. However, this poses risks of folic acid overconsumption, which would mask vitamin B_{12} deficiency. Thus, a UL for folic acid (1000 mcg) was established for synthetic forms of the vitamin[322] and based on case reports of neurological effects in vitamin B_{12}-deficient

patients taking folate supplements. Many participants (Table C.0.7) were above the UL for folate; a significantly higher proportion (n = 16, 16% vs n = 61, 5% for the BCNS; p < 0.0001) compared to the BCNS. However, because most were taking some form of vitamin B_{12} supplement, over-consumption of synthetic folic acid was not a concern.

Figure 4.0.10: Median folate intakes from food sources (including fortified foods) and from food sources (including fortified foods) and supplements by age and gender and compared to the British Columiba Nutrition Survey (BCNS) (expressed as Dietary Folate Equivalents[1]/day)



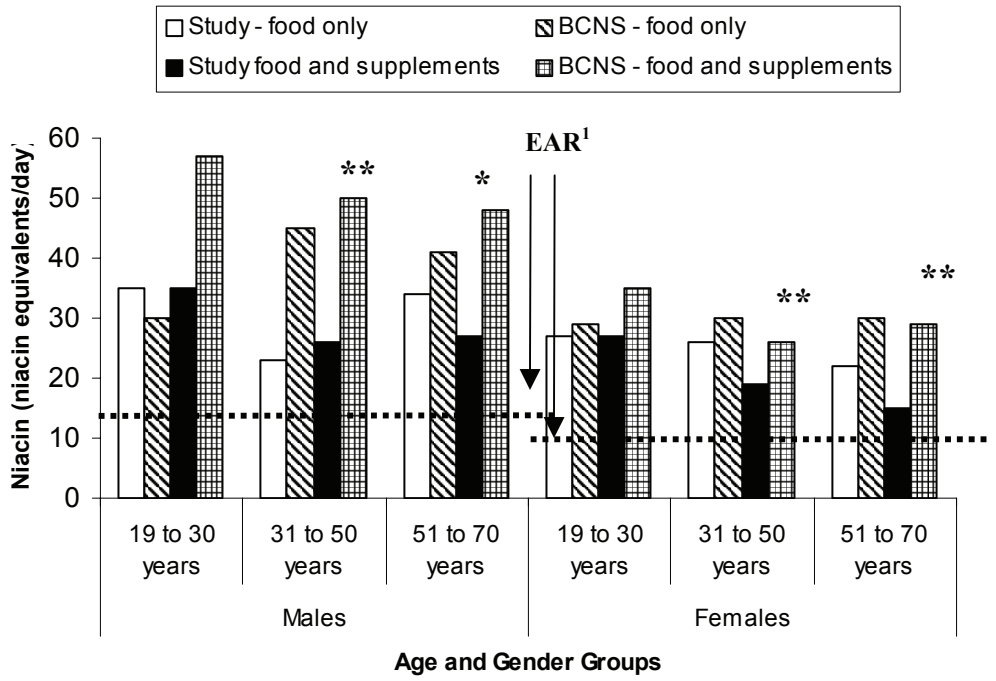
[1]Dietary Folate Equivalents (DFE) = values that adjust for the differences in absorption of food folate and synthetic folic acid. 1 mcg of DFE = 0.6 mcg of folic acid from fortified food or as a supplement taken with a meal = 1 mcg food folate = 0.5 mcg of folic acid from a supplement taken on an empty stomach
[2]EAR = Estimated Average Requirement (320 micrograms of DFE/day)
*Significant differences for that age and gender group between the sample and BCNS at $p < 0.05$ for food intakes

4.8.2 Niacin

No significant differences were found between the overall study sample and BCNS (Figure 4.0.11, Figure C.0.9). Food intakes of niacin were significantly lower in males aged 31 to 50 years ($t = -10.21$, $p < 0.0001$, 95% CI -34.92 to -23.08) and 51 to 70 years ($t = -5.65$, $p < 0.05$, 95% CI -42.95 to -17.05), and females aged 31 to 50 years ($t = -8.57$, $p < 0.0001$, 95% CI -28.30 to -17.70) and 51 to 70 years ($t = -20.32$, $p < 0.0001$,

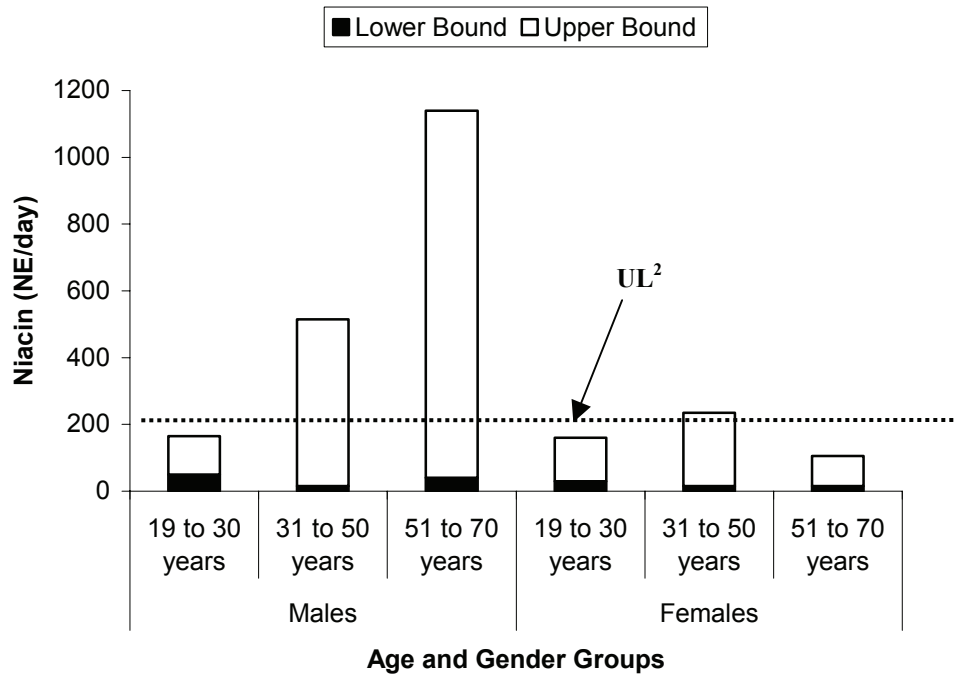
95% CI -60.51 to -49.49). For niacin intake (food and supplements), no significant differences were found among the groups [F (5, 91) = 1.05, ns] (Figure C.0.9). The UL for niacin is based on synthetic forms (i.e., supplements, fortified foods, and pharmacological agents) that induce flushing. Niacin excesses may be due to people receiving pharmacological doses to manage serum cholesterol levels. Synthetic niacin was estimated from the supplement data (Figure 4.0.12) and suggests that for the majority of those who took supplements the ULs were exceeded. Most participants exceeded the ULs for all groups and the overall proportion of participants (n = 27, 28%) was significantly higher ($p < 0.05$) than the BCNS (n = 219, 17%) (Table C.0.8).

Figure 4.0.11: Median niacin intakes from food and from food and supplements by age and gender and compared to the British Columiba Nutrition Survey (BCNS) (expressed as Niacin Equivalents[+]/day)



[+]Niacin equivalents = amount of niacin including from tryptophan (1 mg of niacin ~ about 60 mg of dietary tryptophan)
[1]EAR = Estimated Average Requirement (12 grams/day for males; 11 grams/day for females)
Significant differences between the sample and BCNS at $^{*}p < 0.05$ and $^{**}p < 0.0001$ for food plus supplements

Figure 4.0.12: Lower and upper levels of niacin intake from supplement sources only (expressed as Niacin Equivalents (NE)[1]/day)

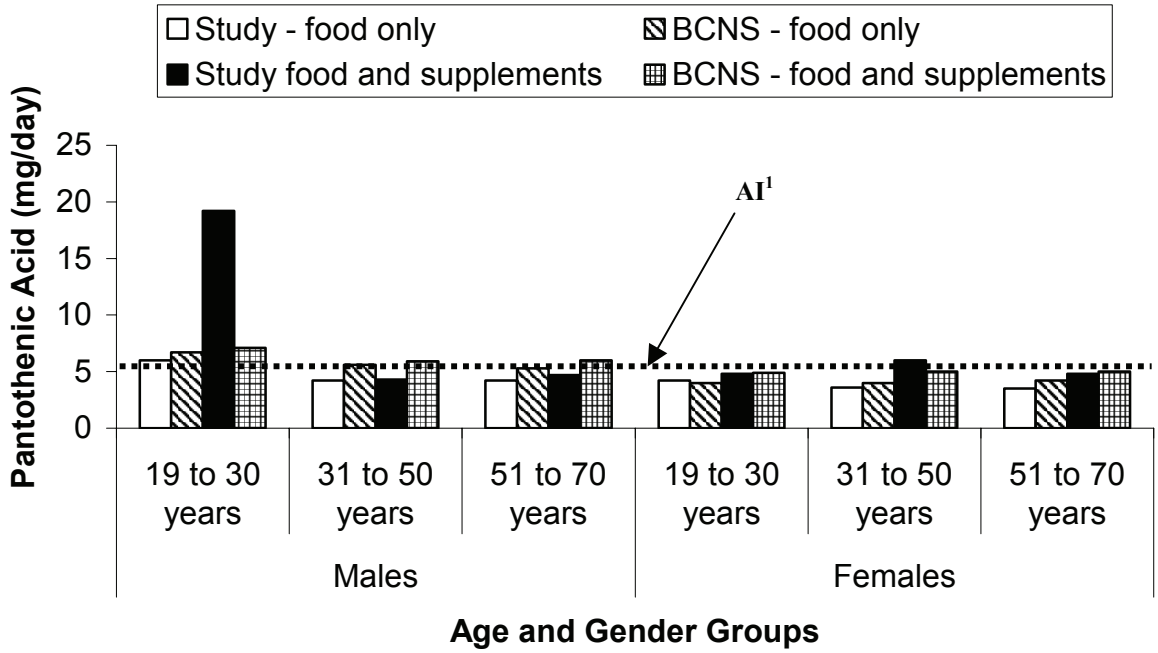


[1]Niacin equivalents = amount of niacin, including the niacin that can theoretically be made from tryptophan.
[2]UL = Tolerable Upper Intake Levels (35 mg/day)

4.8.3 Pantothenic Acid

Only males aged 19 to 30 years met the AI (5 mg/d) for pantothenic acid (Figure 4.0.13 and Table C.0.9). The health implications of pantothenic acid intakes below the AI are unclear[322]. When supplement intake of pantothenic acid was included (Figure 4.0.13), median intakes approached or exceeded the AI. No significant differences were found between the study group and BCNS or among the study groups for food [$F(5, 91) = 1.69$, ns] or food and supplement sources ($\chi^2 = 7.7$, df = 5, p ns) (Figure C.0.10).

Figure 4.0.13: Median pantothenic acid intakes from food and from food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)



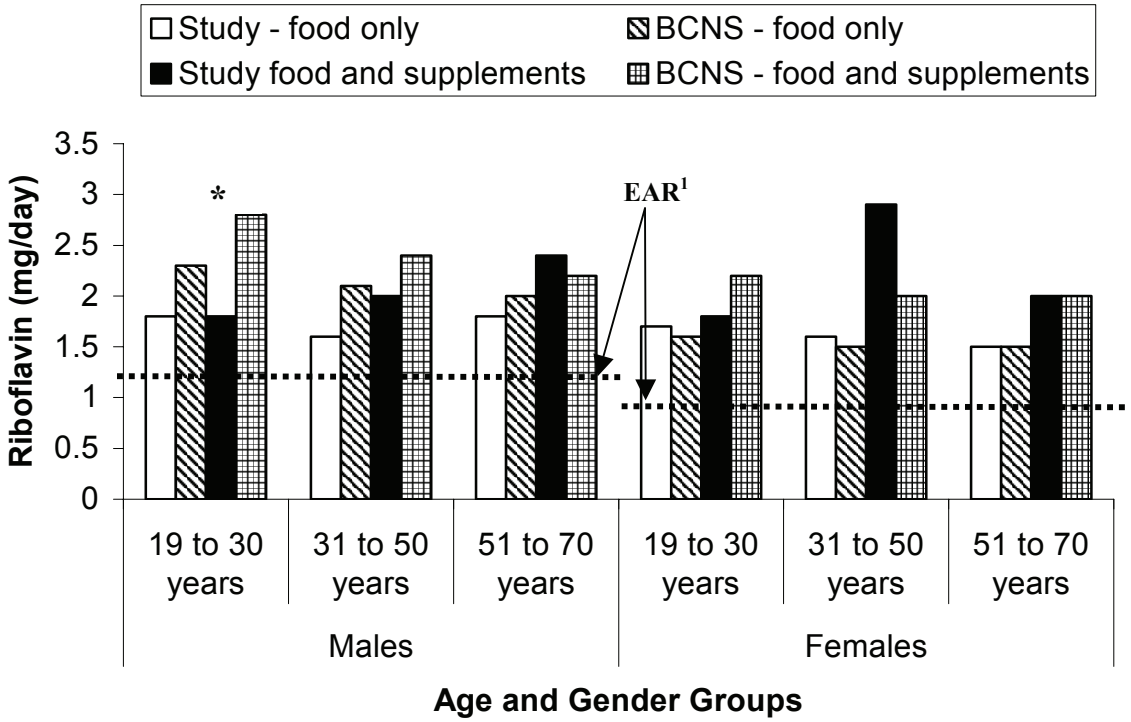
[1]AI = Adequate Intakes (5 mg/day)

4.8.4 Riboflavin

The overall proportion of the study sample that was below the EAR (n = 20, 21%) for food sources of riboflavin was significantly higher than the BCNS (n = 54, 4%; $p < 0.0001$) (Table C.0.10). The distributions of riboflavin intakes from food sources for each subgroup are outlined in Figure C.0.11 (Appendix C). No significant differences were found among the groups [$F(5, 91) = 0.71$, ns].

The overall proportion of the study sample that was below the EAR (n = 12, 12%) for food and supplement sources of riboflavin was significantly higher than the BCNS (n = 40, 41%; $p < 0.0001$). Median intakes of riboflavin including supplements (Figure 4.0.15) showed significant differences between the study and BCNS groups for males 19 to 30 years ($p < 0.05$). Based on the Kruskal-Wallis test, there were no significant differences among the groups ($\chi^2 = 3.1$, df = 5, ns) (Figure C.0.11).

Figure 4.0.14: Median riboflavin intakes from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

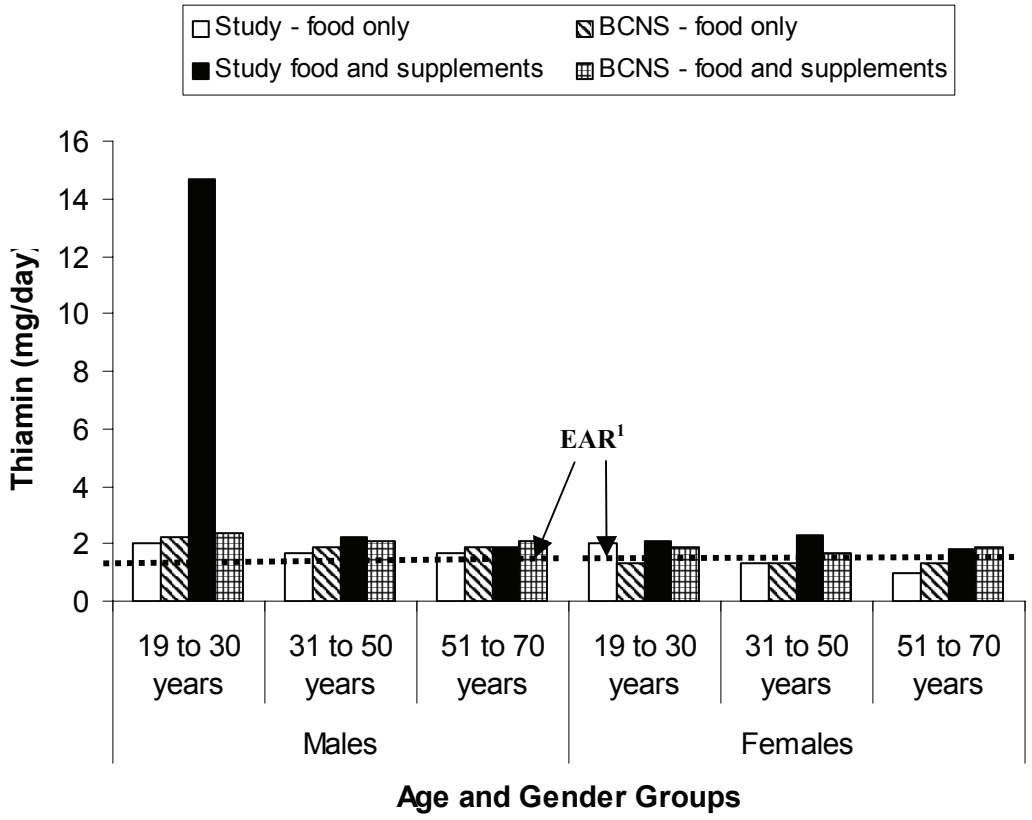


[1]EAR = Estimated Average Requirement (Males = 1.1 mg/day, females = 0.9 mg/day)
[*]Significant differences at $p < 0.05$ between the study and BCNS samples for food and supplement intakes

4.8.5 Thiamin

Thiamin deficiency is of concern as the overall proportion of participants below the EAR (n = 25, 26%) was significantly higher than the BCNS (n = 105, 8%; $p < 0.0001$) (Table C.0.11). There were no significant differences among the study groups [$F(5, 92) = 1.89$, ns] (Figure C.0.12). When food and supplement sources were combined (Figures 4.0.15 and C.0.12), the proportion below the EAR remained significantly higher (n = 11, 11%; n = 72, 5%; $p < 0.05$). Males 19 to 30 years had median thiamin intakes from food and supplement sources that were appreciably higher than all other groups; however, this subgroup had a small sample size so this result should be interpreted with caution. There were no significant differences found among the study groups ($\chi^2 = 2.0$, df = 5, ns).

Figure 4.0.15: Median thiamin intakes from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

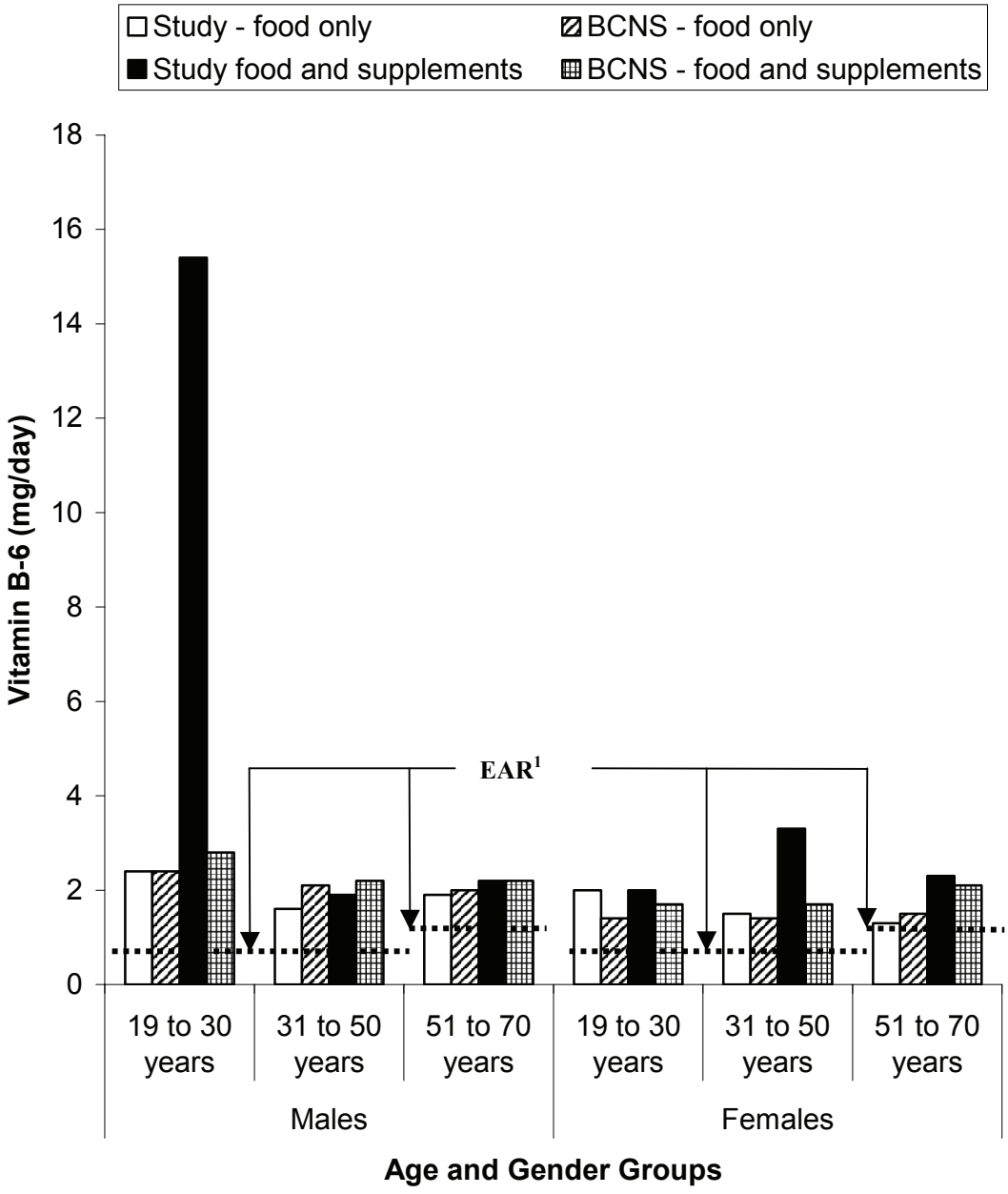


[1]EAR = Estimated Average Requirement (Males = 1.0 mg/day, females = 0.9 mg/day)

4.8.7 Vitamin B_6

The proportion of the sample below the EAR for vitamin B_6 was significantly higher than the BCNS (n = 24, 25%; n = 216, 16%, $p < 0.05$) (Figure 4.0.16 and Table C.0.12). When supplements were included in estimating vitamin B_6 intake the overall proportion of the sample not meeting the EAR remained significantly higher than the BCNS (n = 19, 20% vs; n = 113, 9%, $p < 0.001$) (Table C.0.12). One male between 19 to 30 years had an unusually high vitamin B_6 intake from food and supplements. For females over 19 and males over 31 years, more than 5% were above the ULs. No significant differences were found among the study groups either for food [$F(5, 91) = 1.37$, ns] or food plus supplement intakes [$F(5, 91) = 0.25$, ns] (Figure C.0.13).

Figure 4.0.16: Median vitamin B_6 intakes from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

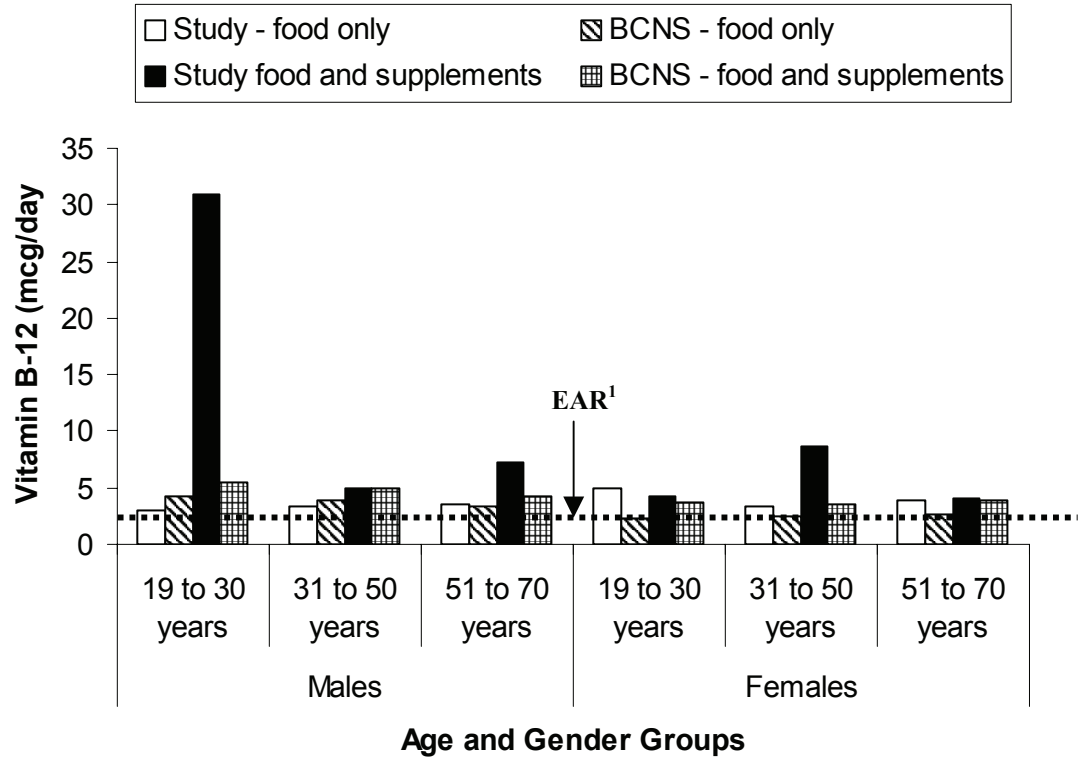


[1]EAR = Estimated Average Requirement (Males and females (19 to 50) = 1.1 mg/day, males (51 to 70) = 1.4, and females (51 to 70) = 1.3 mg/day)

4.8.8 Vitamin B_{12}

The EAR for vitamin B_{12} maintains hematological status[322]. The sample had a significantly higher proportion of intakes below the EAR compared to the BCNS (n = 26, 27% vs n = 241, 18%; $p < 0.05$). Supplements increased median intakes. Because 10-30% of those over 50 years tend to have decreased absorption of this vitamin[322], estimated intakes from supplements alone were generated for this age group. None of the males and 24% of females over 50 years were below the EAR from supplements alone (Table C.0.13). Median intakes for males aged 19 to 30 years were unusually high (Figure 4.0.17); no significant differences were found between the study sample and BCNS or among the study subgroups [F (5, 92) = 0.24, ns] (Figure C.0.14).

Figure 4.0.17: Median vitamin B_{12} intakes from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as micrograms/day)

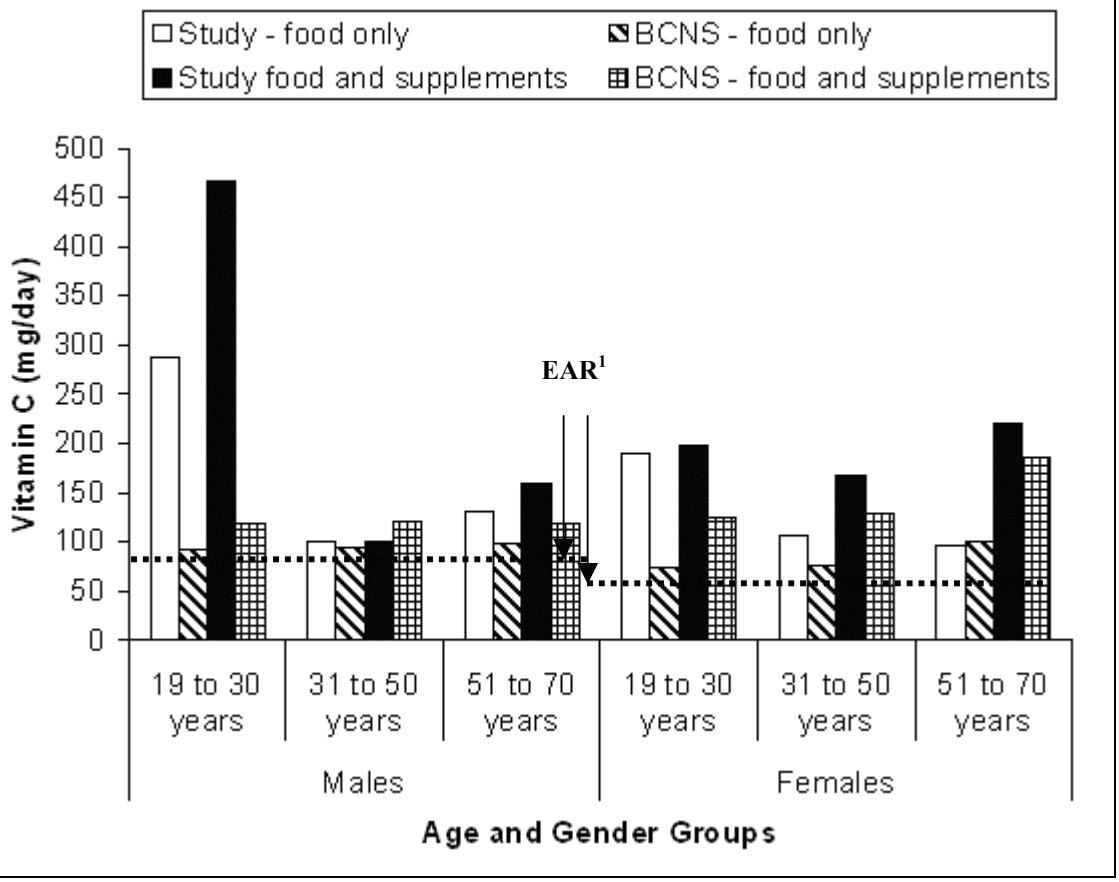


[1]EAR = Estimated Average Requirement (2.0 mg/day)

4.8.9 Vitamin C

The major food sources of vitamin C were fruit juices, for which the sample and BCNS did not differ (Figure 4.0.18). However, differences were found among the study groups [$F(5, 96) = 3.56$, $p < 0.05$] with intakes of males aged 19 to 30 years significantly higher than other males, females aged 31 to 50 years, and females aged 51 to 70 years (Figure C.0.15). Supplementation of vitamin C was common (Figure 4.0.18 and Table C.0.14), but did not differ from the BCNS or among the study groups [$F(5, 91) = 1.07$, ns]. The UL for vitamin C (2000 mg/d) is the amount consumed that results in diarrhoea; therefore, over-consumption of vitamin C is not an important health issue.

Figure 4.0.18: Median vitamin C intake from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)



[1]EAR = Estimated Average Requirement (Males = 75 mg/day and females = 60 mg/day)

Because smoking cigarettes compromises vitamin C properties, the *DRI*s[322] recommend that smokers consume 35 mg more per day than non-smokers. Of the 20 smokers, 40% (n = 8) had vitamin C intakes below the EAR, which did not differ from the non-smokers (35%; z = 0.51, ns). When considering intakes from food and supplements, 15% of non-smokers were below the EAR compared to 30% of smokers (z = 0.42, ns).

4.8.10 Other Vitamins

Other essential vitamins include vitamins D and E, for which the data in the nutrient analysis database were incomplete (i.e., 50% coverage or less). Table 4.0.7 highlights the intakes from food and supplement sources of these nutrients and provides a conservative estimate of the proportion that exceeded the ULs. Six percent of males aged 31 to 50 years and 29% of males aged 51 to 70 years exceeded the UL for the vitamin D. For males aged 51 to 70 years, 14% exceeded the UL for vitamin E.

Table 4.0.7: Intakes from food and supplements of vitamin D and vitamin E

Nutrient	Gender	Age (years)	N	Mean ± SD[1]	50th percentile (25th; 75th)	UL[2]	% > UL[2]
Vitamin D (mcg)	Males	19-30	5	2 ± 2	1 (0.3; 3)	50	0
		31-50	16	27 ± 93	2.0 (0.3; 6)		6
		51-70	7	26 ± 43	4 (3; 11)		29
	Females	19-30	9	5 ± 6	2 (1; 9)		0
		31-50	39	5 ± 6	3 (1; 8)		0
		51-70	21	4 ± 3	3 (1; 4)		0
Vitamin E – alpha tocopherol (mg)	Males	19-30	5	6 ± 6	4 (1; 10)	1000	0
		31-50	16	72 ± 148	7 (1; 60)		0
		51-70	7	321 ± 658	8 (2; 405)		14
	Females	19-30	9	11 ± 14	4 (2; 19)		0
		31-50	39	103 ± 225	6 (2; 43)		0
		51-70	21	109 ± 217	7 (1; 90)		0

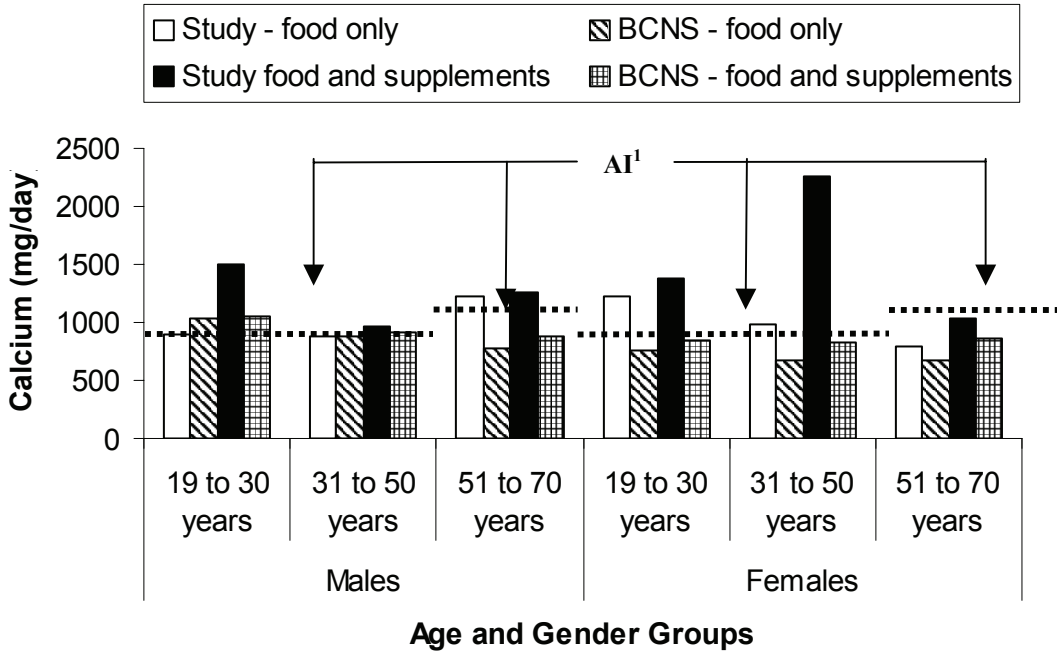
[1]Standard Deviation
[2]Tolerable Upper Intake Levels

4.9 Minerals

4.9.1 Calcium

Low calcium intakes were an issue for both sexes and worsened with age (Figure 4.0.16 and C.0.16). There were no significant differences between the study sample and BCNS or among the subgroups found no differences [F (5, 91) = 2.07, ns]. When supplements were included in the estimation (Figure 4.0.19 and Table C.0.15), there was a significantly higher proportion of the sample (n = 6, 6%) that exceeded the UL compared to the BCNS (n = 10, < 1%, $p < 0.0001$). Differences were found among the study groups [F (5, 91) = 5.58, $p < 0.001$]; males 19 to 30 and 31 to 50 years were significantly higher than males 51 to 70 years ($p < 0.05$). Each female group was also significantly higher than males aged 51 to 70 years (range of $p < 0.05$ to $p < 0.001$) (Figure C.0.16).

Figure 4.0.19: Median calcium intakes from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

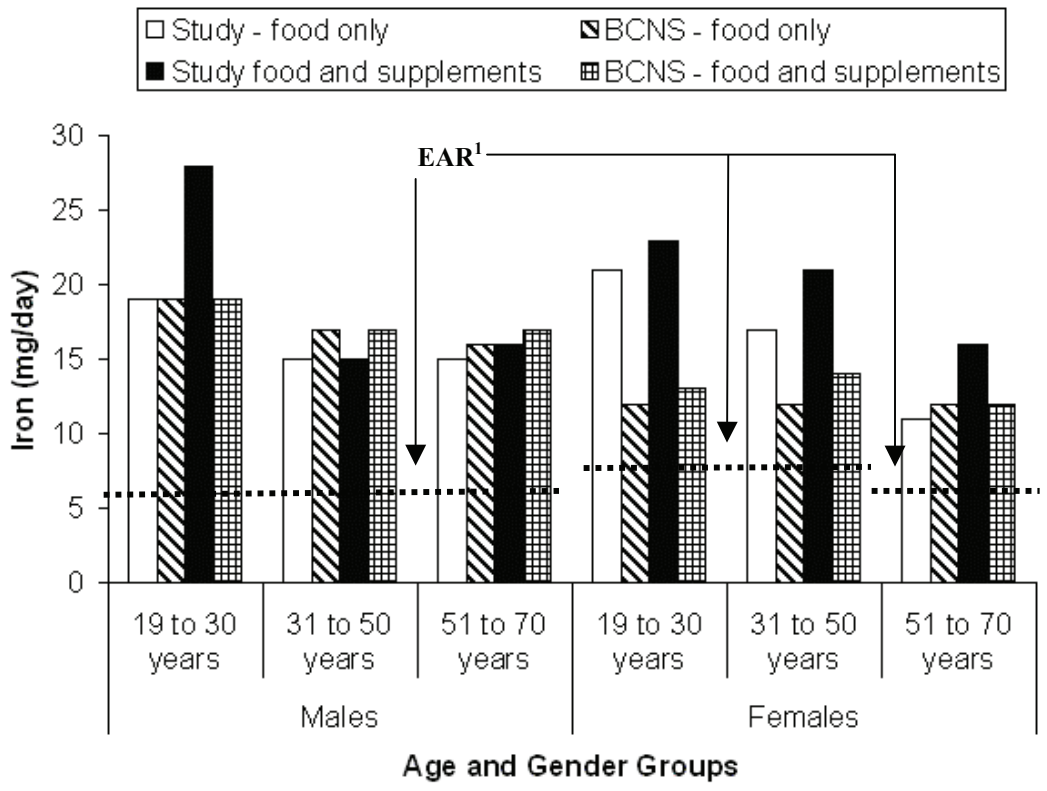


[1]AI = Adequate Intakes (Males and females 19 to 51 years = 1000 mg/day; males and females 51 to 70 years = 1200 mg/day)

4.9.2 Iron

Males 31 to 50 years and all females were at risk for inadequate iron intakes (Figure 4.0.20 and Table C.0.16). There were three females who exceeded the UL for food sources of iron. This was due to consumption of supplemental drinks. Based on food and supplement intakes, a significantly higher proportion of study participants (n = 7, 7%) exceeded the UL compared to the BCNS (n = 22, 2%; $p < 0.001$); excess intakes were a problem for females 19 to 30 years and those in the older age groups. There were significant differences among the study groups ($\chi^2 = 21.83$, df = 5, $p < 0.001$).

Figure 4.0.20: Median iron intakes from food and food plus supplement sources by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

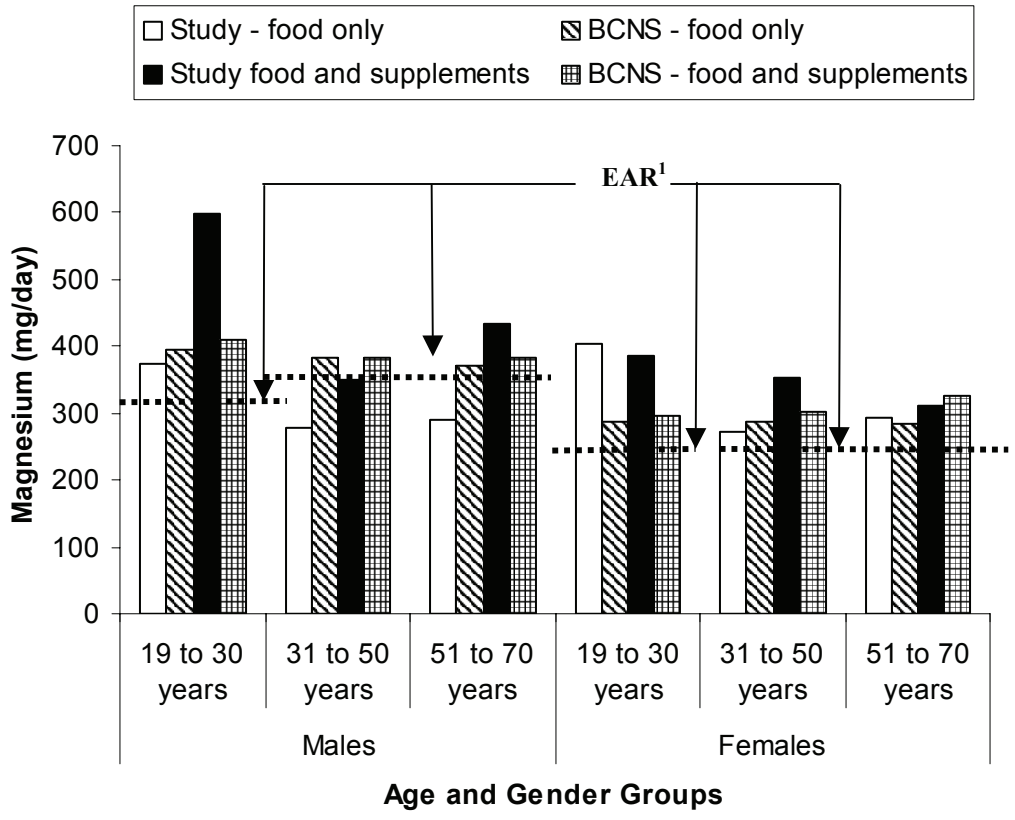


[1]EAR = Estimated Average Requirement (males = 6 mg/day; females 19 to 50 = 8 mg/day and females over 50 = 5 mg/day)

4.9.3 Magnesium

The proportion below the EAR is high (Figure 4.0.21 and Table C.0.17) but no differences were found (Figure C.0.18) compared to the BCNS or among the study groups [F (5, 91) = 0.91, ns]. Supplements affected intakes for those 31 to 50 years (Figure 4.0.21), but many remained below the EAR (Table C.0.17). No differences were found for magnesium intakes between the study and BCNS or among the study groups [F (5, 91) = 0.49, ns]. The UL is based on gastrointestinal effects from consumption of 350 mg or more of the synthetic form. Some between 31 to 70 years were above the UL and this did not take into account other pharmacological sources (e.g., laxatives, antacids).

Figure 4.0.21: Median magnesium intakes from food and supplements by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams/day)

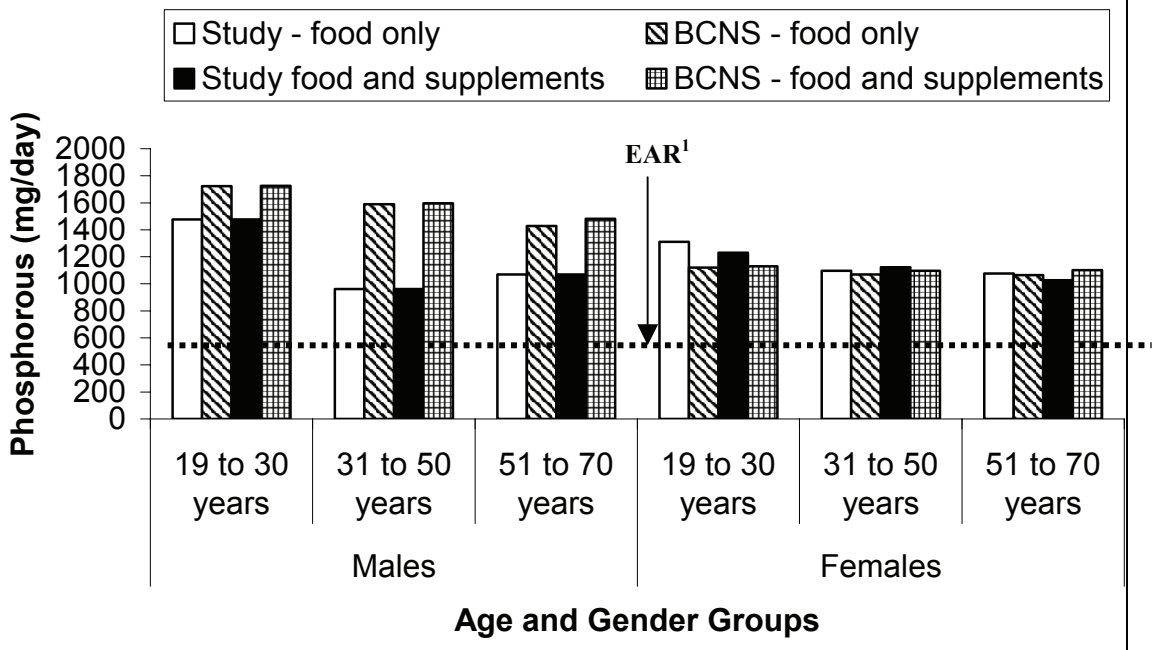


[1]EAR = Estimated Average Requirement (males 19 to 30 yrs = 330 mg/day, 31 to 70 years = 350 mg/day; females 19 to 30 years = 255 mg/day and 31 to 70 years = 265 mg/day)

4.9.4 Phosphorous

Inadequate phosphorus intakes (Figure 4.0.22 and Table C.0.18) were a problem for those 31 to 70 years, but the health implications of this are unclear. The proportion of participants with food intakes of phosphorous below the EAR was significantly higher compared to the BCNS (n = 12, 12% vs n = 18, 1%, $p < 0.0001$). Significant differences remained between the sample and BCNS for EARs remained even when supplemental sources were included (n = 9, 9% vs n = 18, 1%, $p < 0.0001$). The study groups did not differ [$F(5, 91] = 1.14$, ns] for food; however, differences were found for food and supplements [$F(5, 91) = 12.55$, $p < 0.0001$] with intakes of males aged 51 to 70 years significantly higher than all other groups ($p < 0.001$) (Figure C.0.19). When food and supplement sources were considered, the inadequate intakes of males 51 to 70 years and females 31 to 70 years persisted (Table C.0.18).

Figure 4.0.22: Median phosphorous intakes from food and food plus supplement sources by age and gender compared to BCNS (expressed as milligrams/day)

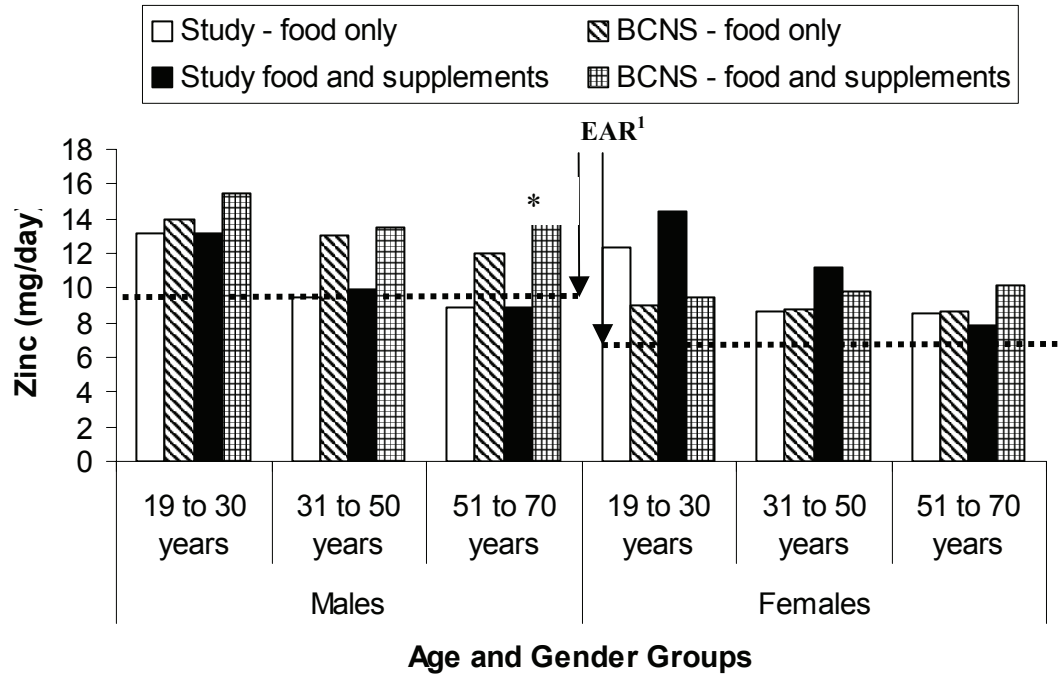


[1]EAR = Estimated Average Requirement (580 mg/day)

4.9.5 Zinc

A large proportion of individuals (25%–57%), especially those over 50 years, were below the EAR for zinc (Table C.0.19 and Figure 4.0.23). The study sample had a significantly higher proportion of participants below the EAR (n = 38, 39%) compared to the BCNS (n = 202, 15%; $p < 0.0001$). Supplements helped to reduce the proportion below the EAR for those 9 to 50 years (Tables C.0.19). In addition, the proportion of the sample below the EAR (n = 31, 32%) remained significantly higher than the BCNS (n = 156, 12%, $p < 0.0001$). Supplement use also contributed to excess intakes (Figure 4.0.23 and Table C.0.19) for males 31 to 50 years and all female groups. Intakes of zinc for the study and BCNS differed significantly in males 51 to 70 years ($t = -21.4$, $p < 0.0001$, 95% CI -9.06 to -7.54). There were no differences among the study groups [$F(5, 91) = 0.44$, ns].

Figure 4.0.23: Median zinc intakes from food and supplements by age and gender and compared to BCNS (expressed as milligrams per day)



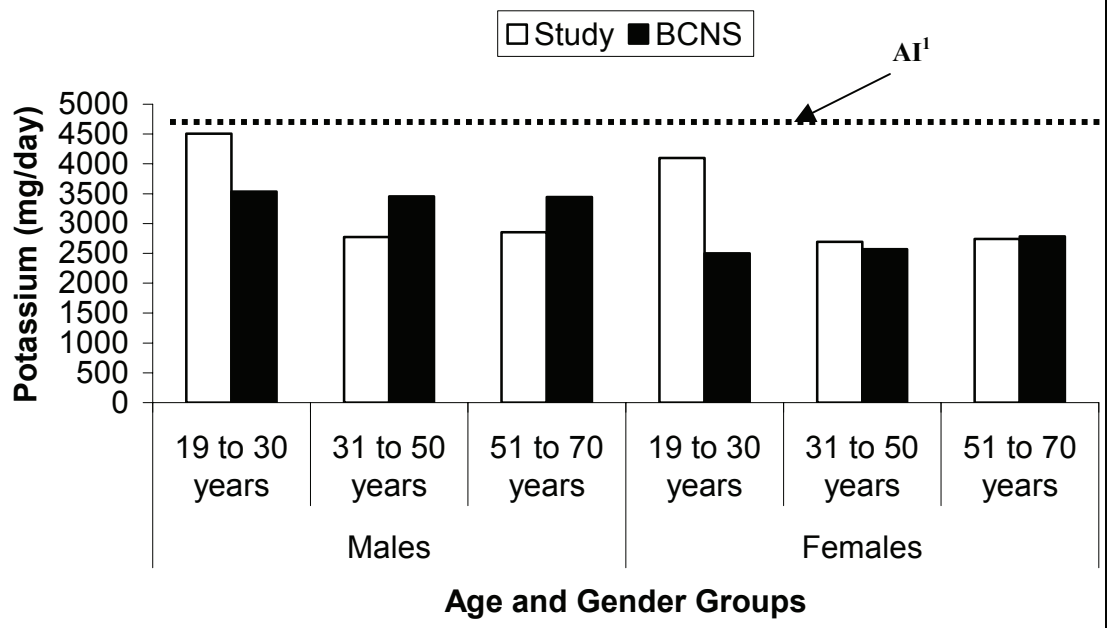
[1]EAR = Estimated Average Requirement (males = 9.4 mg/day and females = 6.8 mg/day)
*Significant difference between study and BCNS for food and supplement intakes at $p < 0.0001$

4.10 Electrolytes

4.10.1 Potassium

Median intakes for potassium were well below the AI in this study and the BCNS, especially for females over 50 years (Figure 4.0.24). A higher prevalence of inadequate intakes was evident for those aged 19 to 30 years but there were no significant differences between the study and BCNS groups or among the study groups [F (5, 91) = 2.2, ns] (Figure C.0.21). However, differences between males aged 19 to 30 years and females over 50 years approached significance (p = 0.054). Dietary deficiency of potassium is uncommon in normal diets because this mineral is widely distributed in food, but people who rely heavily on processed foods without fresh fruits and vegetables, or are on a very low-calorie diet with limited food choices, risk deficiency.

Figure 4.0.24: Median potassium intakes from food by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

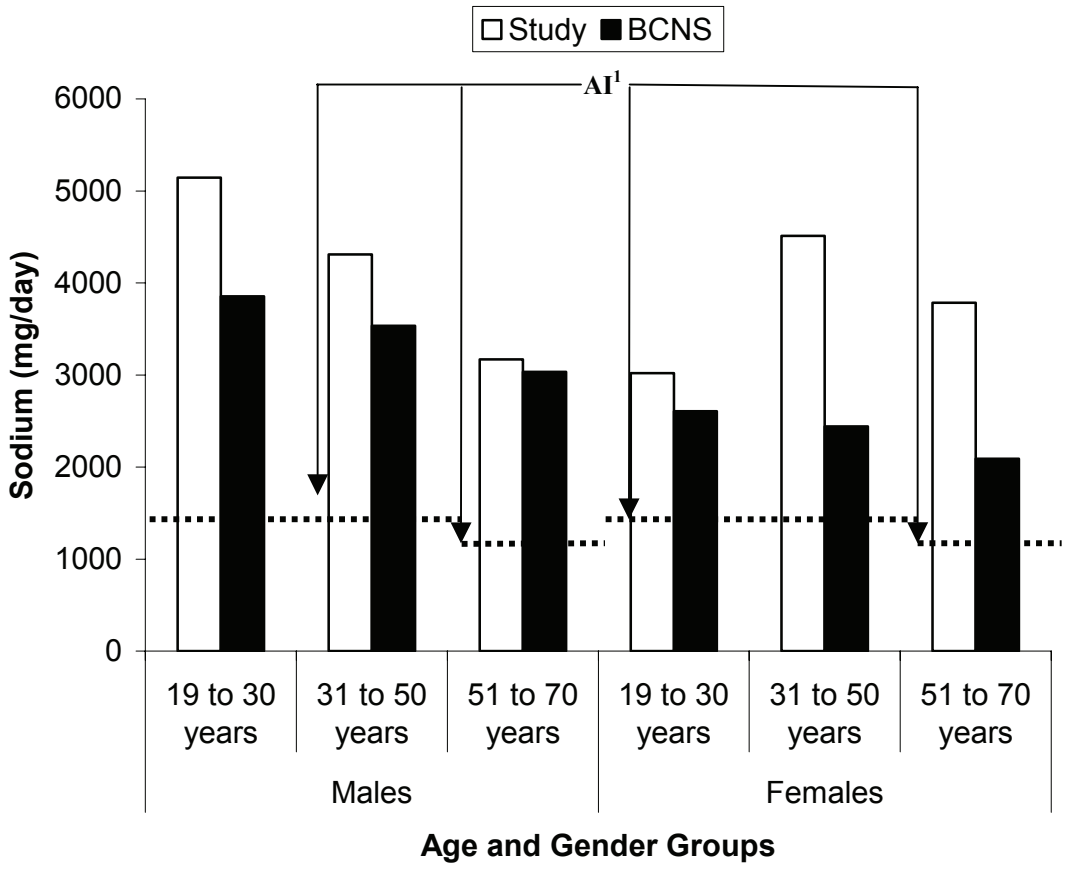


[1]AI = Adequate Intakes (4700 mg/day)

4.10.2 Sodium

The main dietary source of sodium was common salt (sodium chloride). Although the requirement for sodium has not been well established, estimates of sodium requirements for humans have been as low as 115 mg per day for sedentary adults living in a temperate climate. As the results suggest (Figure 4.0.25 and Figure C.0.21), this level was exceeded substantially. The AI for sodium is 1.5 g/day (1500 mg/day) for adults 19 to 30 years; 1.3 g/day (1300 mg/day) for those 51 to 70 years. The median intakes exceeded this amount in all groups and were consistently higher than the BCNS; there were no significant differences. There were also no differences among the study groups [F (5, 91) = 0.33, ns].

Figure 4.0.25: Median sodium intakes from food by age and gender and compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)



[1]AI = Adequate Intakes (males and females 19 to 50 years = 1500 mg/day; over 50 years = 1300 mg/day)

4.11 Other Minerals

Other essential minerals include chromium, copper, iodine, manganese, molybdenum, and selenium. However, the data for these in the nutrient database were incomplete (i.e., 50% coverage or less). Table 4.0.8 shows intakes from food and supplement sources of nutrients that have a UL to give a conservative estimate of the proportion of the sample that exceeded these. As the results indicate, only manganese intakes for women aged 30 to 50 years exceeded the UL for 8% of that group. For manganese, there was also one subject in each of the youngest male and female groups who were close to the UL (intakes between 10 to 11 mg).

Table 4.0.8: Intakes from food and supplements of copper, iodine, manganese, molybdenum, and selenium by age and gender

Nutrient	Gender	Age (yrs)	N	Mean ± SD[1]	50th percentile (25th; 75th)	UL[2]	% > UL[2]
Copper (mcg)	Males	19-30	5	2328 ± 692	2400 (1700; 2900)	10,000	0
		31-50	16	1980 ± 1426	2355 (1730; 2925)		0
		51-70	7	1727 ± 1157	1460 (960; 1770)		0
	Females	19-30	9	1619 ± 852	1305 (1060; 2180)		0
		31-50	39	2071 ± 1308	1890 (990; 2835)		0
		51-70	21	1920 ± 1468	1545 (680; 2400)		0
Iodine (mcg)	Males	19-30	5	33.8 ± 25.2	23.3 (18.5; 49.1)	1100	0
		31-50	16	34.2 ± 56.9	15.2 (0.2; 18)		0
		51-70	7	32.5 ± 49.6	6.2 (4.7; 39.6)		0
	Females	19-30	9	2.1 ± 1.8	2.2 (0.2; 3.4)		0
		31-50	39	33.6 ± 48.1	8.8 (3.3; 40.3)		0
		51-70	21	22.9 ± 40.3	14.2 (4.4; 20.8)		0

[1]SD = Standard Deviation
[2]Tolerable Upper Intake Levels

Table 4.0.8: Intakes from food and supplements of copper, iodine, manganese, molybdenum, and selenium by age and gender /continued

Nutrient	Gender	Age (yrs)	N	Mean ± SD[1]	50th percentile (25th; 75th)	UL[2]	% > UL[2]
Man-ganese (mg)	Males	19-30	5	6.5 ± 4.9	6.8 (2.3; 10.7)	11	0
		31-50	16	3.3 ± 1.6	2.3 (2.0; 5.1)		0
		51-70	7	3.7 ± 1.5	4.2 (2.6; 4.8)		0
	Females	19-30	9	4.3 ± 2.7	2.9 (2.7; 5.1)		0
		31-50	39	4.0 ± 3.3	3.2 (1.7; 5.1)		8
		51-70	21	3.7 ± 2.1	3.4 (2.1; 4.5)		0
Molyb-denum (mcg)	Males	19-30	5	14.3 ± 15.9	6.8 (5.8; 22.8)	2000	0
		31-50	16	17.3 ± 28.4	5.9 (3.3; 7.9)		0
		51-70	7	3.9 ± 3.3	1.9 (1.0; 7.0)		0
	Females	19-30	9	2.9 ± 3.5	1.5 (0.2; 4.5)		0
		31-50	39	10.0 ± 17.8	3.9 (1.6; 10.0)		0
		51-70	21	14.8 ± 22.3	6.6 (1.6; 19.1)		0
Selenium (mcg)	Males	19-30	5	123.5 ± 84.9	101.7 (65.0; 182.0)	400	0
		31-50	16	86.2 ± 32.5	80.8 (59.0; 118.0)		0
		51-70	7	98.9 ± 69.0	73.2 (53.9; 154.9)		0
	Females	19-30	9	64.0 ± 16.9	67.5 (59.7; 73.1)		0
		31-50	39	85.3 ± 64.4	68.2 (36.7; 115.1)		0
		51-70	21	66.3 ± 42.4	59.8 (29.6; 94.8)		0

[1]SD = Standard Deviation
[2]Tolerable Upper Intake Levels

4.12 Biochemical Indicators

Because the levels of a nutrient in blood or tissues can also be affected by genetic influences, lifestyle factors such as smoking or physical activity, or the intake of other nutrients, it is useful to examine biochemical indicators of dietary intake. As previously indicated, selected nutrients were evaluated in a subsample of the study participants (Tables 4.0.9 and 4.0.10). While 50 samples were obtained, two could not be used due to reasons cited previously regarding their ineligibility. In addition, the laboratory could not draw folate samples for all participants.

Table 4.0.9: Values of biochemical indicators for males

	Normal range	n	Mean ± SD[1]	25th	50th	75th	% Within range	% Below normal range	% Above normal range
Males									
Serum Folate (nmol/L)	> 12	7	--	--	--	--	100	0	--
RBC Folate (nmol/L)	630-1790	7	779 ± 152	657	730	843	86	14	0
Vitamin B_{12} (pmol/L)	154-530	18	501 ± 348	251	374	742	73	0	27
Ferritin (mcg/L)	15-370	18	163 ± 154	63	93	243	93	0	7
Vitamin E – A-Tocopherol (µmol/L)	13-24	18	30 ± 7	23	30	35	27	0	73
Albumin (g/L)	35-50	18	46 ± 4	42	46	49	93	0	7
Cholesterol (mmol/L)	2.0-5.2	18	5 ± 1	4.5	5	5.5	73	0	27 (>5.2) 20 (> 6.2)

[1]SD = Standard Deviation

[*]Probability of symptomatic deficiency: < 75 pmol/L = high; 75-150 pmol/L = moderate; 150-220 pmol/L = low; > 220 pmol/L = rare

Table 4.0.10: Values of biochemical indicators for females

	Normal range	n	Mean ± SD[1]	25th	50th	75th	% Within range	% Below normal range	% Above normal range
Females									
Serum folate (nmol/L)	> 12	7	--	--	--	--	100	0	--
RBC Folate (nmol/L)	630-1790	20	1036 ± 277	861	984	1114	100	0	--
Vitamin B_{12} (pmol/L)	154-530*	30	402 ± 221	255	321	527	71	4 (75-150)*	25
Ferritin (mcg/L)	15-200	30	59 ± 34	37	52	79	93	7	0
Vitamin E – A-Tocopherol (µmol/L)	13-24	30	32 ± 10	25	31	35	14	0	86
Albumin (g/L)	35-50	30	44 ± 3	43	44	46	100	0	0
Cholesterol (mmol/L)	2.0-5.2	30	5 ± 1	4.6	5.4	6.2	39	0	61 (>5.2) 25 (>6.2)

[1]SD = Standard Deviation

*Probability of symptomatic deficiency: < 75 pmol/L = high; 75-150 pmol/L = moderate; 150-220 pmol/L = low; > 220 pmol/L = rare

Low levels of RBC folate were a concern for one male. For females, a small proportion had low levels of Vitamin B_{12} (n = 1) and ferritin (n = 2). The high prevalence of values above the normal range was also surprising, especially for vitamin B_{12} and vitamin E (even after controlling for cholesterol). For males, one participant was above the normal ranges for ferritin and albumin. It is important to note that those who provided blood samples volunteered for this. Therefore, blood levels of nutrients may not accurately reflect that of the general mood disorder population.

4.13 Chapter Summary

In this chapter, estimates of usual food and nutrient intakes were reported and compared with the BCNS. Data for nutrient intakes based on food and supplemental sources where also presented. Finally, bloodwork data of a subsample were outlined. Usual median intakes for food energy appeared to be under-reported for certain subgroups. As a result of slightly under-reported energy intakes in some groups, nutrient intakes may also have been slightly under-reported.

Most participants were within the Acceptable Macronutrient Distribution Ranges for carbohydrates, total fat, and protein; however, about half the sample participants were consuming more than 35% of their energy from fat. This is considerably higher than the general population estimates of one-quarter. Many had inadequate intakes of fibre, α-linolenic and linoleic acid, thiamin, riboflavin, niacin, folate, vitamin B_{12}, vitamin B_6, pantothenic acid, vitamin C, calcium, magnesium, potassium, iron, phosphorous, and zinc, identifying these nutrients as potential health concern. Intake of nutritional supplements contributed to decreasing the proportion of inadequacy for several nutrients. Conversely, it also contributed to excessive levels of nutrients for some when compared to the ULs. Concern for nutrient excesses included folate, niacin, vitamin B_6, vitamin C, vitamin D, vitamin E, calcium, iron, magnesium, zinc, and manganese. Sodium intakes from diet sources alone also far exceeded recommended upper levels.

In addition to nutrient intakes, the biochemical levels of specific nutrients were measured in a subsample of participants. Surprisingly, for a small proportion of males, low levels of RBC folate were a concern. For a small proportion of females, low levels of Vitamin B_{12} and ferritin were also a concern. A substantial proportion of males and females had levels that exceeded the normal ranges for vitamin B_{12} and vitamin E (even

after controlling for cholesterol). For males, there were also some who were above the normal ranges for ferritin and albumin.

The following chapter presents the results outlining the various determinants of food selection. The subsequent chapter compares nutrients among types of mood disorder symptomatology and predictors of food intake.

CHAPTER FIVE: DETERMINANTS OF FOOD INTAKE

This chapter examined if the sample's nutrient intake levels differed significantly according to sociodemographic, mental health status and health-related variables.

5.1 Sociodemographic Factors

Chapter Four examined the influences of age and gender on nutrient intakes. The effects of education, income, marital status (see Table 5.0.1), and food insecurity on nutrient intakes are detailed in this section. For education level, significant differences were found only for pantothenic acid intakes [$F (3, 93) = 5.54$, $p < 0.05$] and the health implications of this result are unclear. Other nutrient intakes did not appear to vary by education level, which suggests that this difference did not bias the results.

As income levels increased significantly higher intakes for protein [$F (3, 93) = 4.40$, $p < 0.05$], fibre [$F (3, 93) = 3.07$, $p < 0.05$]), thiamin [$F (3, 93) = 6.41$, $p < 0.001$], niacin [$F (3, 93) = 5.01$, $p < 0.05$], vitamin B_6 [$F (3, 93) = 3.02$, $p < 0.05$], iron [$F (3, 93) = 4.36$, $p < 0.05$], sodium [$F (3, 93) = 4.30$, $p < 0.05$], and zinc [$F (3, 93) = 3.01$, $p < 0.05$] emerged. Food security, a variable associated with income, was also examined. Those who indicated they were food insecure had significantly lower intakes for protein, vitamin C, and sodium ($p < 0.05$) and higher intakes for polyunsaturated fat ($p < 0.05$) than those who indicated food security.

For marital status, intakes for energy ($t = -2.19$, SE = 195.03, 95% CI = -815.01 to -40.63, $p < 0.05$), fibre ($t = -2.40$, SE = 2.87, 95% CI = -12.60 to -1.19, $p < 0.05$), magnesium ($t = -2.58$, SE = 29.67, 95% CI = -135.29 to -17.51, $p < 0.05$), phosphorous ($t = -2.51$, SE = 96.83, 95% CI = -435.51 to -51.04, $p < 0.05$), potassium ($t = -2.37$, SE = 255.51, 95% CI = 1112.03 to -97.54, $p < 0.05$) consumed were significantly lower for those who were considered single (i.e., widowed, divorced, separated, or never married).

5.2 Nutrition Factors

Table 5.0.2 presents data on selection of foods based on some health or food-related concern. The main health concerns affecting food selection were body weight, heart disease, cancer, and osteoporosis. In addition, most participants were concerned about fibre and fat content. Other nutrition factors included antioxidants (15%), B vitamins (4%), trace minerals (2%), vitamin D (3%), essential fatty acids (6%), protein (2%), as well as detoxifying (2%) and organic foods (2%).

Table 5.0.1: Education, income, marital status, and intakes of selected nutrients

Nutrient	Education level[1]		Income[2]		Marital status[3]
	F value[4]	P value	F value[4]	P value	t value (SE[5], 95% CI[6], p-value)
Calories (kcal)	0.99	0.402	1.40	0.247	**-2.194 (SE = 195.033, 95% CI = -815.007 to -40.629, p = 0.031)**
Protein (g)	1.64	0.186	**4.40**	**0.006**	-0.780 (SE = 6.410, 95% CI = -17.839 to 7.610, p = 0.427)
Carbohydrates (g)	0.85	0.471	1.24	0.301	-1.666 (SE = 28.691, 95% CI = -104.749 to 9.169, p = 0.099)
Fibre (g)	0.88	0.454	**3.07**	**0.032**	**-2.400 (SE = 2.873, 95% CI = -12.598 to -1.191, p = 0.018)**
Fat (g)	0.32	0.810	1.01	0.391	-1.8178 (SE = 10.469, 95% CI = -39.815 to 1.753, p = 0.072)
- Omega 3 fats (g)	0.27	0.849	0.18	0.913	-1.015 (SE = 0.117, 95% CI = -0.351 to 0.114, p = 0.313)
- Omega 6 fats (g)	1.59	0.196	1.75	0.163	-1.364 (SE = 1.052, 95% CI = -3.523 to 0.655, p = 0.176)
Cholesterol (mg)	0.10	0.958	1.53	0.211	0.256 (SE = 47.140, 95% CI = -81.495 to 105.671, p = 0.798)
Thiamin (mg)	2.07	0.110	**6.41**	**0.001**	-1.169 (SE = 0.186, 95% CI = -0.586 to 0.152, p = 0.245)
Riboflavin (mg)	1.47	0.229	2.53	0.062	-0.668 (SE = 0.175, 95% CI = -0.464 to 0.230, p = 0.506)
Niacin (mg)	1.52	0.2143	**5.01**	**0.0029**	0.093 (SE = 1.921, 95% CI = -3.634 to 3.992, p = 0.926)
Vitamin B_6 (mg)	1.59	0.1969	**3.02**	**0.0339**	-1.628 (SE = 0.204, 95% CI = -0.738 to 0.073, p = 0.107)

Bolded cells represent significant results

[1]Groups subdivided as 1) less than high school; 2) high school graduate; 3) some post-secondary but below bachelor's level; and 4) bachelor's degree or above

[2]Groups subdivided as 1) Income below $20,000; 2) Income between $20,000 and $35,000; 3) Income between $35,000 and $55,000; and 4) Income over $55,000

[3]Groups subdivided as 1) Married or common-law; and 2) Single (includes widowed, divorced, separated, never married)

[4]Degrees of freedom of 4, 93

[5]Standard error

[6]Confidence intervals

Table 5.0.1: Education, income, marital status, and intakes of selected nutrient /continued					
Nutrient	**Education level[1]**		**Income[2]**		**Marital status[3]**
	F value[4]	**P value**	**F value[4]**	**P value**	**t value (SE[5], 95% CI[6], p-value)**
Vitamin B_{12} (mcg)	0.38	0.7664	1.21	0.3102	-1.197 (SE = 0.761, 95% CI = -2.423 to 0.600, p = 0.234)
Vitamin C (mg)	2.32	0.0807	1.38	0.2525	0.031 (SE = 24.457, 95% CI = -47.802 to 49.305, p = 0.976)
Folate (mcg)	0.34	0.7971	2.55	0.0608	0.260 (SE = 47.963, 95% CI = -82.751 to 107.688, p = 0.796)
Pantothenic acid (mg)	5.54	0.0015	1.42	0.2419	-1.951 (SE = 0.418, 95% CI = -1.646 to 0.014, p = 0.054)
Calcium (mg)	0.18	0.9093	1.93	0.1306	-1.603 (SE = 99.735, 95% CI = -357.895 to 38.104, p = 0.112)
Iron (milligrams)	1.24	0.3008	**4.36**	**0.0064**	-0.902 (SE = 1.881, 95% CI = -5.431 to 2.037, p = 0.369)
Magnesium (mg)	0.40	0.7500	2.20	0.0934	**-2.576 (SE = 29.663, 95% CI = -135.288 to -17.512, p = 0.012)**
Zinc (mg)	0.60	0.6147	**3.01**	**0.0342**	-1.458 (SE = 1.289, 95% CI = -4.439 to 0.680, p = 0.148)
Phosphorous (mg)	1.91	0.1337	2.23	0.0898	**-2.512 (SE = 96.832, 95% CI = -435.512 to -51.042, p = 0.014)**
Potassium (mg)	0.94	0.4236	1.55	0.2068	**-2.367 (SE = 255.507, 95% CI = -1112.028 to -97.536, p = 0.020)**
Sodium (mg)	0.53	0.6604	**4.30**	**0.0069**	-0.174 (SE = 884.202, 95% CI = -1909.489 to 1601.236, p = 0.8620)

Bolded cells represent significant results

[1]Groups subdivided as 1) less than high school; 2) high school graduate; 3) some post-secondary but below bachelor's level; and 4) bachelor's degree or above

[2]Groups subdivided as 1) Income below $20,000; 2) Income $20,000 to $35,000; 3) Income $35,000 to $55,000; and 4) Income over $55,000

[3]Groups subdivided as 1) Married or common-law; and 2) Single (includes widowed, divorced, separated, never married)

[4]Degrees of freedom of 4, 93

[5]Standard error

[6]Confidence intervals

Table 5.0.2: Food selection and concern about health conditions and specific nutrition components in foods	
	n (%)
Concern (n=97)	
Body weight	71 (73%)
Heart disease and/or high blood pressure	47 (48%)
Cancer	40 (41%)
Osteoporosis	40 (41%)
Diabetes	33 (34%)
Food allergy or intolerance	30 (31%)
Nutrition components (n=97)	
The amount and type of fat content	63 (65%)
The fibre content	63 (65%)
The calcium content	48 (49%)
The iron content	32 (33%)
Other vitamins, minerals, or components they contain	35 (36%)

Participants were asked about selecting foods to avoid specific nutrition components. Most reported avoiding certain foods because of fat, sugar, or saturated fat content; some avoided, for example, red meat (2%), caffeine (92%), colours, additives, preservatives, or sweeteners (5%), high glycemic index foods (2%), dairy products (2%), and alcohol (1%). A fraction of participants (7%) reported being vegetarian and none were vegan.

5.3 Health-Related Conditions

Table 5.0.3 outlines health conditions reported by the participants. Previous mental health diagnoses included anxiety disorder (13%), panic disorder (5%), seasonal affective disorder (4%), post-traumatic stress disorder (4%), obsessive-compulsive disorder (3%), bulimia (2%), borderline personality disorder (2%), attention deficit hyperactivity disorder (1%), and social disorder (1%). No significant nutrient intake differences were found among the sample when all physical health conditions were compared between

those who did and did not indicate they had that condition. Only 19% of participants indicated that they were following any special diet based on their health condition. When nutrient intakes were analyzed, it was found that those following a special diet had significantly lower intakes of carbohydrates (255 ± 103) than those not following a special diet (341 ± 145) ($t = 2.40$, $p < 0.05$, 95% CI 14.775 to 156.066).

Table 5.0.3: Other medical conditions (present or past)	
Condition	**n (%)**
Previous mental health diagnoses	89 (92%)
Heart disease or stroke, high cholesterol, or high blood pressure (excludes during pregnancy)	43 (44%)
Osteoporosis	14 (14%)
Diabetes (excludes gestational diabetes)	12 (12%)
Cancer	12 (12%)

5.4 Nutrition Education

When questioned about the *Eating Well with Canada's Food Guide*, 92% (n = 89) of participants claimed to have heard of it, but fewer than one-third (n = 30; 31%) actually used it. Participants indicated that they used it as a tool for planning meals (n = 22; 23%), assessing fruit and vegetable intake, and managing portion control (n = 10; 10%). Those who indicated they use the Food Guide had significantly higher intakes of pantothenic acid ($p < 0.05$) than those who indicated they didn't use the Food Guide.

5.5 Attitudes about Eating and the Body

Respondents were also asked about their attitudes to eating and their body (see Food Selection Questionnaire in Appendix B). For example, Question 16 asked, "How comfortable do you feel about your body when you see yourself in a mirror?"

5.5.1 Bodily Comfort and Gender

The proportion of respondents who reported being very uncomfortable ($p < 0.0001$) or neutral ($p < 0.05$) about their bodies was higher than in the BCNS (Table 5.0.4). The proportions that were somewhat comfortable ($p < 0.001$) or very comfortable ($p < 0.05$)

were lower than the BCNS. More females were very uncomfortable with their body than the BCNS ($p < 0.001$). When age was considered, participants aged 19 to 30 years ($p < 0.05$) and those 31 to 50 ($p < 0.001$) and 51 to 70 years ($p < 0.05$) who answered "neutral" numbered higher compared to the BCNS. No differences were found among subgroups ($\chi^2 = 17.32$, $p = 0.632$). When this analysis was conducted for men and women separately, there were no differences (data not shown).

Table 5.0.4: Comparison of gender groups between the study and the British Columbia Nutrition Survey (BCNS) sample based on level of bodily comfort[1]

Level of bodily comfort	Study sample (n=89); n (%)			BCNS (n=1821); n (%)		
	Total	Men (n=20)	Women (n=69)	Total	Men (n=867)	Women (n=954)
1 – very uncomfortable	20 (22)***	2 (10)	17 (25)***	109 (6)	35 (4)	86 (9)
2 – somewhat uncomfortable	20 (22)	6 (30)	17 (25)	382 (21)	130 (15)	258 (27)
3 – neutral	33 (37)*	5 (25)	18 (26)	419 (23)	208 (24)	210 (22)
4 – somewhat comfortable	12 (13)**	5 (25)	14 (20)	583 (32)	303 (35)	267 (28)
5 – very comfortable	5 (6)*	2 (10)	3 (4)*	328 (18)	191 (22)	134 (14)

[1]Respondents were asked, "How comfortable do you feel about your body when you see yourself in a mirror?"
*Significant differences between the study sample and BCNS $p < 0.05$
**Significant differences between the study sample and BCNS $p < 0.0001$
***Significant differences between the study sample and BCNS $p < 0.00001$

5.5.2 Eating Attitude and Behaviour According to Gender

Women were more likely than men to agree with statements reflecting cognitive dietary restraint or disinhibition (range of $p < 0.05$ to $p < 0.0001$) (Table 5.0.5). Compared to the BCNS, the sample were more likely to usually ($p < 0.01$) or always ($p < 0.05$) restrict their food intake in a conscious effort to control weight, go on eating binges sometimes or at least weekly ($p < 0.0001$), and were moderately or very likely ($p < 0.0001$) to consciously eat less than they wanted (Table 5.0.6). There were significant differences found for the question asking about eating binges and total fat intake [$F (3, 85) = 3.86$, $p < 0.05$] with those never going on binges having significantly lower total fat (85 ± 45) than those who binge at least weekly (140 ± 53) ($p < 0.05$). For the binge eating

question, significant differences were also found for cholesterol intake [$F(3, 85) = 3.53$, $p < 0.05$] with those never going on binges having significantly lower intakes (225.85 ± 160.03) than those who rarely went on eating binges (442.08 ± 215.68) ($p < 0.05$).

Table 5.0.5: Comparison between the study and British Columbia Nutrition Survey (BCNS) sample for those agreeing with eating attitude and behaviour statements

Attitude/ behaviour statement	Study sample (n=89); n (%)			BCNS (n=1821); n (%)		
	Total	Men (n=28)	Women (n=69)	Total	Men (n=867)	Women (n=954)
I do not eat some foods because they make me fat[R]	51 (53)*	11 (55)	42 (61)*	728 (40)	312 (36)	420 (44)
When I feel 'down' or sad, I often overeat[D]	45 (46)***	14 (70)***+	31 (45)*	364 (20)	104 (12)	267 (28)
I deliberately take small helpings as a means of controlling my weight[R]	49 (51)***	6 (30)	32 (46)	528 (29)	191 (22)	334 (35)
Sometimes when I start eating, I just can't seem to stop[D]	49 (51)***	7 (35)	34 (49)***	401 (22)	165 (19)	191 (24)
When I feel lonely, I console myself by eating[D]	49 (51)***	9 (45)***	31 (45)***	237 (13)	43 (5)	200 (21)

[R]Statement reflects cognitive dietary restraint and [D]Statement reflects disinhibition (Stunkard and Messick, 1985[288])
Significant differences between study sample and BCNS at: *$p < 0.05$, **$p < 0.001$ or ***$p < 0.0001$
+Significant differences between men and women in the study sample $p < 0.05$

Table 5.0.6: Comparison between the study and the British Columbia Nutrition Survey (BCNS) sample by responses to eating attitude and behaviour statements

Attitude/ behaviour statement	Study sample (n=89); n (%)			BCNS (n=1823); n (%)		
	Total	Men (n=28)	Women (n=69)	Total	Men (n=868)	Women (n=955)
How often are you restricting your food intake in a conscious effort to control your weight?[R]						
Rarely	27 (28)***	8 (27)*	19 (28)*	930 (51)	503 (58)	411 (43)
Sometimes	30 (31)	6 (23)	23 (33)	529 (29)	208 (24)	324 (34)
Usually	27 (28)**	10 (34)**	19 (28)*	273 (15)	113 (13)	162 (17)
Always	13 (13)*	4 (16)*	8 (12)	109 (6)	43 (5)	57 (6)
Do you go on eating binges* even though you are not hungry?[D]						
Never	34 (35)***	9 (32) ***	25 (36)***	1385 (76)	712 (82)	678 (71)
Rarely	14 (14)	7 (25)	8 (12)	255 (14)	113 (13)	153 (16)
Sometimes	35 (36)***	7 (25)***	29 (42)***	146 (8)	43 (5)	105 (11)
At least weekly	15 (15)***	5 (18)***	7 (10)***	18 (1)	9 (1)	19 (2)
How likely are you to consciously eat less than you want?[R]						
Unlikely	28 (29)***	9 (32)*	21 (30)**	984 (54)	521 (60)	468 (49)
Slightly likely	17 (18)	6 (21)	9 (13)*	456 (25)	191 (22)	267 (28)
Moderately	30 (31)***	8 (27)*	31 (45)***	292 (16)	113 (13)	181 (19)
Very likely	18 (19)***	5 (18)*	8 (12)*	91 (5)	43 (5)	38 (4)

[R]Statement reflects cognitive dietary restraint and [D]disinhibition (Stunkard and Messick, 1985[288])
*Binge eating means eating a very large amount of food in a very short period of time, and feeling that you can't control how much you're eating
Significant differences between study sample and BCNS at: $^{*}p < 0.05$, $^{**}p < 0.001$ or $^{***}p < 0.0001$

Compared to the BCNS, women were more likely to usually ($p < 0.05$) restrict food intake in a conscious effort to control weight, that they sometimes ($p < 0.0001$) or at least weekly ($p < 0.001$) go on eating binges even though they are not hungry, and were moderately ($p < 0.0001$) or very likely ($p < 0.05$) to consciously eat less than they wanted. More males usually ($p < 0.001$) or always ($p < 0.05$) restricted their food intake in a conscious effort to control weight, were more likely to sometimes or at least weekly

($p < 0.0001$) go on eating binges even though they were not hungry, and were moderately ($p < 0.0001$) or very ($p < 0.05$) likely to consciously eat less than they wanted.

Scores for cognitive dietary restraint and disinhibition had high values for skewness (restraint skewness = 0.865 ± 0.051 SE; disinhibition skewness = 1.534 ± 0.050 SE). The square root transformation was applied but the distributions remained skewed (square root of restraint skewness = 0.162 ± 0.048 SE; square root of disinhibition skewness = 0.846 ± 0.052 SE). Thus, nonparametric analyses were conducted. Table 5.0.7 shows mean restraint scores according to gender for three *DRI*–based age groups. Females in the study 19 to 30 and 51 to 70 years had significantly lower scores than in the BCNS. Compared to the BCNS, all groups had higher disinhibition scores ($p < 0.0001$, except for women aged between 51 and 70 years, which was $p < 0.001$). Comparisons of restraint and disinhibition scores showed no significant differences according to psychiatric diagnosis. Comparisons by symptom scores showed significant association of increased symptoms with higher restraint scores only for the Ham-D scores (χ^2 (2 df) = 7.136, $p < 0.05$) and disinhibition scores (χ^2 (2 df) = 8.300, $p < 0.05$).

Table 5.0.7: Mean restraint scores[1] of men, women, and both sexes combined by age[1] and compared to the British Columbia Nutrition Survey (BCNS)

Age group (years)	Study sample (n=89); Mean ± SD[2]			BCNS (n=1320); Mean ± SD[2]		
	Total	Men (n=20)	Women (n=69)	Total	Men (n=596)	Women (n=724)
19-30	0.917 ± 0.793	1.333 ± 0.577	0.778 ± 0.833*	1.240 ± 1.606	1.240 ± 1.606	1.421 ± 1.623
31-50	1.449 ± 0.614	1.214 ± 0.426	1.543 ± 0.657	1.377 ± 1.532	1.377 ± 1.532	1.655 ± 1.666
51-70	1.380 ± 0.724	1.556 ± 0.726	1.261 ± 0.752*	1.421 ± 1.645	1.542 ± 1.685	1.718 ± 1.752
All ages	1.358 ± 0.694	1.346 ± 0.562	1.362 ± 0.739	1.337 ± 1.385	1.234 ± 1.283	1.467 ± 1.587

[1]Restraint scores reflect the perception that food intake is consciously limited in an attempt to control body weight
[2]Standard Deviation
Scores are based on five items taken from the restraint subscale of the Three-Factor Eating Questionnaire[288]
*Significant differences between the study sample and BCNS at $p < 0.05$

Table 5.0.8: Mean disinhibition scores[1] of men, women, and both sexes combined by age[1] and compared to the British Columbia Nutrition Survey (BCNS)

Age group (years)	Study sample (n=89); Mean ± SD[2]			BCNS (n=1320); Mean ± SD[2]		
	Total	Men (n=20)	Women (n=69)	Total	Men (n=596)	Women (n=724)
19-30	2.25 ± 0.965***	2.000 ± 1.732***	2.333 ± 0.707***	0.634 ± 1.023	0.402 ± 0.762	0.907 ± 1.209
31-50	1.959 ± 1.136***	2.143 ± 1.167***	1.886 ± 1.132***	0.760 ± 1.164	0.538 ± 0.943	0.975 ± 1.310
51-70	1.596 ± 1.209***	1.667 ± 1.414***	1.435 ± 1.199**	0.544 ± 1.004	0.244 ± 0.528	0.816 ± 1.231
All ages	1.858 ± 1.156***	1.962 ± 1.280***	1.820 ± 1.116***	0.534 ± 1.036	0.409 ± 0.743	0.763 ± 1.045

[1]Disinhibition scores reflect the perception that control over food intake can be lost in response to certain situations, leading to overeating. Scores are based on four items taken from the disinhibition subscale of the Three-Factor Eating Questionnaire[288]
[2]Standard Deviation
Significant differences between the study sample and BCNS at $^{**}p < 0.001$ or $^{***}p < 0.0001$

5.6 Body Mass Index, Physical Activity, Bodily Comfort, Psychiatric Factors and Eating Behaviour

Relationships among BMI, bodily comfort, psychiatric factors and eating behaviour were examined. Data reported in the BCNS indicated that BMI was associated to some extent with physical activity level. For this reason, associations between BMI and body comfort level were examined with exercise as a co-variate.

5.6.1 Bodily Comfort Level, BMI, and Activity

Those who were less comfortable with their bodies tended to have higher BMIs but there were no significant differences among the groups (Table 5.0.9). Females that were very uncomfortable ($p < 0.05$), neutral ($p < 0.05$), somewhat comfortable ($p < 0.001$), and very comfortable ($p < 0.0001$) had higher BMIs compared to the BCNS. Males who were somewhat uncomfortable ($p < 0.001$), somewhat comfortable ($p < 0 .05$), and very comfortable ($p < 0.05$) had higher BMIs compared to the BCNS. Regardless of bodily comfort level, participants tended to have higher BMIs compared to the BCNS.

Table 5.0.9: Comparison of body mass index (BMI) by level of bodily comfort[1] by gender between the study and British Columbia Nutrition Survey (BCNS)

Level of bodily comfort[1]	Body mass index (kg/m^2)					
	Study sample (n=89); Mean ± SD[2]			BCNS (n=1821); Mean ± SD[2]		
	Total	Men (n=29)	Women (n=60)	Total	Men (n=867)	Women (n=954)
1 – very uncomfortable	31.6 ± 9.1*	28.6 ± 5.0	34.0 ± 11.1***	29.3 ± 7.9	29.6 ± 8.2	29.2 ± 7.8
2 – somewhat uncomfortable	28.7 ± 6.2	32.6 ± 8.2***	27.6 ± 5.4	28.2 ± 6.5	29.0 ± 5.4	27.7 ± 7.0
3 - neutral	27.3 ± 4.0*	27.2 ± 3.5	27.4 ± 4.2*	26.1 ± 5.4	26.6 ± 5.2	25.6 ± 5.6
4 – somewhat comfortable	26.9 ± 6.6**	27.9 ± 6.6*	26.2 ± 6.9*	25.2 ± 4.5	25.9 ± 3.9	24.2 ± 5.0
5 – very comfortable	28.7 ± 4.7***	29.3 ± 5.4*	28.4 ± 2.9***	25.1 ± 5.4	26.4 ± 5.6	23.2 ± 5.6

[1]Respondents were asked "How comfortable do you feel about your body when you see yourself in a mirror?"
[2]Standard Deviation
Significant differences between the study sample and BCNS at: $^{*}p < 0.05$, $^{**}p < 0.001$, or $^{***}p < 0.0001$

The association between body comfort level, BMI, and physical activity indicated that the average BMI for all comfort and activity levels were over 25, with the exception of inactive women who were very uncomfortable with their body, and both gender groups who were inactive and somewhat uncomfortable with their bodies. Direct comparisons could not be made with the BCNS as physical activity was measured differently.

5.6.2 Bodily Comfort Level, BMI, and Psychiatric Factors

BMI varied according to diagnosis (χ^2 (8 df) = 9.559, p = 0.2973) (Table 5.0.10). The proportion of those with bipolar disorder who were very uncomfortable with their body was lower than for those with depression ($p < 0.05$). Analysis of bodily comfort, BMI, and psychiatric symptoms and functioning using multiple linear regression with age and gender as covariates indicated no significant results (data not shown).

Table 5.0.10: Comparison of body mass index (BMI) by level of bodily comfort[1] according to psychiatric condition

Level of bodily comfort[1]	Bipolar Disorder (n=55)		Major Depressive Disorder (n=34)	
	n (%)	BMI (Mean ± SD[2])	n (%)	BMI (Mean ± SD[2])
1 – very uncomfortable	7 (12.7)*	33.1 ± 10.0	11 (32.4)	30.5 ± 8.9
2 – somewhat uncomfortable	11 (20.0)	27.0 ± 5.4	5 (14.7)	32.9 ± 6.7
3 - neutral	17 (30.9)	27.1 ± 4.0	13 (38.2)	27.6 ± 4.2
4 – somewhat comfortable	10 (18.2)	28.0 ± 7.0	5 (14.7)	24.9 ± 5.8
5 – very comfortable	10 (18.2)	29.5 ± 4.5	0	--

[1]Respondents were asked "How comfortable do you feel about your body when you see yourself in a mirror?"
[2]Standard Deviation
*Significant differences between diagnoses at $p < 0.05$

Additional analyses were done for bodily comfort and the various nutrients. There were no significant associations found between level of bodily comfort and intakes of the various nutrients investigated in Chapter Four.

5.7 Food Beliefs and Nutrition Knowledge

Eighty-eight percent of participants believed that what one eats and drinks has an effect on or can prevent major diseases. Most participants correctly identified what foods were high in fat; however, more than 15% of participants incorrectly thought that white bread and soda were high in fat (Table 5.0.11).

Table 5.0.11: Interpretation of food intake and knowledge of high fat and high fibre foods

	n (%)
Do you think your diet is:	
High in fibre	42 (43%)
Low in fibre	21 (22%)
High in fat	22 (23%)
Low in fat	41 (42%)
Don't know	13 (13%)

Table 5.0.11: Interpretation of food intake and knowledge of high fat and high fibre foods /continued	
	n (%)
Which of the following foods are high in fat:	
White bread	28 (29%)
Soda	18 (19%)
Broiled fish	5 (5%)
Bananas	3 (3%)
Cold cuts or ham	78 (80%)
Don't know	5 (5%)
Which of the following foods are high in fibre:	
Red meat	1 (1%)
Lettuce	38 (39%)
Carrots	63 (65%)
White rice	8 (8%)
Apples	69 (71%)

5.8 Chapter Summary

Several determinants of food intake were examined and, where feasible, comparisons were made with the BCNS. Many differences were found among nutrient intakes based on income (similar results for food insecurity) and marital status, suggesting that these factors may bias the results. These variables were therefore included in the multivariate analyses in the following chapter.

Most participants had concurrent medical conditions and this probably explains why many indicated that concerns about improving or maintaining health affected their food selection. However, beliefs about selecting foods based on fat, fibre, and sugar content were not reflected in the food group and nutrient data reported in Chapter Four nor did they significantly affect nutrient intakes. Those who indicated they followed a special diet had significantly lower intakes of carbohydrates while those who indicated they use the Food Guide had significantly lower pantothenic acid intakes.

Many significant differences in attitudes about eating and the body emerged between the study and the BCNS. The proportion of respondents who were very uncomfortable or neutral about their body was significantly higher than in the BCNS. Females who were

very uncomfortable with their body were significantly more numerous than in the BCNS. Frequency of binge eating significantly affected total fat and cholesterol intakes. Gender differences existed for cognitive restraint and disinhibition. A significantly higher proportion of females were more likely to agree with statements reflecting cognitive dietary restraint or disinhibition when compared to the BCNS. The proportion of men who usually or always restricted their food intake in a conscious effort to control their weight was significantly higher than in the BCNS. Finally, significantly more males were very likely to consciously eat less than they wanted compared to females. Females aged 19 to 30 years and all participants aged 51 to 70 years had significantly lower restraint scores than in the BCNS. For all groups, study participants had significantly higher disinhibition scores when compared to the BCNS.

Measures of bodily comfort, restraint, and disinhibition were also examined according to psychiatric factors and BMI. Hamilton Depression scores were significantly associated with restraint and disinhibition measures. Not surprisingly, associations between body comfort level and BMI revealed that those who were less comfortable with their bodies tended to have a higher mean BMI. For men, those who were somewhat uncomfortable, or were somewhat or very comfortable with their bodies also had significantly higher BMIs compared to the BCNS.

In the following chapter, the data from Chapters Three to Five will be analyzed using multivariate statistical procedures to help describe associations between nutrient levels and psychiatric symptoms and functioning.

CHAPTER SIX: RESULTS — NUTRIENT STATUS AND MOOD DISORDER SYMPTOMS

This chapter examined the following research queries: 1) What sociodemographic, mental health status and health-related variables are associated with energy and macronutrient intakes in individuals with mood disorders?; 2) What nutrients, sociodemographic, and health-related variables are associated with mental health status in individuals with mood disorders?; and 3) What nutrient intake, sociodemographic, mental health status and health-related variables are associated with blood levels of selected nutrients in individuals with mood disorders? Bivariate analyses and multiple regression models are analyzed to examine these questions.

6.1 Assessment of Nutrient Intakes from Food Sources and Mood Disorders

Bivariate statistical tests were conducted to examine the relationships between nutrient intakes and mood disorders. Correlation analyses between specific nutrients and symptom rating scales are presented. For all correlations, scatterplots were reviewed to verify the relationship.

6.1.1 Energy and Macronutrients from Food Sources

Bivariate relationships among food intakes of energy, the macronutrients, mood disorder subtypes, and mood symptoms are outlined (Table 6.0.1). Table 6.0.2 presents the correlations among selected macronutrients, mood symptoms, and Global Assessment of Functioning (GAF) scores. Based on the two-sample Wilcoxon rank-sum (Mann-Whitney) test, significant differences were found only when comparing those who had Young Mania Rating Scale scores below (< 20) or above (≥ 20) the cut-off values and energy intakes ($z = 2.152$, $p < 0.05$). There was a large median difference in caloric intakes for these subgroups. There were no significant correlations between Hamilton Depression Scale (Ham-D) and Young Mania Rating Scale (YMRS) scores and the macronutrients. One may have anticipated some differences for α-linolenic acid; however, as the figures suggest, the median intakes were almost identical. Weak, positive significant Spearman correlations (Table 6.0.2) were found for GAF scores and energy ($r = 0.2138$, $p < 0.05$), carbohydrates ($r = 0.2141$, $p < 0.05$), fibre ($r = 0.2575$, $p < 0.05$), total fat ($r = 0.2146$, $p < 0.05$), and linoleic acid ($r = 0.2146$, $p < 0.05$).

Table 6.0.1: Bivariate relationships between food intake of macronutrients, mood disorder subtype, and mood symptoms

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[3] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Energy (kcal)	2544 (1802; 3051)	2529 (1938; 3309)	z = -0.401, p = 0.689	**2521 (1938; 3051)**	**988 (651; 1326)**	**z = 2.152, p = 0.0314**	2367 (1918; 2998)	3021 (1670, 3213)	z = -1.040, p = 0.299
Carbohydrate (g)	324 (211; 407)	305 (245; 405)	z = -0.363, p = 0.717	305 (215; 405)	237 (155; 320)	z = 0.855, p = 0.392	304 (212; 403)	332 (215; 517)	z = -0.758, p = 0.448
• Fibre (g)	23 (17; 29)	23 (13; 34)	z = -0.462, p = 0.644	23 (15; 30)	13 (10; 17)	z = 1.530, p = 0.126	23 (15; 30)	24 (17; 29)	z = 0.022, p = 0.983
Protein (g)	96 (68; 112)	92 (65; 172)	z = -1.00, p = 0.316	94 (68; 109)	102 (67; 137)	z = -0.311, p = 0.756	93 (67; 109)	100 (68; 115)	z = -0.444, p = 0.657
Fat (g)	91 (64; 125)	90 (67; 141)	z = -0.279, p = 0.781	90 (64; 125)	74 (53; 95)	z = 0.622, p = 0.533	90 (65; 125)	87 (57; 121)	z = 0.087, p = 0.931
• Saturated (g)	28 (18; 40)	28 (20; 43)	z = -0.267, p = 0.789	28 (18; 38)	27 (24; 30)	z = 0.130, p = 0.897	28 (19; 39)	25 (18; 36)	z = 0.444, p = 0.657
• Polyunsaturated (g)	11 (7; 16)	11 (8; 17)	z = 0.565, p = 0.572	11 (7; 16)	5 (3; 7)	z = 1.729, p = 0.084	11 (7; 16)	12 (11; 14)	z = -0.991, p = 0.322

Bolded cells represent significant results
[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale

Table 6.0.1: Bivariate relationships between food intake of macronutrients, mood disorder subtype, and mood symptoms /continued

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[3] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Fat continued..									
• Monounsaturated (g)	23 (16; 29)	23 (18; 32)	z = -0.132, p = 0.895	22 (16; 29)	18 (13; 23)	z = 0.760, p = 0.447	22 (16; 28)	24 (18; 33)	z = -0.630, p = 0.529
• Linoleic acid (g)	3 (2; 4)	4 (2; 5)	z = 0.997, p = 0.319	3 (2; 5)	2 (0.1; 3)	z = 1.165, p = 0.244	3 (1; 5)	5 (3; 7)	z = -1.640, p = 0.101
• α-Linolenic acid (g)	0.3 (0.1; 0.5)	0.2 (0.1; 0.4)	z = -1.092, p = 0.275	0.3 (0.1; 0.4)	0.3 (0; 0.6)	z = 0.278, p = 0.781	0.2 (0.1; 0.4)	0.4 (0.2; 0.4)	z = -0.959, p = 0.338
• Cholesterol (mg)	217 (154; 415)	257 (149; 430)	z = -0.103, p = 0.9179	226 (149; 405)	424 (158; 690)	z = -0.570, p = 0.5685	224 (147; 407)	225 (173; 405)	z = -0.325, p = 0.745

Bolded cells represent significant results
[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale

Table 6.0.2: Spearman's correlations for selected macronutrients from food intake versus mood disorder symptoms and functioning scores

Nutrient	Young Mania Rating Scale Scores r (p-value)	Ham-D Depression Scale Scores r (p-value)	Global Assessment of Functioning Scores r (p-value)
Energy	-0.108 (p = 0.308)	0.006 (p = 0.954)	**0.214 (p = 0.041)**
Protein	-0.005 (p = 0.960)	0.039 (p = 0.708)	0.194 (p = 0.063)
Carbohydrates	-0.181 (p = 0.086)	-0.095 (p = 0.361)	**0.214 (p = 0.040)**
Fibre	-0.103 (p = 0.330)	-0.186 (p = 0.072)	**0.258 (p = 0.013)**
Total fat	-0.030 (p = 0.781)	0.020 (p = 0.852)	**0.215 (p = 0.040)**
Linoleic acid	0.078 (p = 0.470)	0.083 (p = 0.433)	**0.240 (p = 0.023)**
α-Linolenic acid	0.076 (p = 0.479)	0.073 (p = 0.490)	0.163 (p = 0.124)

Bolded cells represent significant results

6.1.2 Vitamins from Food Sources

The same analysis as outlined in Section 6.1.1 was used to examine bivariate relationships among food intakes of the B vitamins and vitamin C, mood disorder subtypes, and mood symptoms and revealed no significant differences (Table 6.0.3). However, there were apparent differences in intakes of folate and vitamin C according to diagnostic group as well Ham-D and YMRS cut-offs. Table 6.0.4 presents the correlations among the vitamins, mood symptoms, and GAF scores. Based on the two-sample Wilcoxon rank-sum (Mann-Whitney) test, no significant differences were found among the B vitamins and vitamin C based on mood disorder subtype or symptoms. Weak, positive significant Spearman correlations were found for YMRS scores and vitamin C ($r = -0.2495$, $p < 0.05$). The GAF scores significantly correlated ($p < 0.05$) with riboflavin ($r = 0.2993$), niacin ($r = 0.2523$), folate ($r = 0.2188$), vitamin B_6 ($r = 0.2339$), vitamin B_{12} ($r = 0.2751$), and pantothenic acid ($r = 0.2751$).

Table 6.0.3: Bivariate relationships between food intake of selected vitamins, mood disorder subtype, and mood symptoms

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups -statistic, p-value	Descriptive statistics by YMRS[3] Cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Folate (DFE)[3]	114 (58, 226)	40 (24, 71)	z = 1.248, p = 0.212	151 (161, 232)	96 (48, 208)	z = -1.125, p = 0.261	104 (57, 281)	95 (48, 208)	z = -0.476, p = 0.634
Niacin (NE)[4]	16 (12, 23)	16 (12, 24)	z = -0.299, p = 0.765	13 (12, 13)	27 (19, 34)	z = -0.130, p = 0.897	20 (16, 27)	16 (12, 22)	z = -1.559, p = 0.119
Panto-thenic acid (mg)	4 (3, 5)	4 (2, 6)	z = 0.633, p = 0.527	7 (2, 12)	4 (3, 5)	z = -0.445, p = 0.656	5 (4, 6)	4 (3, 5)	z = -1.577, p = 0.115
Riboflavin (mg)	2 (1, 2)	2 (1, 2)	z = 0.556, p = 0.578	3 (2, 4)	2 (1, 2)	z = -1.685, p = 0.092	2 (1, 3)	2 (1, 2)	z = -0.682, p = 0.495
Thiamin (mg)	2 (1, 2)	1 (1, 2)	z = 0.870, p = 0.384	1 (1, 1)	1 (1, 2)	z = 0.441, p = 0.660	2 (1, 2)	1 (1, 2)	z = -1.067, p = 0.286

[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale
[5]Dietary Folate Equivalents
[6]Niacin Equivalents

Table 6.0.3: Bivariate relationships between food intake of selected vitamins, mood disorder subtype, and mood symptoms /continued

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups -statistic, p-value	Descriptive statistics by YMRS[3] Cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Vitamin B_6 (mg)	2 (1, 2)	2 (1, 2)	z = 0.345, p = 0.730	1 (1, 1)	2 (1, 2)	z = -0.144, p = 0.885	2 (1, 3)	2 (1, 2)	z = -0.942, p = 0.346
Vitamin B_{12} (mcg)	3 (2, 5)	3 (2, 5)	z = 1.220, p = 0.222	6 (5, 7)	3 (2, 5)	z = -1.611, p = 0.107	4 (3, 4)	3 (2, 5)	z = -0.690, p = 0.490
Vitamin C (mg)	127 (63, 217)	100 (48, 171)	z = 0.791, p = 0.429	36 (12, 61)	110 (66, 184)	z = 1.763, p = 0.078	127 (63, 281)	108 (63, 182)	z = -0.531, p = 0.596

[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale

Table 6.0.4: Spearman's correlations for vitamins from food intake versus mood disorder symptoms and functioning scores

Nutrient	YMRS[1] Scores r (p-value)	Ham-D[2] Scores r (p-value)	GAF[3] Scores r (p-value)
Thiamin	0.026 (p = 0.807)	-0.055 (p = 0.601)	0.179 (p = 0.089)
Riboflavin	-0.022 (p = 0.837)	-0.043 (p = 0.678)	**0.299 (p = 0.004)**
Niacin	-0.044 (p = 0.676)	-0.034 (p = 0.744)	**0.252 (p = 0.015)**
Folate	-0.098 (p = 0.365)	-0.076 (p = 0.475)	**0.219 (p = 0.039)**
Vitamin B_6	-0.088 (p = 0.409)	-0.051 (p = 0.628)	**0.234 (p = 0.026)**
Vitamin B_{12}	0.129 (p = 0.226)	-0.001 (p = 0.991)	**0.216 (p = 0.040)**
Pantothenic acid	-0.089 (p = 0.406)	0.063 (p = 0.549)	**0.275 (p = 0.008)**
Vitamin C	**-0.250 (p = 0.017)**	-0.033 (p = 0.750)	0.125 (p = 0.236)

Bolded cells represent significant results
[1]Young Mania Rating Scale; [2]Hamilton Depression Scale; [3]Global Assessment of Functioning

6.1.3 Minerals from Food Sources

Significant correlations were found between GAF scores and calcium (r = 0.2461), phosphorus (r = 0.2996), potassium (r = 0.2642), iron (r = 0.3117) (all $p < 0.05$), magnesium (r = 0.4104, $p < 0.0001$), and zinc (r = 0.3529, $p < 0.001$) (Table 6.0.5). Better nutrient intakes tended to improve overall functioning. Relationships among food intakes, disorder types, and symptoms showed no significant differences (Table 6.0.6).

Table 6.0.5: Spearman's correlations for minerals from food intake versus mood disorder symptoms and functioning scores

Nutrient	YMRS[1] Scores r (p-value)	Ham-D[2] Scores r (p-value)	GAF[3] Scores r (p-value)
Calcium	-0.005 (p = 0.965)	-0.065 (p = 0.535)	**0.246 (p = 0.018)**
Magnesium	-0.133 (p = 0.209)	-0.027 (p = 0.794)	**0.410 (p = 0.000)**
Phosphorus	-0.068 (p = 0.520)	0.087 (p = 0.405)	**0.300 (p = 0.004)**
Sodium	-0.105 (p = 0.324)	0.164 (p = 0.114)	0.006 (p = 0.952)
Potassium	-0.194 (p = 0.065)	-0.017 (p = 0.868)	**0.264 (p = 0.011)**
Iron	-0.088 (p = 0.409)	-0.109 (p = 0.295)	**0.312 (p = 0.003)**
Zinc	0.060 (p = 0.574)	0.066 (p = 0.525)	**0.353 (p = 0.001)**

Bolded cells represent significant results
[1]Young Mania Rating Scale; [2]Hamilton Depression Scale; [3]Global Assessment of Functioning

Table 6.0.6: Bivariate relationships between food intake of selected minerals, mood disorder subtype, and mood symptoms

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[1] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Calcium (mg)	897 (682; 1233)	991 (647; 1280)	z = -0.745, p = 0.456	761 (291; 1231)	930 (652; 1233)	z = 0.518, p = 0.604	952 (683; 1233)	897 (647; 1231)	z = 0.054, p = 0.957
Iron (mg)	13 (15, 22)	15 (11, 23)	z = 0.416, p = 0.677	19 (14; 25)	15 (11; 22)	z = -0.467, p = 0.641	15 (12; 24)	15 (11; 22)	z = 0.173, p = 0.862
Magnesium (mg)	294 (201; 379)	283 (177; 383)	z = -0.004, p = 0.997	228 (158; 299)	282 (198; 383)	z = 0.700, p = 0.484	280 (206; 423)	286 (188; 378)	z = -0.682, p = 0.4951
Phosphorus (mg)	1087 (822; 1492)	1101 (863; 1535)	z = -0.685, p = 0.494	670 (499; 842)	1097 (836; 1492)	z = 1.555, p = 0.120	1175 (897; 1669)	1081 (827; 1458)	z = -0.790, p = 0.429
Zinc (mg)	9 (6; 13)	9 (6, 13)	z = -0.011, p = 0.991	10 (5; 15)	9 (6; 13)	z = -0.026, p = 0.979	9 (7; 13)	9 (6; 13)	z = -0.352, p = 0.725

[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale

Table 6.0.6: Bivariate relationships between food intake of selected minerals, mood disorder subtype, and mood symptoms /continued

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[1] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Potassium (mg)	3032 (2061; 3753)	2740 (2048; 3394)	z = 0.450, p = 0.653	1254 (382; 2126)	2958 (2061; 3671)	z = 1.737, p = 0.082	3560 (2560; 3917)	2814 (2030; 3456)	z = -1.397, p = 0.162
Sodium (mg)	3820 (3170; 5580)	4510 (2889; 6543)	z = -1.214, p = 0.225	3293 (834; 5748)	3879 (3018; 5854)	z = -0.026, p = 0.979	5118 (3785; 7630)	3765 (2993; 5648)	z = -1.516, p = 0.130

6.2 Nutrients from Food and Supplement Sources and Mood Disorders

6.2.1 Vitamins from Food and Supplement Sources

Results of the bivariate relationships among food and supplement intakes of the B vitamins and vitamin C, mood disorder subtypes, and symptoms are presented in Tables 6.0.7 and 6.0.8. Contrary to the food intake data, no significant relationships among the B vitamins and vitamin C emerged. This suggests that other interacting substances in foods (e.g., phytochemicals) may have contributed to these results.

Table 6.0.7: Spearman's correlations for food and supplement sources of vitamins versus mood disorder symptoms and functioning scores

Nutrient	YMRS[1] Scores r (p-value)	Ham-D[2] Scores r (p-value)	GAF[3] Scores r (p-value)
Thiamin	0.123 (p = 0.247)	-0.012 (p = 0.911)	0.091 (p = 0.394)
Riboflavin	-0.099 (p = 0.352)	-0.099 (p = 0.346)	0.167 (p = 0.114)
Niacin	-0.159 (p = 0.134)	-0.073 (p = 0.489)	0.104 (p = 0.328)
Folate	-0.058 (p = 0.587)	-0.047 (p = 0.656)	0.130 (p = 0.220)
Vitamin B_6	-0.118 (p = 0.273)	-0.080 (p = 0.449)	0.115 (p = 0.283)
Vitamin B_{12}	-0.060 (p = 0.577)	-0.030 (p = 0.780)	0.061 (p = 0.568)
Pantothenic acid	-0.107 (p = 0.320)	-0.058 (p = 0.586)	0.143 (p = 0.180)
Vitamin C	-0.050 (p = 0.637)	-0.039 (p = 0.708)	0.191 (p = 0.070)

[1]Young Mania Rating Scale; [2]Hamilton Depression Scale; [3]Global Assessment of Functioning

6.2.2 Minerals from Food and Supplement Sources

Those with YMRS scores over 20 had significantly lower median intakes of magnesium ($z = 2.044$, $p < 0.05$) and zinc ($z = 2.070$, $p < 0.05$). Those with Ham-D scores over 17 had significantly lower iron intakes ($z = 2.528$, $p < 0.05$). Ham-D scores correlated negatively with iron ($r = -0.2209$, $p < 0.05$); GAF scores positively correlated ($p < 0.05$) with calcium ($r = 0.2819$, magnesium ($r = 0.2418$), phosphorus ($r = 0.3030$), potassium ($r = 0.2122$), iron ($r = 0.2986$), and zinc ($r = 0.2182$) (Tables 6.0.9 and 6.0.10).

Table 6.0.8: Bivariate relationships between combined food and supplement intake of selected vitamins, mood disorder subtype, and mood symptoms

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[3] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs –median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Folate (DFE)[5]	104 (58; 214)	94 (36; 208)	z = -1.010, p = 0.313	148 (64; 232)	98 (45; 211)	z = -0.367, p = 0.714	91 (40; 281)	98 (49; 211)	z = 0.223, p = 0.824
Niacin (NE)[6]	26 (17; 330	27 (20; 35)	z = 0.623, p = 0.533	11 (2; 21)	27 (19; 34)	z = 1.520, p = 0.129	29 (20; 40)	26 (18; 34)	z = -1.030, p = 0.303
Pantothenic acid (mg)	8 (3; 54)	6 (3; 53)	z = -1.103, p = 0.270	629 (2; 1256)	6 (3; 53)	z = -0.159, p = 0.874	5 (2; 32)	7 (3; 57)	z = 0.956, p = 0.339
Riboflavin (mg)	3 (2; 12)	2 (1; 7)	z = -0.735, p = 0.462	14 (2; 25)	3 (1; 10)	z = -0.472, p = 0.637	2 (1; 26)	3 (2; 10)	z = 1.216, p = 0.224

[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale
[5]Dietary Folate Equivalents
[6]Niacin Equivalents

Table 6.0.8: Bivariate relationships between combined food and supplement intake of selected vitamins, mood disorder subtype, and mood symptoms

Nutrient	Descriptive statistics by diagnosis – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[3] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs –median (25th; 75th percentile)		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Thiamin (mg)	2 (1; 5)	2 (1; 5)	z = -0.786, p = 0.432	2 (1; 2)	2 (1; 5)	z = 0.629, p = 0.529	2 (1; 27)	2 (1; 5)	z = 0.404, p = 0.687
Vitamin B_6 (mg)	3 (2; 7)	2 (1; 5)	z = -1.197, p = 0.231	3 (2; 5)	3 (1; 7)	z = -0.026, p = 0.979	2 (1; 27)	3 (1; 7)	z = 0.586, p = 0.558
Vitamin B_{12} (mcg)	7 (3; 34)	6 (3; 16)	z = -0.738, p = 0.461	6 (5; 6)	7 (3; 31)	z = 0.185, p = 0.853	4 (2; 58)	7 (3; 27)	z = 0.886, p = 0.375
Vitamin C (mg)	188 (102; 632)	160 (74; 402)	z = -1.072, p = 0.284	122 (61; 184)	185 (81; 600)	z = 0.838, p = 0.402	121 (66; 281)	189 (89; 646)	z = 1.519, p = 0.129

[1]Bipolar Disorder
[2]Major Depressive Disorder
[3]Young Mania Rating Scale
[4]Hamilton Depression Scale

Table 6.0.9: Spearman's correlations for food and supplement sources of minerals versus mood disorder symptoms and functioning scores

Nutrient	YMRS[1] Scores r (p-value)	Ham-D[2] Scores r (p-value)	GAF[3] Scores r (p-value)
Calcium	-0.036, (p = 0.736)	-0.068 (p = 0.518)	**0.282 (p = 0.007)**
Magnesium	-0.063 (p = 0.553)	0.046 (p = 0.662)	**0.242 (p = 0.021)**
Phosphorus	-0.009 (p = 0.937)	-0.042 (p = 0.692)	**0.303 (p = 0.004)**
Sodium	-0.097 (p = 0.364)	0.162 (p = 0.120)	-0.029 (p = 0.782)
Potassium	-0.152 (p = 0.154)	0.085 (p = 0.420)	**0.212 (p = 0.043)**
Iron	-0.144 (p = 0.177)	**-0.221 (p = 0.033)**	**0.299 (p = 0.004)**
Zinc	**-0.254 (p = 0.016)**	0.134 (p = 0.201)	**0.218 (p = 0.038)**

Bolded cells represent significant results
[1]Young Mania Rating Scale; [2]Hamilton Depression Scale; [3]Global Assessment of Functioning

6.3 Assessment of Protein, Vitamin, and Mineral Inadequacies and Excesses

Table 6.0.11 presents odds ratio results of inadequate nutrient intakes from foods based on the Estimated Average Requirements (EAR) and bipolar disorder. There was insufficient data for depression. Table 6.0.11 also presents data of excess nutrient intakes from food and supplements as determined by values that exceeded the Upper Tolerable Intake Levels (UL) and bipolar disorder. There was insufficient data to examine odds ratios in comparison to mood symptom scores that did/did not exceed the cut-off values.

The only significant odds ratios for bipolar disorder and having inadequate nutrient intakes from food sources were for vitamin B_{12} (OR = 0.31, 95% CI 0.11 to 0.85) overall and for females (OR = 0.34, 95% CI 0.11 to 1.00). Values of the odds ratios of those with bipolar disorder having nutrient intakes from food and supplement sources that exceeded the UL that were less than one included niacin, calcium, magnesium (supplement use only), and zinc. Conversely, the odds ratio of excess folate, vitamin B_6, and iron intakes and bipolar disorder was greater than one. None of the overall or gender-specific odds ratios of nutrient excess and bipolar disorder were significant. Interestingly, iron showed a dissimilar trend to the overall odds ratio and odds ratio for males. Folate, niacin, and vitamin B_6 all exceeded an odds ratio of one. None of these odds ratios were significant.

Table 6.0.10: Bivariate relationships between combined food and supplement intake of selected minerals, mood disorder subtype, and mood symptoms

Nutrient	Bipolar Disorder – median (25th; 75th percentile)		Comparison of diagnostic groups - statistic, p-value	Descriptive statistics by YMRS[3] cut-offs – median (25th; 75th percentile)		Comparison of YMRS[3] subgroups - statistic, p-value	Descriptive statistics by Ham-D[4] cut-offs – median (25th; 75th percentile		Comparison of Ham-D[4] subgroups - statistic, p-value
	BD[1]	MDD[2]		≥ 20	< 20		≥ 17	< 17	
Calcium (mg)	1200 (876; 1601)	1212 (952; 1766)	z = 0.940, p = 0.347	1215 (1200; 1231)	1211 (880; 1654)	z = -0.026, p = 0.979	1197 (601; 1327)	1212 (905; 1654)	z = 0.733, p = 0.464
Iron (mg)	21 (15; 35)	19 (11; 35)	z = -0.921, p = 0.357	151 (14; 290)	21 (14; 35)	z = -0.341, p = 0.7334	**14 (10; 19)**	**23 (15; 36)**	**z = 2.528, p = 0.012**
Magnesium (mg)	349 (201; 464)	423 (203; 548)	z = 0.422, p = 0.673	**80 (3; 158)**	**375 (204; 520)**	**z = 2.044, p = 0.041**	398 (179; 528)	359 (202; 512)	z = -0.085, p = 0.932
Phosphorus (mg)	1287 (935; 1645)	1101 (859; 1567)	z = -0.716, p = 0.4741	1724 (842; 2607)	1186 (869; 1643)	z = -0.393, p = 0.6943	1129 (673; 1669)	1186 (886; 1643)	z = 0.754, p = 0.451
Zinc (mg)	10 (6; 15)	11 (7; 21)	z = 0.983, p = 0.326	**3 (1; 5)**	**11 (7; 19)**	**z = 2.070, p = 0.039**	10 (7; 20)	11 (6; 18)	z =-0.143, p = 0.886

Bolded cells represent significant results

[1]BD = Bipolar Disorder; [2]MDD= Major Depressive Disorder; [3]YMRS= Young Mania Rating Scale; [4]Ham-D= Hamilton Depression Scale

Table 6.0.11: Odds ratios of bipolar disorder, inadequate (food sources only), and excess intakes (food and supplements) of selected nutrient intakes

Nutrient	Odds of nutrient inadequacy (foods) and Bipolar Disorder (95% CI)			Odds of nutrient excess (food and supplements) and Bipolar Disorder (95% CI)		
	Overall odds ratio	Males Odds ratio	Females Odds ratio	Overall odds ratio	Males Odds ratio	Females Odds ratio
Protein	0.42 (0.08 to 1.94)	0.09 (0.001 to 2.53)	0.65 (0.09 to 4.27)	*****	*****	*****
Folate	0.58 (0.12 to 2.25)	****	0.67 (0.13 to 3.13)	6.75 (0.85 to 303.87)	****	7.2 (0.85 to 31.02)
Niacin	*	****	1.30 (0.20 to 9.47)	0.72 (0.25 to 2.14)	0.29 (0.04 to 2.02)	1.04 (0.25 to 4.71)
Vitamin B_6	**	****	****	1.36 (0.27 to 8.95)	0.41 (0.03 to 6.97)	3.27 (0.30 to 167.01)
Vitamin B_{12}	**0.31 (0.11 to 0.85)**	****	**0.34 (0.11 to 1.00)**	*****	*****	*****
Calcium	***	***	***	0.20 (0.003 to 2.68)	****	0.35 (0.006 to 7.19)

Bolded cells represent significant results
*Separate EARs according to gender
**Separate EARs according to gender and age groups
***No EAR for this nutrient
****Cannot be calculated due to one cell being of 0 value
*****No UL for this nutrient
[1]Supplement sources only

Table 6.0.12: Odds ratios of bipolar disorder, inadequate (food sources only), and excess intakes (food and supplements) of selected nutrient intakes

Nutrient	Odds of nutrient inadequacy (foods) and Bipolar Disorder (95% CI)			Odds of nutrient excess (food and supplements) and Bipolar Disorder (95% CI)		
	Overall odds ratio	Males Odds ratio	Females Odds ratio	Overall odds ratio	Males Odds ratio	Females Odds ratio
Iron	***	***	***	1.10 (0.32 to 4.07)	1.17 (0 .17 to 9.49)	0.71 (0.09 to 5.81)
Mag-nesium	**	****	****	0.82 (0.33 to 2.04)[1]	0.56 (0.07 to 3.69)[1]	0.90 (0.30 to 2.67)[1]
Phos-phorus	1.32 (0.32 to 6.47)	****	1.5 (0.32 to 7.88)	****	****	****
Zinc	*	****	1.10 (0.40 to 3.00)	0.3 (0.03 to 2.25)	****	0.46 (0.04 to 4.40)

Bolded cells represent significant results
*Separate EARs according to gender
**Separate EARs according to gender and age groups
***No EAR for this nutrient
****Cannot be calculated due to one cell being of 0 value
*****No UL for this nutrient
[1]Supplement sources only

6.4 Assessment of Nutrients from Blood Samples and Mood Disorders

Correlations among blood levels and mental health status variables indicated that only YMRS scores correlated with ferritin ($r = 0.332$, $p < 0.05$) (Table 6.0.12). There was insufficient data to examine YMRS scores exceeding 20. RBC folate was significantly lower for Ham-D scores above the cut-off ($t = -2.382$, $p < 0.05$) (Table 6.0.13).

Table 6.0.13: Spearman's correlations for selected biochemical indicators versus mood disorder symptoms and functioning scores

Nutrient	YMRS[1] Scores r (p-value)	Ham-D[2] Scores r (p-value)	GAF[3] Scores r (p-value)
RBC folate	0.212 (p = 0.299)	0.369 (p = 0.059)	0.208 (p = 0.308)
Vitamin B_{12}	-0.075 (p = 0.650)	0.299 (p = 0.055)	0.203 (p = 0.210)
Vitamin E	-0.091 (p = 0.587)	-0.069 (p = 0.667)	0.027 (p = 0.869)
Ferritin	**0.332 (p = 0.039)**	0.178 (p = 0.259)	-0.256 (p = 0.112)
Albumin	0.137 (p = 0.406)	0.067 (p = 0.675)	0.115 (p = 0.480)
Cholesterol	-0.105 (p = 0.526)	0.142 (p = 0.369)	0.069 (p = 0.671)

Bolded cells represent significant results
[1]Young Mania Rating Scale; [2]Hamilton Depression Scale; [3]Global Assessment of Functioning

6.5 Models of Nutritional Status

Relationships among: 1) energy, macronutrients, sociodemographic, and health-related variables; 2) biochemical nutrient indicators, nutrients, sociodemographic, and health-related variables; 3) psychiatric symptoms and functioning, sociodemographic, and health-related variables with nutrients from food sources then from food and supplement sources; and 4) psychiatric symptoms and functioning, sociodemographic, biochemical indicators of nutrient status, and health-related variables were analyzed.

The variables chosen for each regression depended on significant relationships found in prior analyses as well as the study's hypotheses. For example, age, gender, income, and marital status appeared to have significant relationships with the intake of various nutrients and were therefore included. Where marital status was included in the analysis it was dropped from the final results, possibly due to its potential overlap with income, and it is therefore excluded from the analyses. Diagnostics were applied to the final model as described in Chapter Three.

Table 6.0.14: Bivariate relationships between blood values of selected nutrients, mood disorder subtype, and mood symptoms

Nutrient	Bipolar Disorder – median (25^{th}; 75^{th} percentile)	Major Depressive Disorder – median (25^{th}; 75^{th} percentile)	Comparison of diagnostic groups – statistic, p-value	YMRS[1] –median (25^{th}; 75^{th} percentile)	Ham-D[2] –median (25^{th}; 75^{th} percentile)		Comparison of Ham-D[2] sub-groups – statistic, p-value
				< 20	≥ 17	< 17	
Serum folate	54 (37; 55)	54 (50; 56)	t = -0.650, p = 0.529	54 (44; 54)	*	54 (44; 54)	*
RBC folate	920 (746; 1077)	915 (768; 1118)	t = -1.067, p = 0.296	915 (746; 1077)	1114 (1074; 1591)	908 (756; 1008)	**t = -2.382, p = 0.025**
Vitamin B_{12}	327 (230; 502)	360 (297; 629)	t = -0.201, p = 0.842	349 (259; 565)	489 (230; 742)	321 (255; 494)	t = -0.921, p = 0.362
Ferritin	61 (46; 93)	59 (34; 121)	t = 0.457, p = 0.650	61 (44; 93)	49 (21; 243)	62 (46; 93)	t = -1.583, p = 0.121
Vitamin E	30 (25; 35)	32 (24; 35)	t = 0.042, p = 0.967	30 (25; 35)	29 (25; 33)	30 (25; 35)	t = 0.608, p = 0.547
Albumin	44 (42; 47)	46 (44, 48)	t = -1.748, p = 0.088	44 (42; 47)	44 (42; 47)	44 (43; 47)	t = 0.317, p = 0.753
Cholesterol	5 (4; 6)	5 (4; 6)	t = -0.364, p = 0.718	5 (4; 6)	5 (4; 7)	5 (4; 6)	t = -0.634, p = 0.530

Bolded cells represent significant results

*Insufficient data

[1]Young Mania Rating Scale; [2]Hamilton Depression Scale; [3]Global Assessment of Functioning

6.5.1 Energy and Macronutrient Consumption

6.5.1.1 Energy Intake

The following model was analyzed:

Energy (kcal) = Gender (male/female) + Income (≤$20000/>$20000) + Age (years) + Disorder (bipolar/major depressive disorder) + Special Diet (yes/no)

Psychiatric medication was included, but it was dropped from the analyses involving macronutrients because most participants were taking medication. The interaction of age and type of disorder was a significant predictor of energy intake (regression coefficient = 18.554, SE = 8.338, t = 2.23, $p < 0.05$, 95% CI 1.967 to 35.141) (Table 6.0.14). Diagnostics of this model indicated that the linear model represented the data well.

Table 6.0.15: Estimates of energy intake

	Model 1
Adjusted R^2	0.2383
Regression estimates	
Intercept	5008.07 (SE = 1393.087, t = 3.59, p = 0.001, 95% CI 2236.774 to 7779.363)
Gender	-814.91 (SE = 752.698, t = -1.08, p = 0.282, 95% CI -2312.267 to 682.444)
Income	**-401.86 (SE = 196.229, t = -2.05, p = 0.044, 95% CI -792.217 to -11.493)**
Age	**-37.89 (SE = 9.708, t = -3.90, p = 0.000, 95% CI -57.198 to -18.576)**
Special diet	225.93 (SE = 1309.629, t = 0.17, p = 0.863, 95% CI -2379.337 to 2831.201)
Type of disorder	1043.23 (SE = 393.231, t = 2.65, p = 0.010, 95% CI 260.972 to 1825.496)
Age*disorder	**18.55 (SE = 8.338, t = 2.23, p = 0.029, 95% CI 1.967 to 35.141)**
Age*Special diet	-7.50 (SE = 25.889, t = -0.29, p = 0.773, 95% CI -58.999 to 44.005)
Gender*disorder	-3.73 (SE = 15.486, t = -0.24, p = 0.810, 95% CI -34.532 to 27.080)
Gender*Special diet	-763.37 (SE = 479.975, t = -1.59, p = 0.116, 95% CI -1718.197 to 191.448)

Bolded cells represent significant results
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

6.5.1.2 Protein Intake

The following regression model was analyzed:

Protein (grams) = Gender (male/female) + Income (≤$20000/>$20000) + Age (years) + Disorder (bipolar/major depressive disorder) + Special Diet (yes/no)

In Model Three, age (regression coefficient = -0.787, SE = 0.252, t = -3.13, $p < 0.05$, 95% CI -1.287 to -0.287), gender (regression coefficient = 13.635, SE = 6.836, t = 1.99, $p < 0.05$, 95% CI 0.052 to 27.218), and income (regression coefficient = 13.635, SE = 6.836, t = 1.99, $p < 0.05$, 95% CI 0.052 to 27.218) were predictors of protein intake. Type of disorder was possibly a confounder of gender.

Table 6.0.16: Estimates of protein intake

	Model 1	Model 2	Model 3
Adjusted R^2	0.0966	0.0842	0.0945
Regression estimates			
Intercept	147.264 (SE = 20.099, t = 7.33, p < 0.001, 95% CI 107.296 to 187.233)	129.153 (SE = 14.228, t = 9.08, p < 0.001, 95% CI 100.877 to 157.429)	129.279 (SE = 12.670, t = 10.20, p < 0.001, 95% CI 104.103 to 154.455)
Gender	24.622 (SE = 25.349, t = 0.97, p = 0.334, 95% CI -25.787 to 75.032)	13.618 (SE = 6.926, t = 1.97, p = 0.052, 95% CI -0.145 to 27.381)	13.635 (SE = 6.836, t = 1.99, p = 0.049, 95% CI 0.052 to 27.218)
Income	-13.373 (SE = 6.797, t = -1.97, p = 0.052, 95% CI -26.889 to 0.142)	-12.981 (SE = 6.527, t = -1.99, p = 0.050, 95% CI -25.953 to -0.001)	-13.003 (SE = 6.402, t = -2.03, p = 0.045, 95% CI -25.723 to -0.283)
Age	-1.179 (SE = 0.402, t = -2.93, p = 0.004, 95% CI -1.978 to -0.379)	-0.786 (SE = 0.258, t = -3.05, p = 0.003, 95% CI -1.298 to -0.274)	-0.787 (SE = 0.252, t = -3.130, p = 0.002, 95% CI -1.287 to -0.287)
Special diet	55.001 (SE = 44.979, t = 1.22, p = 0.225, 95% CI -34.444 to 144.446)	2.287 (SE = 7.971, t = 0.29, p = 0.775, 95% CI -13.553 to 18.128)	2.284 (SE = 7.92, t = 0.29, p = 0.774, 95% CI -13.461 to 18.029)
Type of disorder	-41.567 (SE = 24.493, t = -1.70, p = 0.093, 95% CI -90.274 to 7.141)	0.127 (SE = 6.386, t = 0.02, p = 0.984, 95% CI -12.563 to 12.817)	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.15: Estimates of protein intake /continued			
	Model 1	**Model 2**	**Model 3**
Regression estimates			
Age* Disorder	0.918 (SE = 0.512, t = 1.79, p = 0.077, 95% CI -0.101 to 1.937)	--	--
Age* Special diet	-0.987 (SE = 0.889, t = -1.11, p = 0.270, 95% CI -2.755 to 0.782)	--	--
Gender* Disorder	-0.138 (SE = 0.529, t = -0.26, p = 0.795, 95% CI -1.190 to 0.914)	--	--
Gender* Special diet	-15.027 (SE = 16.540, t = -0.91, p = 0.366, 95% CI -47.917 to 17.864)	--	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

6.5.1.3 Carbohydrate Intake

The following model was analyzed:

Carbohydrate (grams) = Gender (male/female) + Income (≤$20000/>$20000) + Age (years) + Disorder (bipolar/major depressive disorder) + Special Diet (yes/no)

In the final model (Model Three), age (regression coefficient = -4.336, SE = 1.090, t = 3.98, p < 0.001, 95% CI -6.503 to -2.170) and gender (regression coefficient = -67.341, SE = 29.809, t = -2.26, p = 0.026, 95% CI -126.591 to -8.092) were significant predictors of carbohydrate intake. Special diet was close to significance (regression coefficient = -67.013, SE = 34.069, t = -1.97, p = 0.052, 95% CI -134.728 to 0.702) (Table 6.0.16). Diagnostic plots and Cook's Distance calculation revealed one observation as possibly influential. The model was analyzed with this observation deleted and the results remained similar to those presented for Model Three.

Table 6.0.17: Estimates of carbohydrate intake			
	Model 1	**Model 2**	**Model 3**
Adjusted R^2	0.1690	0.1867	0.1923
Regression estimates			
Intercept	705.671 (SE = 223.6024, t = 3.16, p = 0.002, 95% CI 260.854 to 1150.487)	690.798 (SE = 87.679, t = 7.88, p = 0.000, 95% CI 516.499 to 865.097)	669.295 (SE = 80.618, t = 8.30, p = 0.000, 95% CI 509.058 to 829.532)
Gender	-31.772 (SE = 114.136, t = -0.28, p = 0.781, 95% CI -258.824 to 195.280)	-69.294 (SE = 30.069, t = -2.30, p = 0.024, 95% CI -129.069 to -9.519)	-67.341 (SE = 29.809, t = -2.26, p = 0.026, 95% CI -126.591 to -8.092)
Income	-48.941 (SE = 29.861, t = -1.64, p = 0.105, 95% CI -108.344 to 10.462)	-51.374 (SE = 28.09, t = -1.83, p = 0.071, 95% CI -107.215 to 4.467)	-48.593 (SE = 27.652, t = -1.76, p = 0.082, 95% CI -103.554 to 6.369)
Age	-6.405 (SE = 1.793, t = -3.57, p = 0.001, 95% CI -9.972 to -2.839)	-4.470 (SE = 1.114, t = -4.01, p = 0.000, 95% CI -6.684 to -2.256)	-4.336 (SE = 1.090, t = 3.98, p = 0.000, 95% CI -6.503 to -2.170)
Special diet	-50.697 (SE = 197.157, t = -0.26, p = 0.798, 95% CI -442.905 to 341.511)	-67.508 (SE = 34.195, t = -1.97, p = 0.052, 95% CI -135.485 to 0.469)	-67.013 (SE = 34.069, t = -1.97, p = 0.052, 95% CI -134.728 to 0.702)
Type of disorder	-144.981 (SE = 109.261, t = -1.33, p = 0.188, 95% CI -362.336 to 72.375)	-17.547 (SE = 27.593, t = -0.64, p = 0.527, 95% CI -72.400 to 37.307)	--
Age* Type of disorder	2.729 (SE = 2.271, t = 1.20, p = 0.233, 95% CI -1.789 to 7.247)	--	--
Age* Special diet	-0.134 (SE = 3.896, t = -0.03, p = 0.973, 95% CI -7.885 to 7.617)	--	--
Gender* Type of disorder	1.046 (SE = 2.354, t = 0.44, p = 0.658, 95% CI -3.638 to 5.729)	--	--
Gender* Special diet	-43.507 (SE = 72.207, t = -0.60, p = 0.548, 95% CI -187.151 to 100.136)	--	--

Shaded cells indicate final model chosen
SE = standard error; t = t-value; CI = confidence interval; ns = not significant

6.5.1.4 Fat Intake

The following model was analyzed:

Fat (g) = Gender (male/female) + Income (≤$20000/>$20000) + Age (years) + Disorder (bipolar/major depressive disorder) + Special Diet (yes/no)

In the final model (Model 3), age (regression coefficient = -1.204, SE = 0.426, t = -2.83, $p < 0.05$, 95% CI -2.050 to -0.357) and gender (regression coefficient = -27.582, SE = 11.650, t = -2.37, $p < 0.05$, 95% CI -50.737 to 4.427) were significant, suggesting that these are predictors of fat intake in this population (Table 6.0.17). However, it is important to note that the interaction of gender and special diet was close to significance ($p = 0.054$); possibly suggesting effect modification. Diagnostic plots revealed that the linear model explains the variation related to the independent variable.

Table 6.0.18: Estimates of fat intake

	Model 1	Model 2	Model 3
Adjusted R^2	0.1150	0.0824	0.0928
Regression estimates			
Intercept	327.119 (SE = 85.097, t = 3.84, p = 0.000, 95% CI 157.835 to 496.404)	211.196 (SE = 34.344, t = 6.15, p = 0.000, 95% CI 142.923 to 279.469)	209.793 (SE = 31.506, t = 6.66, p = 0.000, 95% CI 147.171 to 272.415)
Gender	-93.753 (SE = 43.437, t = -2.16, p = 0.034, 95% CI -180.163 to -7.343)	-27.709 (SE = 11.778, t = -2.35, p = 0.021, 95% CI -51.124 to -4.295)	-27.582 (SE = 11.650, t = -2.37, p = 0.020, 95% CI -50.737 to 4.427)
Income	-20.971 (SE = 1.364, t = -1.85, p = 0.069, 95% CI -43.578 to 1.636)	-14.699 (SE = 11.003, t = -1.34, p = 0.185, 95% CI -36.573 to 7.174)	-14.518 (SE = 10.807, t = -1.34, p = 0.183, 95% CI -35.998 to 6.962)
Age	-0.880 (SE = 0682, t = -1.29, p = 0.201, 95% CI -2.238 to 0.477)	-1.212 (SE = 0.436, t = -2.78, p = 0.007, 95% CI -2.080 to -0.345)	-1.204 (SE = 0.426, t = -2.83, p = 0.006, 95% CI -2.050 to -0.357)

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.17: Estimates of fat intake /continued			
	Model 1	**Model 2**	**Model 3**
Regression estimates continued			
Special diet	53.290 (SE = 75.032, t = 0.71, p = 0.480, 95% CI -95.974 to 202.553)	-9.462 (SE = 13.394, t = -0.71, p = 0.482, 95% CI -36.089 to 17.165)	-9.430 (SE = 13.314, t = -0.71, p = 0.481, 95% CI -35.894 to 17.034)
Type of disorder	-5.812 (SE = 41.582, t = -0.14, p = 0.889, 95% CI -88.531 to 76.907)	1.145 (SE = 10.808, t = -0.11, p = 0.916, 95% CI -22.631 to 20.342)	--
Age*Type of disorder	0.113 (SE = 0.864, t = 0.13, p = 0.896, 95% CI -1.606 to 1.833)	--	--
Age*Special diet	-0.820 (SE = 1.483, t = -0.55, p = 0.582, 95% CI -3.770 to 2.129)	--	--
Gender* Disorder	1.098 (SE = 0.896, t = -1.23, p = 0.224, 95% CI -2.880 to 0.684)	--	--
Gender* Special diet	-53.664 (SE = 27.480, t = -1.95, p = 0.054, 95% CI -108.331 to 1.003)	--	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

6.5.2 Biochemical Indicators

6.5.2.1 Red Blood Cell (RBC) Folate

The following model was analyzed:

RBC Folate (nmol/L) = Folate Intake (dietary folate equivalents) + Gender (male/female) + Income (≤$20000/>$20000) + Age (years) + Disorder (bipolar/major depressive disorder) + Psychiatric medication (yes/no)

In the initial analysis the interaction term dietary folate and medications was included but dropped from the analysis. In the final model (Model Three), dietary folate intake (regression coefficient = 1.610, SE = 0.310, t = 5.20, p < 0.001, 95% CI 0.964 to 2.257), and psychiatric medication (regression coefficient = -872.614, SE = 192.374, t = -4.54, p < 0.001, 95% CI -1273.899 to -471.330) continued to be significant, suggesting that these variables are significant predictors of RBC folate (Table 6.0.18). Diagnostics indicated that the model explains the variation related to the independent variable.

Table 6.0.19: Estimates of red blood cell folate

	Model 1	Model 2	Model 3
Adjusted R^2	0.5836	0.6065	0.6239
Regression estimates			
Intercept	1562.428 (SE = 383.754, t = 4.07, p = 0.001, 95% CI 752.778 to 2372.077)	1546.702 (SE = 334.716, t = 4.62, p = 0.000, 95% CI 846.133 to 2247.270)	1505.619 (SE = 304.407, t = 4.95, p = 0.000, 95% CI 870.638 to 2140.600)
Dietary folate intake	2.936 (SE = 1.819, t = 1.61, p = 0.125, 95% CI -0.902 to 6.775)	1.561 (SE = 0.350, t = 4.46, p = 0.000, 95% CI 0.829 to 2.293)	1.610 (SE = 0.310, t = 5.20, p = 0.000, 95% CI 0.964 to 2.257)
Income	-140.667 (SE= 102.959, t = -1.37, p = 0.190, 95% CI -357.892 to 76.558)	-162.703 (SE = 97.651, t = -1.67, p = 0.112, 95% CI -367.088 to 41.682)	-151.807 (SE = 89.997, t = -1.69, p = 0.107, 95% CI -339.538 to 35.924)
Age	0.515 (SE = 7.194, t = 0.49, p = 0.631, 95% CI -11.664 to 18.694)	0.719 (SE = 4.342, t = 0.17, p = 0.870, 95% CI -8.370 to 9.808)	1.332 (SE = 3.849, t = 0.35, p = 0.733, 95% CI -6.696 to 9.361)
Gender	-62.265 (SE = 151.062, t = -0.41, p = 0.685, 95% CI -380.978 to 256.448)	62.029 (SE = 76.368, t = 0.81, p = 0.427, 95% CI -97.812 to 221.870)	61.253 (SE = 74.619, t = 0.82, p = 0.421, 95% CI -94.400 to 216.907)
Psychiatric medication	-800.582 (SE = 216.703, t = -3.69, p = 0.002, 95% CI -1257.785 to -343.380)	-856.511 (SE = 202.588, t = -4.23, p = 0.000, 95% CI -1280.533 to -432.490)	-872.614 (SE = 192.374, t = -4.54, p = 0.000, 95% CI -1273.899 to -471.330)

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.18: Estimates of red blood cell folate /continued

	Model 1	Model 2	Model 3
Type of disorder	-35.271 (SE = 94.385, t = -0.37, p = 0.713, 95% CI -234.407 to 163.864)	-30.627 (SE = 91.505, t = -0.33, p = 0.742, 95% CI -222.148 to 160.895)	--
Age*Diet folate intake	-0.020 (SE = 0.035, t = -0.57, p = 0.576, 95% CI -0.094 to 0.054)	--	--
Gender*Diet folate intake	1.050 (SE = 1.096, t = -0.96, p = 0.352, 95% CI -3.362 to 1.263)	--	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

A regression analysis was also attempted for ferritin, however results indicated overfitting of the model and the data is not reported.

6.5.3 Food Sources of Nutrients and Psychiatric Symptoms and Functioning

6.5.3.1 Mood Disorder Symptoms

To address the tertiary objectives, two separate models to predict mood symptoms were analyzed:

Hamilton Depression Scores = Gender (male/female) + Age (years) + Income (≤$20000/>$20000) + Energy (kilocalories) + Psychiatric medication (yes/no) + Age*Energy + Gender*Energy

Young Mania Rating Scale Scores = Gender (male/female) + Age (years) + Energy intake (kilocalories) + Vitamin C (mg) + Psychiatric medication (yes/no) + Age*Energy + Gender*Energy + Age*Vitamin C + Gender*Vitamin C

Vitamin C was included in the latter model because earlier analysis in this study and some research suggests it is associated with manic symptoms. All regression estimates for both models were non-significant (data not shown). Diagnostic plots for the final models suggested that all assumptions had been met. Vitamin C may not have shown to

be significantly related to mania symptoms due to the smaller sample analyzed or when considered in the context of other variables, the correlational relationship may not persist.

6.5.3.2 Global Assessment of Functioning

The model analyzed to predict GAF Scores was as follows:

Global Assessment of Functioning Scores =	Gender (male/female) + Age (years) + Energy (kilocalories) + Carbohydrates (g) + Total fat (g) + Omega 6 fats (g) + Vitamin B_2 (mg) + Vitamin B_3 (mg) + Folate (mcg) + Vitamin B_6 (mg) + Vitamin B_{12} (mg) + Pantothenic Acid (mg) + Calcium (mg) + Iron (mg) + Phosphorus + Potassium (mg) + Zinc (mg) + Psychiatric medication (yes/no)

The model included all nutrients that were significantly correlated with GAF scores. Model One considered age and gender as effect modifiers of energy (Table 6.0.19). Other interaction terms were not included due to the high variable-to-sample-size ratio. The interaction terms for age, gender, and energy were non-significant. Model Two analyzed all independent variables with the interaction terms omitted. Total fat (regression coefficient = 0.392, SE = 0.190, t-value = 2.07, p = 0.042, 95% CI 0.014 to 0.771) was significant. Diagnostic plots for the final model (Model Two) were analyzed suggesting that all model assumptions had been met.

6.5.4 Food and Supplement Sources of Nutrients and Psychiatric Symptoms and Functioning

6.5.4.1 Hamilton Depression Scores

The model analyzed was as follows:

Hamilton Depression Scores =	Gender (male/female) + Age (years) + Iron (mg) + Psychiatric medication (yes/no) + Age*Iron + Gender*Iron

Iron was significantly correlated with Ham-D scores and was included in the model. The first model considered age and gender as effect modifiers of iron intake and all regression estimates were non-significant. The second model analyzed all independent variables without the interaction terms and found no significant regression coefficients. To assess for potential effects of confounding, a third model was analyzed, ruling out condition as a confounder. Diagnostic plots for the final model were analyzed, suggesting that all model assumptions had been met (all data not shown).

Table 6.0.20: Estimates of Global Assessment of Functioning scores					
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Adjusted R^2	0.0677	0.0738	0.0873	0.0541	0.0792
Regression estimates					
Intercept	32.113 (SE = 27.279, t = 1.18, p = 0.243, 95% CI -22.366 to 86.592)	44.644 (SE = 14.090, t = 3.17, p = 0.002, 95% CI 16.520 to 72.769)	45.161 (SE = 12.881, t = 3.51, p = 0.001, 95% CI 19.457 to 70.865)	42.515 (SE = 12.832, t = 3.31, p = 0.001, 95% CI 6.929 to 68.101)	48.544 (SE = 11.285, t = 4.30, p = 0.000, 95% CI 26.078 to 71.010)
Gender	-3.856 (SE = 10.850, t = -0.36, p = 0.723, 95% CI -25.525 to 17.813)	-0.466 (SE = 3.954, t = -0.12, p = 0.907, 95% CI -8.358 to 7.426)	-0.438 (SE = 3.914, t = -0.11, p = 0.911, 95% CI -8.249 to 7.372)	-1.151 (SE = 3.812, t = -0.30, p = 0.764, 95% CI -8.751 to 6.449)	-2.348 (SE = 3.689, t = -0.64, p = 0.526, 95% CI -9.693 to 4.997)
Age	0.555 (SE = 0.367, t = 1.51, p = 0.135, 95% CI -0.178 to 1.288)	0.145 (SE = 0.138, t = 1.05, p = 0.296, 95% CI -0.130 to 0.421)	0.145 (SE = 0.137, t = 1.06, p = 0.293, 95% CI -0.128 to 0.419)	0.161 (SE = 0.139, t = 1.16, p = 0.251, 95% CI -0.116 to 0.438)	0.141 (SE = 0.131, t = 1.07, p = 0.287, 95% CI -0.120 to 0.402)
Energy	-0.030 (SE = 0.020, t = -1.50, p = 0.139, 95% CI -0.071 to 0.010)	-0.036 (SE = 0.020, t = -1.84, p = 0.071, 95% CI -0.075 to 0.003)	-0.037 (SE = 0.019, t = -1.94, p = 0.056, 95% CI -0.074 to 0.001)	0.001 (SE = 0.003, t = 0.44, p = 0.660, 95% CI -0.004 to 0.006)	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.19: Estimates of Global Assessment of Functioning scores /continued					
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Regression estimates continued					
Carbo-hydrates	0.139 (SE = 0.081, t = 1.72, p = 0.090, 95% CI -0.022 to 0.301)	0.134 (SE = 0.080, t = 1.68, p = 0.098, 95% CI -0.0256 to 0.293)	0.136 (SE = 0.077, t = 1.76, p = 0.083, 95% CI -0.0018 to 0.289)	--	--
Total fat	0.417 (SE = 0.192, t = 2.17, p = 0.033, 95% CI 0.034 to 0.800)	0.392 (SE = 0.190, t = 2.07, p = 0.042, 95% CI 0.014 to 0.771)	0.397 (SE = 0.181, t = 2.19, p = 0.032, 95% CI 0.036 to 0.759)	--	--
Omega 6	-0.436 (SE = 0.437, t = -1.00, p = 0.323, 95% CI -1.308 to 0.437)	-0.484 (SE = 0.433, t = -1.12, p = 0.268, 95% CI -1.347 to 0.380)	-0.493 (SE = 0.417, t = -1.18, p = 0.241, 95% CI -1.325 to 0.339)	--	--
Vitamin B_2	0.182 (SE = 4.173, t = 0.04, p = 0.965, 95% CI -8.151 to 8.515)	-0.690 (SE = 3.927, t = -0.18, p = 0.861, 95% CI -8.529 to 7.149)	-0.683 (SE = 3.898, t = -0.18, p = 0.861, 95% CI -8.462 to 7.095)	-0.888 (SE = 3.916, t = -0.23, p= 0.821, 95% CI -8.697 to 6.920)	1.521 (SE = 3.262, t = 0.47, p = 0.642, 95% CI -4.974 to 8.015)

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.19: Estimates of Global Assessment of Functioning scores /continued					
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Regression estimates /continued					
Vitamin B_3	0.263 (SE = 0.327, t = 0.80, p = 0.424, 95% CI -0.03901 to 0.917)	0.286 (SE = 0.325, t = 0.88, p = 0.382, 95% CI -0.362 to 0.934)	0.286 (SE = 0.322, t = 0.89, p = 0.378, 95% CI -0.357 to 0.929)	0.133 (SE = 0.309, t = 0.43, p = 0.667, 95% CI -0.482 to 0.749)	0.049 (SE = 0.245, t = 0.20, p = 0.843, 95% CI -0.439 to 0.537)
Vitamin B_6	-0.494 (SE = 2.97, t = -0.17, p = 0.869, 95% CI -6.433 to 5.445)	-0.096 (SE = 2.940, t = -0.03, p = 0.974, 95% CI 0.965 to 5.773)	-0.132 (SE = 2.894, t = -0.05, p = 0.964, 95% CI -5.906 to 5.642)	2.018 (SE = 2.750, t = 0.73, p = 0.466, 95% CI -3.465 to 7.500)	1.426 (SE = 2.252, t = 0.63, p = 0.528, 95% CI -3.057 to 5.909)
Vitamin B_{12}	-0.849 (SE = 0.524, t = -1.62, p = 0.110, 95% CI -1.895 to 0.197)	-0.783 (SE = 0.514, t = -1.52, p = 0.132, 95% CI -1.809 to 0.243)	-0.793 (SE = 0.500, t = -1.59, p = 0.117, 95% CI -1.789 to 0.204)	-0.868 (SE = 0.492, t = -1.76, p = 0.082, 95% CI -1.850 to 0.113)	-0.745 (SE = 0.461, t = -1.62, p= 0.110, 95% CI -1.662 to 0.172)
Pantothenic acid	1.116 (SE = 1.531, t = 0.73, p = 0.469, 95% CI -1.942 to 4.174)	1.282 (SE = 1.507, t = 0.85, p = 0.398, 95% CI -1.726 to 4.291)	1.306 (SE = 1.476, t = 0.88, p = 0.379, 95% CI -1.639 to 4.252)	1.010 (SE = 1.381, t = 0.73, p = 0.467, 95% CI -1.745 to 3.764)	1.027 (SE = 0.196, t = 0.86, p = 0.393, 95% CI -1.353 to 3.407)

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.19: Estimates of Global Assessment of Functioning scores /continued					
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Regression estimates /continued					
Folate	-0.001 (SE = 0.008, t = -0.12, p = 0.905, 95% CI -0.017 to 0.015)	-0.002 (SE = 0.008, t = -0.25, p = 0.800, 95% CI -0.018 to 0.015)	-0.002 (SE = 0.008, t = -0.25, p = 0.806, 95% CI -0.018 to 0.014)	-0.004 (SE = 0.008, t = -0.51, p = 0.612, 95% CI -0.019 to 0.011)	--
Iron	0.163 (SE = 0.321, t = 0.51, p = 0.613, 95% CI -0.477 to 0.804)	0.206 (SE = 0.318, t =0.65, p = 0.520, 95% CI -0.4287 to 0.840)	0.209 (SE = 0.313, t = 0.67, p = 0.507, 95% CI -0.416 to 0.8342)	0.0648 (SE = 0.313, t = 0.21, p = 0.836, 95% CI -0.558 to 0.688)	0.044 (SE = 0.267, t = 0.16, p = 0.871, 95% CI -0.488 to 0.576)
Zinc	0.438 (SE = 0.345, t = 1.27, p = 0.209, 95% CI -0.252 to 1.128)	0.461 (SE = 0.341, t = 1.35, p = 0.181, 95% CI -0.220 to 1.142)	0.461 (SE = 0.339, t = 1.36, p = 0.178, 95% -0.215 to 1.137)	0.369 (SE = 0.342, t = 1.08, p = 0.283, 95% CI -0.312 to 1.051)	0.417 (SE = 0.320, t = 1.30, p = 0.197, 95% CI -0.221 to 1.054)
Calcium	0.006 (SE = 0.005, t = 1.12, p = 0.266, 95% CI -0.005 to 0.017)	0.005 (SE = 0.005, t = 1.04, p = 0.300, 95% CI -0.005 to 0.016)	0.005 (SE = 0.005, t = 1.05, p = 0.297, 95% CI -0.005 to 0.016)	0.003 (SE = 0.005, t = 0.55, p = 0.581, 95% CI -0.007 to 0.013)	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

Table 6.0.19: Estimates of Global Assessment of Functioning scores /continued					
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**
Regression estimates /continued					
Phosphorus	0.007 (SE = 0.007, t = 1.00, p = 0.319, 95% CI -0.007 to 0.022)	0.007 (SE = 0.007, t = 0.96, p = 0.340, 95% CI -0.007 to 0.021)	0.007 (SE = 0.007, t = 0.99, p = 0.327, 95% CI -0.007 to 0.021)	0.007 (SE = 0.007, t = 0.97, p = 0.335, 95% CI -0.007 to 0.020)	--
Potassium	-0.001 (SE = 0.002, t = -0.35, p = 0.727, 95% CI -0.006 to 0.004)	-0.001 (SE = 0.002, t = -0.60, p = 0.553, 95% CI -0.006 to 0.003)	-0.001 (SE = 0.002, t = -0.60, p = 0.551, 95% CI -0.006 to 0.003)	-0.002 (SE = 0.002, t = -1.07, p = 0.287, 95% CI -0.007 to 0.002)	--
Psychiatric medication	-0.172 (SE = 6.160, t = -0.03, p = 0.978, 95% CI -12.475 to 12.130)	0.572 (SE = 6.082, t = 0.09, p = 0.925, 95% CI -11.567 to 12.711)	--	--	--
Age* Energy	-0.000 (SE = 0.000, t = -1.22, p = 0.227, 95% CI -0.000 to 0.000)	--	--	--	--
Gender* Energy	-0.001 (SE = 0.004, t = -0.33, p = 0.740, 95% CI -0.009 to 0.007)	--	--	--	--

Shaded cells indicate final model chosen
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

6.5.4.2 Young Mania Rating Scale Scores

The model analyzed is as follows:

Young Mania Rating Scale Scores =	Gender (male/female) + Age (years) + Iron (mg) + Zinc (mg) + Psychiatric medication (yes/no) + Age*Iron + Gender*Iron + Age*Zinc + Gender*Zinc

Iron and zinc are included in the model as they were significantly correlated with YMRS scores based on the bivariate analysis. The first model (Model One) considered age and gender as effect modifiers of iron and zinc and these estimates were non-significant. The second model analyzed all independent variables with the interaction terms omitted; none of the regression estimates were of statistical significance. Psychiatric medication was not a confounder. Analysis of diagnostic plots for the final model (Model Two) suggested that all model assumptions were met (all data not shown).

6.5.4.3 Global Assessment of Functioning

The model analyzed is as follows:

Global Assessment of Functioning =	Gender (male/female) + Age (years) + Vitamin C (mg) + Calcium (mg) + Iron (mg) + Phosphorus (mg) + Potassium (mg) + Zinc (mg) + Psychiatric Medication (yes/no) + Age*Iron + Gender*Iron + Age*Zinc + Gender*Zinc + Age*Calcium + Gender*Calcium + Age*Phosphorus + Gender*Phosphorus + Age*Potassium + Gender*Potassium + Age*Vitamin C + Gender*Vitamin C

All analyses had no significant results and psychiatric medications were not a confounder (data not shown). Diagnostics suggested that all assumptions had been met.

6.5.5 Blood Values of Nutrients, Psychiatric Symptoms and Functioning

6.5.5.1 Mood Disorder Symptoms

Various biochemical indicators were analyzed to determine if they predict mood disorder symptoms. Two models were analyzed:

Hamilton Depression Scores =	Gender (male/female) + Age (years) + RBC folate (nmol/L) + Vitamin B_{12} (pmol/L) + Ferritin (mcg/L) + Vitamin E (umol/L) + Albumin (g/L) + Cholesterol (mmol/L) + Psychiatric medication (yes/no) + Age*Folate + Gender*Folate + Age*Vitamin B_{12} + Gender*Vitamin B_{12} + Age*Ferritin + Gender*Ferritin + Age*Vitamin E + Gender*Vitamin E + Age Albumin + Gender + Albumin + Age*Cholesterol + Gender*Cholesterol

Young Mania Rating Scale Scores =	Gender (males/females) + Age (years) + RBC folate (nmol/L) + Vitamin B_{12} (pmol/L) + Ferritin (mcg/L) + Vitamin E (umol/L) + Albumin (g/L) + Cholesterol (mmol/L) + RBC folate (nmol/L) + Vitamin B_{12} (pmol/L) + Ferritin (mcg/L) + Vitamin E (umol/L) + Albumin (g/L) + Cholesterol (mmol/L) + Psychiatric medication (yes/no) + Age*Folate + Gender*Folate + Age*Vitamin B_{12} + Gender*Vitamin B_{12} + Age*Ferritin + Gender*Ferritin + Age*Vitamin E + Gender*Vitamin E + Age*Albumin + Gender*Albumin + Age*Cholesterol + Gender*Cholesterol

Neither model analyzed showed significant results. In both models, psychiatric medications were ruled out as a confounder (data not shown). Diagnostic plots for the final model were analyzed and suggested that all model assumptions had been met.

An analysis was also done with GAF scores as the dependent variable and the various blood values, gender, income, and medications as the independent variables. The results suggested model overfitting (i.e., negative adjusted R^2 values) and therefore not reported.

6.6 Models of Dietary Restraint and Disinhibition, Psychiatric Symptoms and Functioning

To examine relationships among psychiatric symptoms and functioning, three separate models were analyzed using GAF, Ham-D, and YMRS scores as the dependent variable; all independent variables were the same. The following is the model for Hamilton Depression Scores:

Hamilton Depression Scores =	Age (years) + Gender + Education + BMI (kg/m^2) + Restraint Scores + Disinhibition Scores + Age*Restraint Scores + Age*Disinhibition Scores + Gender*Restraint Scores + BMI*Restraint Scores + BMI*Disinhibition Scores

The results of this analysis showed that the interaction of age and restraint scores was a significant predictor of Hamilton Depression scores (regression estimate = -0.378, SE = 0.124, t = -3.05, p = 0.003, 95% CI -0.625 to -0.131) (Table 6.0.20). The same models were analyzed using YMRS and GAF scores and no significant results emerged.

Table 6.0.21: Estimates of energy based on dietary restraint and disinhibition scores	
Adjusted R^2	0.4509
Regression estimates	
Intercept	2.211 (SE = 10.323, t = 0.21, p = 0.831, 95% CI -18.345 to 22.766)
Gender	-5.448 (SE = 4.083, t = -1.33, p = 0.186, 95% CI -13.580 to 2.683)
Age	0.197 (SE = 0.158, t = 1.25, p = 0.216, 95% CI -0.117 to 0.511)
Restraint score	**22.154 (SE = 5.838, t = 3.79, p = 0.000, 95% CI 10.528 to 33.780)**
Disinhibition score	-5.508 (SE = 4.736, t = -1.16, p = 0.248, 95% CI -14.940 to 3.923)
BMI	0.0406 (SE = 0.221, t = 0.18, p = 0.855, 95% CI -0.399 to 0.480)
Education	-1.669 (SE = 0.904, t = -1.85, p = 0.069, 95% CI -3.469 to 0.132)
Gender*Restraint	4.347 (SE = 2.664, t = 1.63, p = 0.107, 95% CI -0.958 to 9.652)
Age*Restraint	**-0.365 (SE = 0.122, t = -2.98, p = 0.004, 95% CI -0.608 to -0.121)**
Gender*Disinhibition	0 .871 (SE = 1.324, t = 0.66, p = 0.513, 95% CI -1.765 to 3.506)
Age*Disinhibition	0.081 (SE = 0.060, t = 1.36, p = 0.178, 95% CI -0.038 to 0.199)
BMI*Restraint	-0.033 (SE = 0.039, t = -0.83, p = 0.412, 95% CI -0.111 to 0.046)
BMI*Disinhibition	-0.048 (SE = 0.101, t = -0.48, p = 0.633, 95% CI -0.249 to 0.153)

Bolded cells represent significant results
SE = standard error
t = t-value
CI = confidence interval
ns = not significant

6.7 Chapter Summary

This chapter reported the results of the bivariate and multivariate analyses that examined nutrient intakes, biochemical indicators, selected sociodemographic and health-related variables, diagnosis, psychiatric symptoms, and psychiatric functioning. Bivariate relationships among food intakes and mood symptoms revealed significant differences only with YMRS scores. Nutrient intakes from food had weak, positive correlations for GAF scores and energy, total fat, carbohydrates, linoleic acid, riboflavin, niacin, folate, vitamin B_6, vitamin B_{12}, pantothenic acid, calcium, magnesium, phosphorus, potassium, iron, and zinc intakes. Interestingly, when food and supplement sources were combined, no relationships were found among the vitamins. However, for minerals, significant negative correlations emerged between YMRS scores with zinc and magnesium as well as Ham-D scores with iron. GAF scores correlated positively with calcium, magnesium, phosphorus, potassium, iron, and zinc. Models of the relationships

among macronutrients showed that age and gender were significant predictors of their intakes. For protein intake, income was also significant.

Investigation of inadequate and excess nutrient intakes from food sources based on EAR and UL values and the diagnoses indicated that the odds of vitamin B_{12} inadequate intake was 0.31 less likely for bipolar disorder than for major depressive disorder. Interestingly, iron showed a dissimilar trend to the overall odds ratio for males, suggesting confounding by gender. Biochemical indicators of blood values showed significant differences for RBC folate between those with and without scores above the cut-off for the Ham-D, and the YMRS scores correlated significantly with ferritin. Models analyzed for RBC folate indicated that dietary folate intake and psychiatric medications were significant predictors. Only total fat intake predicted GAF scores.

When nutrients from food and supplement sources were analyzed, no significant results emerged for Ham-D, YMRS, or GAF scores. There was also a surprising lack of significant results for blood levels of nutrients, symptoms, and overall functioning, possibly because the subsample that submitted to bloodwork was statistically inadequate. When dietary restraint and disinhibition scores were examined, only the interaction of age and restraint scores were significant predictors of Ham-D scores. The following chapter discusses these results and the outcome of the preceding three chapters.

CHAPTER SEVEN: DISCUSSION

7.1 Summary of Findings

The primary aim of this study was to evaluate nutrient intakes and their relationship with mental status in individuals with mood disorders. The first stage of the analysis examined nutrient intakes from food and supplemental sources as well as selected biochemical indicators of nutrient status and addressed the research queries:

1) Compared to the *Dietary Reference Intakes (DRIs)*, was there evidence of inadequate (i.e., less than the Estimated Average Requirement or Adequate Macronutrient Distribution Ranges) and/or excess (i.e., greater than the Tolerable Upper Intake Levels or Adequate Macronutrient Distribution Ranges) nutrient intakes?
2) Did nutrient intakes differ from those in a general population sample?
3) Did blood levels of selected nutrients occur outside the reference ranges?

In the second stage, bivariate analyses were conducted to examine the research query regarding whether nutrient intake levels in the study sample differed significantly according to specific sociodemographic, mental health status and health-related variables. Analysis for significant associations among nutrient and biochemical data with socio-demographic, health-related factors and mental status measures were conducted.
The final stage of the analysis examined predictors of macronutrient intakes, selected biochemical parameters, and mental status and addressed the following research queries:

1) What sociodemographic, mental health status and health-related variables were associated with energy and macronutrient intakes?
2) What nutrients, sociodemographic, and health-related variables were associated with mental health status?
3) What nutrient intake, sociodemographic, mental health status and health-related variables were associated with blood levels of selected nutrients?

As hypothesized, results from the stage one analyses revealed concerns about potential nutrient inadequacies and excesses. Many participants did not meet the minimum recommendations for grains, vegetables and fruit, and meat and alternatives as recommended in *Eating Well with Canada's Food Guide*, and their nutrient data reflected this. The study sample had generally higher rates of micronutrient inadequacies than the

sample analyzed in the British Columbia Nutrition Survey (BCNS). Based on nutrient intakes from food alone, a substantial percentage of the sample had inadequate intakes of fibre, α-linolenic and linoleic acid, thiamin, riboflavin, niacin, folate, vitamin B_{12}, vitamin B_6, vitamin C, pantothenic acid, calcium, magnesium, potassium, iron, phosphorus, and zinc. With the exception of phosphorus, all these nutrients have well-established roles related to brain function, thus their inadequate levels present unique relevance in this population. Phosphorus has not been specifically studied in relation to mental health. However, as it is a primary component of adenosine triphosphate (ATP), activating and deactivating enzymes, and a component of genetic material in the nuclei of cells (including both DNA and RNA) and of cell membranes and lipoproteins[90], it would have relevance in this population and warrants further study.

The data indicating nutrient inadequacies were also consistent with quantitative measures of food insecurity. Compared to provincial nutrition data, all food security indicators measured (i.e., anxiety over enough to eat, did not have enough food to eat and accessing food assistance programs) were substantially higher. Food insecurity has been linked to a multitude of negative health conditions including heart disease, obesity, high blood pressure, and diabetes[242;243]. Food insecurity also has psychological effects; contributing to higher levels of stress, anxiety, irritability, social isolation, heightened emotional responsiveness, impaired cognitive abilities, eating disorders, and depression[241;323-325]. While quantitative measurement tools used in this study helped to determine food insecurity among study respondents, further research using qualitative methods (e.g., in-depth interviews, focus groups) that examine individual and household experiences of food insecurity will help to develop a more nuanced understanding of the wide-ranging effects, experiences and management of food insecurity in this population.

Another concern with the dietary micronutrient intakes was the consumption of sodium well beyond the recommended upper levels coupled with suboptimal intakes of potassium. Na(+), K(+)-ATPase which can be affected by dietary intakes of sodium and potassium is believed to be involved in the pathogenesis of mood disorders[182]. In addition, dietary electrolytes (e.g., sodium, potassium) may alter cortisol secretion which in turn can affect mood state[189;190].

The third hypothesis of this study indicated that there would be biochemical evidence of nutritional deficiencies. When nutrient status was measured in the blood, a small proportion of males showed low levels of RBC folate. For a small proportion of females, low blood levels of vitamin B_{12} and ferritin were also a concern. Although it was anticipated that even a larger number of biochemical values would have been suboptimal in this population, even these few results are of concern as suboptimal levels of these nutrients impair cognitive function and affect responses to psychiatric interventions. The energy and macronutrient intake results were important. The low energy levels reported are questionable, since more than half of the sample was classified as being overweight or obese. This high prevalence of overweight and obesity indicates that energy balance is a major health problem in this population. Because there was slight under-reporting of energy intakes in some groups, nutrient intakes may also have been slightly under-reported and thus overestimated potential nutritional deficiencies.

However, it is also important to note that this sample generally did not include the most nutritionally vulnerable individuals with mood disorders (e.g., homeless, those with acute illness requiring hospitalization). Inclusion of these individuals would likely have increased prevalence estimates of nutritional inadequacies. The findings related to possible under-reporting of intakes support the need for future research to obtain biological confirmation of apparent dietary inadequacy.

Most participants were within the Acceptable Macronutrient Distribution Ranges (AMDRs) for carbohydrates, total fat, and protein. However, despite being within the acceptable range for total fat, about half were consuming more than 35% of their energy from fat sources, which is considerably higher than general population estimates of 25% of energy from fat sources. These results relate to the food group data suggesting a low intake of fruits and vegetables and fibre. A diet high in total fat reduces hippocampal levels of brain-derived neurotrophic factor, a crucial modulator of synaptic plasticity[326]. Most participants knew about *Eating Well with Canada's Food Guide*, but only one-third of the sample reported using it. This, in combination with participants' fat and food intake data, suggests a need for nutrition education in this population.

While 65 participants (67%) were within the AMDRs for carbohydrates, 27 (28%) fell below the lower bound, with less than 45% of their consumed energy coming from

this macronutrient. Of the 27 who consumed less than the minimum AMDR for carbohydrates, two participants (2% of the total sample) consumed less than the Estimated Average Requirement (EAR) of 100 grams/day and were the focus of particular concern. The EAR level is based on the amount of carbohydrate needed to produce enough glucose to carry out essential brain activities and therefore has significant implications for psychiatric functioning within this population.

Four participants had suboptimal intakes of protein (i.e., less than 10% of total energy intake). Important amino acids such as tryptophan and other neurotransmitter precursors may therefore be lacking in the diets of selected individuals in this population and therefore limits their psychiatric functioning.

The main concern with the macronutrient data was the low intakes of the omega-3 and omega-6 fatty acids. There is substantial indirect and direct evidence of the importance of omega-3 fatty acids in the pathophysiology and treatment of mood disorders. Furthermore, the evidence suggests that an imbalance in the ratio between the omega-6 and omega-3 fatty acids and/or deficiency of omega-3 fatty acids may be responsible for heightened depressive symptoms.

Of particular interest was that over two-thirds of participants reported taking nutritional or non-nutritional supplements. The patterns of self-prescription of supplements in this study is consistent with other findings in individuals with mental disorders[327]. For all groups where supplements were taken, the proportion was consistently higher when compared to the BCNS. Several significant differences emerged among those of both genders aged 31 to 50 years, and to a lesser extent among females aged 51 to 70 years. The major supplements taken included micronutrient combinations, vitamin C, vitamin E, and various natural and herbal remedies.

The nutritional supplement intake data has many important implications in this population. First, the addition of selected supplements to food intakes contributed to decreasing the proportion of inadequacy for several nutrients. Second, supplemental intakes of various minerals correlated significantly with overall psychological functioning. These results lend support to the evidence that the decreasing mineral content of our food supply[4] may have important implications for individuals with mental disorders who may be more sensitive to nutritional depletions[328]. Furthermore, the effects

of these minerals on psychological functioning suggest that the use of multi-ingredient mineral formulas in the treatment of mood disorders should continue to be explored.

However, supplemental nutrient intakes also contributed to excessive levels of some nutrients when compared to the Tolerable Upper Intake Levels (ULs). A substantial proportion of participants exceeded the ULs for folate, niacin, vitamin B_6, vitamin C, calcium, iron, magnesium, zinc, and manganese. The ULs are defined as "the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects for almost all apparently healthy individuals in the specified life-stage group. As intake increases above the UL, the potential risk of adverse effects may increase"[329]. Thus, many participants of this study may be at unnecessary risk due to excess nutrient intakes (e.g., high doses of vitamin B_6 can cause neurological damage). Table A.0.3 (Appendix A) outlines the nutrients in this study that were consumed excessively (i.e., above the UL) and the concerns associated with them. Another important consideration is that most study participants had concurrent medical conditions where supplemental intake may also be contraindicated (e.g., cancer, diabetes). However, it must be noted that the *DRI*s are based on the healthy general population and may have limited applicability in populations with mental disorders. For example, lithium in pharmacological doses would certainly be contraindicated in healthy populations, but in individuals with bipolar disorder these higher amounts are needed for mood stability.

Excessive intakes were confirmed in some of the biochemical data. A substantial proportion of both males and females had levels that exceeded the normal ranges for vitamin B_{12} and vitamin E (after controlling for cholesterol). Some males were also over the normal ranges for ferritin and albumin. While the efficacy of dietary and herbal supplements in mental health care is a matter of debate, the potential to produce adverse reactions and drug interactions is certain. Given this reality and the results of this study, mental health care providers should become knowledgeable about the role of nutrients in the brain so that they can appropriately advise their patients on the use of these complementary therapies in combination with conventional treatments. Furthermore, future research on supplements needs to explore the underlying factors contributing to supplement use in this population and to focus on its efficacy and long-term safety.

Finally, the initial analysis of the data showed that most of the participants were overweight or obese. Excess weight in those with mental illnesses has significant consequences. Recent studies have shown that those with bipolar disorder who are also obese have a poor health-related quality of life[330]. The presence of obesity among individuals with mood disorders has implications for prevention and management of weight gain, particularly weight gain associated with specific psychiatric medications.

The second and third stages of the analysis addressed the final two hypotheses of this study and revealed two key findings. First, when examining attitudes about eating and the body, many important results were indicated. The study sample had significantly more people reporting eating attitudes and behaviours reflective of cognitive dietary restraint and disinhibition compared to those studied in the BCNS. Of particular concern was the significant association of cognitive dietary restraint and age with Hamilton Depression Scores. Cognitive dietary restraint refers to the perception of constantly monitoring and attempting to limit dietary intake in an effort to achieve or maintain a certain body weight[288]. Restrained eaters are characterized by concern about their eating, weight and appearance as well as a variety of cognitive and affective attributes, including low self-esteem and negative body image[263].

Although high dietary restraint appears innocuous, it creates chronic psychological stress that activates the hypothalamic–pituitary–adrenal (HPA) axis and leads to increased release of the stress hormone cortisol[331]. Over time, elevated cortisol has detrimental effects on many body systems, including mood stability[332], calcium homeostasis, and bone metabolism[333]. For example, one study of healthy women revealed that both low dietary intake of omega-3 fatty acids and higher levels of body dissatisfaction together were significantly associated with severity of depression[334]. Prospective investigations are warranted to confirm HPA activity and to explore health implications because the directions of relationships between nutrients, dieting behaviours, and psychiatric functioning are unclear. For example, does food restriction lead to inadequate nutrients and, hence, an alteration in metabolic functions that contribute to illness? Or are the psychological and neurochemical ramifications of dieting separate from biochemical changes attributed to dietary inadequacies sufficient to enhance susceptibility to psychiatric symptoms? It is likely these relationships between food

restriction and psychiatric symptoms have interacting pathways that create a multifactorial model of dieting-induced negative mood state.

Another key finding of the bivariate analyses was the lack of relationship between nutrient intakes and some measures of psychiatric functioning. This finding did not support this study's original hypothesis that there would be differences in mental status variables according to nutrient intakes. Surprisingly, such relationships appeared to be non-existent between nutrient intakes from food and depressive and manic symptoms. This may be due to the fact that the sample selected generally had few participants with severe symptoms. Conversely, there were several weak significant correlations among the macronutrients (total fat, carbohydrate and linoleic acid) micronutrients (6 vitamins and 6 minerals) and Global Assessment of Functioning (GAF) scores, suggesting that better psychological function was associated with improved overall micronutrient intake from food. The variability in the findings may confirm that there are many unifying common pathogenic pathways in mood disorders, which may introduce new targets for the development of therapeutic interventions. These relationships weakened for vitamin intakes when supplements were added. However, when considering food combined with supplement intakes of minerals, significant negative correlations were found YMRS scores with magnesium and zinc as well Ham-D scores and iron. Overall psychological functioning positively correlated with calcium, magnesium, phosphorus, potassium, iron, and zinc. These results suggest that mineral intakes were more closely associated with psychiatric symptoms and functioning than were vitamins. In particular, supplemental mineral intakes may have affected psychiatric symptoms and functioning by correcting potential deficiencies. Theories that link nutrients and mental health indicate that coenzyme systems in those with mood disorders require higher levels of selected minerals to function optimally.

The relationships between mental status and nutrient intakes also weakened when linear regression analysis was conducted. It showed significant results only for total fat intake from food and GAF scores. When nutrients from food and supplement sources were combined, no significant results emerged for depression, mania or global functioning. What was also surprising was a lack of significant results for blood levels of nutrients, mood disorder symptoms, and overall functioning. These results therefore did

not fully support the final hypothesis of this study, that a set of nutrient variables would predict mental status in this population. However, the lack of relationships found among the variables such as biochemical indicators and mental status may be due to the fact that the subsample of 50 participants who provided blood samples was statistically inadequate. In addition, the subsample of individuals who provided blood samples was self-selected and therefore possibly biased these results.

7.2 Strengths and Limitations of Study

The strengths of this study include its use of a relatively large sample of people with confirmed mood disorders within a community setting. Previous studies on nutrition and mood disorders typically had much smaller samples, did not consistently employ methods to ensure verification of diagnosis, and were typically conducted in institutional settings. Other key strengths of this study are that it examined food intakes in comparison to the *DRI*s and *Eating Well with Canada's Food Guide* as well as the separate effects of nutrients from food alone as well as food plus supplements in relation to mental status. The author was unable to locate any other studies conducted to date that have examined nutrient intakes with these dietary standards in this population or the associations of nutrients from food and supplement sources and their relation to mood disorders.

Traditionally, most studies have focused on supplemental intakes from single sources as treatments in mood disorders. Furthermore, many of the clinical trials conducted on specific nutrients have failed to control for intakes from food sources. However, the results of this study suggest that intakes are associated with overall functioning and thus should be measured and controlled for in these experimental designs. This study allowed for comprehensive data collection on nutrition habits, including individual determinants of food intake in this particular population. Finally, this study allowed for comparisons of nutrition data with psychiatric diagnosis, symptoms, and functioning.

However, the study also has some limitations. First, the findings from the survey, particularly the food frequency questionnaire, were subject to recall bias. Furthermore, the modest proportion of males in the sample may also have limited generalizability. Future work in this area should consider using some type of purposeful sampling to ensure that all age and gender groups are represented. However, the membership of the Mood Disorders Association of British Columbia from which the sample was drawn is

small and therefore this proposed sampling method is unlikely to be feasible. The recruitment method did not attempt to ensure representative samples of the groups under study because this was not considered strictly necessary in order to test hypotheses relating food intakes to mental status. Although this study compared study respondents to a general sample, it is important to note that it is not a direct comparative study. One major difference between the two was the lack of respondents over 70 years who could be compared to the same subgroup of the BCNS. Another major difference is that the BCNS was based on one-day intakes and the survey was administered in people's homes; this study examined usual intakes based on three-day intakes and was conducted in an office. In addition, some variables could not be directly compared. For example, the BCNS examined metabolic equivalents (METS) in exercise whereas this study did not.

There are also limitations on collecting dietary information from three-day food records: the process of recording foods may affect the foods eaten; people may alter their intake during the recording period as they may be more conscious of their choices; people may under-report less desirable foods and over-report more nutritious foods; and subjects require a high literacy level. However, three-day food records require fewer people for a study than 24-hour diet recalls and have a low respondent burden.

There were limitations associated with the use of the EARs of the *DRI*s as a standard by which to measure the adequacy of nutrient intake. The age/gender subsamples examined were much smaller than what is required for adequate dietary assessment of a population and therefore limited inferences could be made in this regard. However, few other analytic options were available to utilize in this particular study. The EARs focus on determining the lowest intake of a particular nutrient that one can survive on without developing explicit symptoms or disease. It is difficult to understand the rationale for this approach, but it may partly be due to the fact that there is a bias toward intake recommendations close to intakes that prevail in the population currently and, for at least some nutrients, intakes that should be readily achievable with currently available foods, if only better choices were made[335]. Thus, the use of the EARs to assess inadequacy in individuals with mood disorders may have limited applicability as they may have heightened nutrition needs based on a variety of factors (e.g., medication use that can

interfere with nutrient metabolism). Nonetheless, the EAR does provide a conservative estimate of the magnitude of inadequacy in this study population.

Similarly, the use of the AMDRs of the *DRI*s in this population may also have limited applicability. The AMDRs indicate ideal ranges for each of the macronutrients, but they do not always indicate the choices to make within each group to optimize function. For example, one could be considered to be consuming adequate protein levels while being suboptimal in selected neurotransmitter precursors such as the amino acid tryptophan.

As previously mentioned, comparison to the ULs of the *DRI*s may have little relevance as those with mood disorders may have greater needs than those of the healthy population to optimize psychiatric function. Furthermore, while exceeding the ULs may pose a potential health risk, one must consider whether such risks have a more favourable risk-benefit ratio in the treatment of mood disorders compared to contemporary psychotropic agents. Many psychiatric medications carry significant risk for adverse events (e.g., higher risk of obesity, type 2 diabetes mellitus, cardiovascular conditions, dyslipidemia, orthostatic hypotension). Most nutrient supplements do not carry such significant risks. Because the development of safe, effective treatments to which individuals with mood disorders will adhere is critical, the use of the *DRI*s in this population may require further consideration.

As outlined in Chapter One, at least 22 micronutrients are related to brain function. Furthermore, other important nutrition components, such as the amino acids and phytochemicals found in food, are also associated with cognition. Based on the limitations of existing computerized nutrient analysis systems, this investigation could examine only 15 micronutrients. Therefore, there are still questions about the effects of other nutrients and bioactive substances found in food in relation to psychiatric function. The results of this study indicate that nutrition is important in mental health, particularly overall functioning. These findings suggest that measures of nutrition-related outcomes in this population should include broad-based indices of mental outcome. The closest one used in this study was the GAF scale. Similarly, using broader measures of nutrient-related data such as correlations of food groupings to psychiatric measures could yield different findings to those of the specific nutrients.

While the majority of participants were at least overweight, it is important to note that there are some limitations with using BMI. First, research indicates that the cut-offs may vary for different cultural groups. This possible difference was not accounted for in this study or in the BCNS. Second, BMI is best measured with waist circumference to assess for central adiposity. The BCNS collected waist and hip circumference data; however, for this study it was decided to exclude this anthropometric measure as it is relatively invasive and was not part of the main hypotheses.

While dietary cognitive restraint appeared to have causal links with food intakes and depression scores, it is important to note that the questions used were only a subsample of the Three-Factor Eating Questionnaire. Future research in this area should use the complete questionnaire in order to more fully understand the relationships with psychiatric symptoms and functioning and dietary cognitive restraint.

The study design was subject to a number of vulnerabilities. First, a cross-sectional study does not offer evidence of temporal relationships. Secondly, this investigation is also subject to selection bias. Participants were drawn from an urban sample, and they were members of an organization providing support for those with mood disorders. It is therefore not representative of the population of all possible participants. This selection bias may have particularly overestimated food insecurity in this population as British Columbia has the highest poverty rate for all individuals and families in Canada[336]. Misclassification bias may have emerged with the use of the *DRIs* to classify subjects both in this study and the BCNS according to nutrient inadequacy and excesses due to indications of under-reporting of nutrient intakes. Finally, most measures used in this study are believed to be subject to non-differential measurement error, resulting in a bias towards the null. This particular bias however, would not alter the conclusions.

The minimal knowledge that exists between nutrition and mental status in this population also contributed to the limitations of this study. For example, while the sample size calculation was based on a parameter (i.e., carbohydrate intakes) of relevance in this population, it was inadequate to examine some important relationships, particularly those based on multivariate analysis. Because little is known about potential intervening variables that affect the relationships between nutrition and mental status, issues such as confounding may have emerged despite the many measures instituted to control this.

Analyses of potential interacting variables may have also been missed. With the numerous statistical tests conducted in this study, particularly on the various nutrient intake measures, it is likely that Type I errors occurred. While means to control this were incorporated (e.g., use of Bonferroni adjustments), complete control of this error was likely not feasible. Although this design is described as cross-sectional, limitations such as the relatively small sample size, the limited sample to variable ratios and the many statistical tests may suggest that it instead should be referred to as an exploratory study.

While this study had limitations, it is important to note that it allowed for quantified comparisons of nutrient intakes between mood disorder and general populations, which have never been available prior to this time. This investigation has also determined the prevalence of various nutrition-related factors in a mood disorder population and is therefore useful for future hypothesis generation and planning of health services.

7.3 Future Research Directions

The results of this study suggest many potential avenues for future nutrition research in mood disorders. Specifically, direct comparison studies such as case-control or prospective designs using larger sample sizes would provide further evidence about the relationships between longer-term psychiatric functioning and nutrient intakes. In addition, future studies should include factors such as waist-to-hip ratio, exercise, and measures of other nutrients (vitamin K, vitamin E, boron, betaine, etc.).

The data linking mineral intakes from food and supplement sources and mental status warrants further investigation. Many participants ingest many supplements, so it may also be worth examining their combined effects (e.g., combinations of nutrients with various psychiatric medications) on psychiatric outcomes. Similarly, future studies should attempt to examine specific food groupings and other bioactive compounds in foods and their relationships to mental functioning.

The data on cognitive restraint and disinhibition suggests that future investigations should examine possible associations with psychiatric functioning. In particular, examinations of mechanisms related to cortisol, stress, cognitive dietary restraint, and psychiatric outcomes may provide information for future nutrition education in this population. It may also be worthwhile to investigate relationships of cognitive restraint and disinhibition with food insecurity.

Although biochemical indicators were examined in this study, only a limited self-selected number were measured. Future work in this area should consider other measures such as the essential fats and trace minerals, using larger randomly selected samples to evaluate relationships between nutrients and psychiatric functioning.

As previously cited multiple supplement use in this population was evident from the results of this study. Future investigations should examine the potential impact of supplemental use on mental and physical health outcomes in this population. It would be useful to determine the underlying factors associated with taking these supplements and to examine how this population could be educated effectively about their safe use.

The correlations between the global functioning and nutrient intakes suggest that the study of outcomes in relation to nutritional factors should be broadened. With the high rate of comorbidities in this population, parameters related to quality of life could be considered as well as physical health outcomes. Furthermore, other individual (e.g., self-esteem) and collective determinants (e.g., interpersonal, physical, economic, and social environments) determinants of food and their complex, interacting relationships should be examined in this population. This would further understanding about what determines food intake in this population and ideally lead to interventions that could induce people to undertake healthy eating behaviours.

Age, gender, and income were clearly significant predictors of nutrient intakes in this population. Future studies need to consider these important demographic variables and their effects on nutritional status. For example, income level is closely linked with food insecurity, which was an issue for a large proportion of participants. Future research could be aimed at in-depth qualitative analysis to help understand the psychological, physiological, and emotional consequences associated with food insecurity. Furthermore interventions aimed at increasing individual food security in this population could be examined to determine their potential for benefiting mental health outcomes.

7.4 Conclusions and Relevance to Practice

This study has established that this population (i.e., community dwelling adults with mood disorders) has many nutritional risk factors. They tend to be more overweight than the general population and food insecurity is a major issue. Intakes of several nutrients raised concern: suboptimal fibre, α-linolenic and linoleic acid, calcium, thiamin, niacin,

riboflavin, folate, pantothenic acid, vitamins B_6, B_{12} and C, calcium, magnesium, zinc, potassium, phosphorus, iron (for premenopausal women), and excess total and saturated fat. Supplement use from multiple sources was high, taking many nutrients beyond the ULs. The biochemical data also showed some evidence of excess nutrient intakes. This study provides an important foundation for building policy and programs. In particular, the food group and nutrient data contribute to our understanding of dietary inadequacies and excesses observed in this population. However, there is still considerable progress to be made in nutrition screening and intervention. While dietary intake data are useful, including physical and biochemical indices in a comprehensive nutrition monitoring system will enhance the utility of the intake data.

Dietary advice for those with mood disorders should emphasize the importance of a varied and balanced diet, especially sufficient omega-3 fatty acids. Dietary interventions must take into account that extremely low-fat diets, generally prescribed for their disease-preventing effects, tend to replace saturated fat with omega-6 fatty acids, thereby increasing the ratio of omega-6 to omega-3 fatty acids in the diet. A disproportionate ratio of omega-6 to omega-3 fatty acids may potentiate a relative deficiency in omega-3 fatty acids in tissues, organs, and neuronal membranes and thus contribute to neuro-physiological alterations conducive to depression. Furthermore, the data obtained in this study suggests that those with mood disorders are not physically active enough to attain the fitness levels needed to reduce the health risk associated with excess body weight and to benefit from the mood enhancement of physical activity. Individualized treatment environments are key to enabling individuals to develop and maintain healthy lifestyles, thus enhancing their overall mental and physical functioning.

People with mood disorders have a greater frequency of poor diet for several reasons. Foremost is the potential for depressive episodes to exacerbate a sedentary lifestyle associated with lack of exercise, weight gain, and cardiovascular disease risk. Moreover, manic episodes may be associated with treatment non-adherence. Other factors such as greater medical comorbidity or substance use may also explain the association between mood disorders and poor nutrition status. Finally, as this study revealed, intervening psychological factors affecting food choice may impact food intakes. Exacerbating these risk factors is the fact that individuals with mood disorders are also less likely to report

that their provider discussed diet habits with them. Greater efforts are needed to engage providers in discussing better health habits with those diagnosed with a mood disorder.

This research has added support to the idea that the relationship between mood disorders and nutrient intakes is an important one. It is evident that in the treatment of those with mood disorders, factors of importance include the quantity, relative sufficiency, and the quality of food, the potential psychological barriers affecting food intake as well as the social and environmental contexts in which food choices are made. This research project provides insight regarding the complex nature of nutrition and mental status in individuals with mood disorders. It also identifies some of the strata of data collection and analysis that need to be considered in mental health and nutrition investigations. It is evident that further understanding about the complexities of nutrition and mental status will require systems approaches that can identify core elements, explain the role of each element and their dynamic interactions, and clarify the environment within which a system operates. Such an approach will ensure that holistic strategies are developed and will establish a framework for assessing the impact and effectiveness of strategies and policies prior to and during implementation.

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APPENDIX A: *DIETARY REFERENCE INTAKES*

Table A.0.1: Components of the *Dietary Reference Intakes*

Standard	Definition	Use in Planning for Groups
Estimated Average Requirement (EAR)	A nutrient intake value that is estimated to meet the requirements of half of the healthy individuals in a group. It is used to assess adequacy of intake of population groups and, along with knowledge of the distribution of requirements, to develop RDAs.	Plan for an acceptably low proportion of a group with intakes below the EAR.
Recommended Dietary Allowance (RDA)	The average daily dietary intake level that is sufficient to meet the nutrient requirements of nearly all (98%) healthy individuals in a group. A target for individuals to ensure enough intake.	Do not use the RDA to plan mean intakes for groups. Mean intake at the RDA may lead to an unacceptably high prevalence of inadequate intakes.
Adequate Intake (AI)	A recommended daily intake level based on observed or experimentally determined approximations by a group (or groups) of healthy people.	Median usual intake at or above the AI implies a low prevalence of inadequate intake. The AI should be used with less confidence if it has not been established as a median intake of a healthy group.
Tolerable Upper Intake Level (UL)	A recommended daily nutrient intake that is likely to pose no risks of adverse health effects to almost all individuals in the general population. As intake increases above the UL, the risk of adverse effects increases.	Plan to minimize the proportion of a group with intakes above the UL to minimize potential risk of adverse effects of excessive intake.

Table A.0.2: Criteria Used To Establish *Dietary Reference Intakes* for Adult, Non-pregnant, Non-lactating Population

Nutrient	Criterion EAR	Criterion AI	Criterion UL
Carbohydrate	Brain glucose need	-----	-----
α-Linolenic Acid and Linoleic Acid	-----	Median intake from Continuing Food Survey II	-----
Protein	Nitrogen equilibrium	-----	-----
Folate	Intake needed to maintain red blood cell folate levels	-----	Applied to synthetic forms and based on case reports of progression of neurological effects in vitamin B_{12} deficient patients taking folate supplements
Niacin	Relates intake to urinary excretion of niacin metabolites	-----	Based on the adverse affect of flushing
Pantothenic Acid	-----	Intake sufficient to replace urinary output	-----
Riboflavin	Indicators include urinary output of riboflavin and its metabolites, blood levels, and erythro-cyte glutathione reduc-tase activity coefficient	-----	-----
Thiamin	Normal erythrocyte transketolase activity	-----	-----

Table A.0.2: Criteria Used To Establish *Dietary Reference Intakes* for Adult, Non-pregnant, Non-lactating Population /continued			
Nutrient	**Criterion EAR**	**Criterion AI**	**Criterion UL**
Vitamin B_6	Maintenance of plasma pyridoxal phosphate levels	-----	Adverse affect of sensory neuropathy
Vitamin B_{12}	Level needed for maintenance of hematological status	-----	-----
Vitamin C	Ages 19 to 30 years: Near maximal neutrophil concentration. Ages 31+: extrapolation of near maximal neutrophil concentrations from 19 to 30 years.	-----	Applies to intakes from food and supplements and based on adverse effect of osmotic diarrhea
Iron	Factorial modelling	-----	Gastrointestinal distress
Magnesium	Ages 19 to 70 years: Balance studies Ages 71 plus: Intracellular studies/decreases in absorption	-----	Based on pharmacological sources only
Phosphorus	Ages 19 to 50 years: Serum inorganic phos-phate levels. Ages 51+: Extrapolation of serum inorganic phosphate levels from 19 to 50 years	-----	Based on the effects of hyperphosphatemia
Zinc	Factorial approach-minimal quantity of absorbed zinc adequate to replace endogenous losses	-----	Reduction in erythrocyte copper-zinc superoxide dismutase activity

Table A.0.2: Criteria Used To Establish *Dietary Reference Intakes* for Adult, Non-pregnant, Non-lactating Population /continued			
Nutrient	**Criterion EAR**	**Criterion AI**	**Criterion UL**
Calcium	-----	Ages 19 to 30 years: Desirable calcium retention/factorial. Ages 31 to 50 years: Calcium balance Ages 51 to 70 years: Desirable calcium retention/factorial/change in bone mineral density Ages 71 plus: Extrapolation of desirable calcium retention from 51 to 70/change in bone mineral density/fracture rate	-----
Fibre	-----	Based on the greatest reduction in risk for coronary heart disease and set at 14 g/1000 kcal x median energy intake (kcal/1000 kcal/day) for each age and gender group	-----

Table A.0.3: Critical Adverse Effects of Selected Nutrients With Tolerable Upper Intake Levels[303]	
Nutrient (UL)	**Critical Adverse Effect**
Vitamin A (pre-formed) (3000 mcg)	Liver abnormalities. For women of childbearing age is teratogenic. Other effects include nausea, vomiting, headache, increased cerebrospinal fluid pressure, vertigo, blurred vision, muscular incoordination, nervous system changes, and bone and skin abnormalities.
Vitamin D (50 mcg)	Hypercalcemia. Other effects include anorexia, nausea, vomiting, increased thirst and urination, metastatic calcification of soft tissues (kidneys, blood vessels, heart, lungs), and renal disorders.
Vitamin E (1000 mg α-tocopherol)	Increased tendency to hemorrhage. Adults deficient in vitamin K, including those taking coumarin drugs, have increased risk of coagulation defects.
Vitamin C (2000 mg)	Osmotic diarrhea and gastrointestinal disturbances. Other effects include increased oxalate excretion and kidney stone formation, uric acid excretion, pro-oxidant effects, rebound scurvy, increased iron absorption/iron overload, reduced vitamin B_{12} and copper status, increased oxygen demand, and erosion of dental enamel.
Niacin (35 mg)	Vasodilation (flushing). Gastrointestinal effects for those treated with nicotinic acid. Hepatic toxicity has been reported in patients treated medically with the vitamin.
Vitamin B_6 (100 mg)	Neuropathy.
Folate (1000 mcg)	Precipitate or exacerbate the neurological damage of vitamin B_{12} deficiency.
Choline (3.5 g)	Hypotension, fishy body odour, nausea and diarrhea.
Calcium (2.5 g)	Kidney stone formation or milk-alkali syndrome (hypercalcemia and renal insufficiency). Also affects iron, zinc, magnesium, and phosphorus absorption
Phosphorus (3 or 4 g)	Hyperphosphatemia. Other effects include hypocalcemia, adjusted calcium-regulating hormones, and calcification of nonskeletal tissues (especially kidneys).
Magnesium (350 mg)	Osmotic diarrhea, nausea, abdominal cramping, serious neurological and cardiac symptoms, and death.
Selenium (400 mcg)	Hair and nail brittleness/loss, gastrointestinal disturbances, skin rash, garlic breath, fatigue, irritability, and nervous system disorders.
Iron (45 mg)	Gastrointestinal side effects, impaired zinc absorption, increased risk for vascular disease and cancer, and iron overload.
Copper (10 mg)	Liver damage. Other effects include abdominal pain, cramps, nausea, diarrhea, and vomiting.
Zinc (40 mg)	Influences copper metabolism, epigastric pain, nausea, vomiting, abdominal cramps, diarrhea, headaches, and immune response impairment.
Manganese (11 mg)	Elevated blood magnesium concentration and neurotoxicity.

Table adapted from Davison KM, Dominik B (2009). *Audits and More: A Nutrition and Food Service Audit Manual for Adult Residential Care Facilities with 25 or more Persons in Care*. Victoria: Crown Publications[337]

APPENDIX B: STUDY FORMS

LETTER OF INTRODUCTION

Mood Disorders Association of British Columbia
Suite 201, 2730 Commercial Drive, Vancouver, B.C. V5N 5P4
Phone: 604-873-0103 Fax: 604-873-3095
Email: mdabc@telus.net Website: www.mdabc.ca

Date

{Member address}

Dear _____:

I am writing to some of our members about an interesting study being conducted through our office and sponsored by the University of Calgary: *researchers are exploring the nutritional status of individuals who suffer from mood disorders* as a means to develop strategies for treatment.

Your name has been selected randomly from the MDABC membership list, which is why you are receiving this letter. While participation in the study is voluntary, we want to encourage you to consider it seriously for several reasons. The information collected will help plan nutrition programs and services for individuals with mood disorders. It will also help us to explore whether the nutritional risk factors that occur are due to diet or not.

Most people find it interesting to participate in research. If you participate, you will complete a food record, listing all foods and beverages you consume over 3 days. The food record will be mailed to you and a registered dietitian will phone you to explain how to complete it. You may also be asked to provide a small sample of blood to assess your nutrient status. Then you will be asked to come to the MDABC office in Vancouver for an appointment with an interviewer and a registered dietitian. The interview (roughly 90 minutes) will include questions about your current mental health and your diet. Also, your weight and height will be measured.

..2

We realize that you may feel that this study expects a lot from you. You will be offered a $50 honourarium for your time, travel and parking expenses. If traveling to the MDABC office is not feasible, then perhaps a home interview can be arranged. Finally, if you would like to know your individual results, the research coordinator can review those with you.

All information that you provide will be confidential. You may refuse to answer any specific questions or to have blood or body measurements taken. Also, if you agree to participate and then change your mind, you may withdraw from the study at any time. Participation in research is always voluntary, and your relationship with the MDABC will not be harmed in any way if you decide not to do this.

If you have any questions or if you want to call to volunteer, please call Karen Davison, Research Coordinator at [redacted]. Otherwise, in a few days someone from our office will phone you to ask whether it's okay for us to give Karen your name and number. She would like to phone you to describe the study and see if you want to participate. Your participation is very important to us and to the future of mood disorder treatments. The results of this study will help develop strategies that will enhance the quality of life of individuals who suffer from mood disorders. Your help is most appreciated.

Sincerely,

Ed Rogers, President
Mood Disorders Association of British Columbia

NON-RESPONSE QUESTIONNAIRE

ID # | | | |

To determine if people who say yes to the study are different than people who say no, I would like to ask you 5 questions about your eating habits and background.

1.During the past month, did you eat bread?

1 ☐ Yes
2 ☐ No

⇒**If yes**, what type of bread did you usually eat? (Do NOT read list. Check (√) only one.)

1 ☐ Whole wheat (100%, 60%), multigrain/cracked wheat, rye or pumpernickel
2 ☐ White bread
3 ☐ Other: _____
4 ☐ Don't know

2.During the past month, did you use milk?

1 ☐ Yes
2 ☐ No

⇒**If yes**, what type of milk did you usually use? (Do NOT read list, Check (√) only one.)

1 ☐ Whole milk
2 ☐ 2% milk
3 ☐ 1% milk
4 ☐ Skim milk
5 ☐ Powdered milk
6 ☐ Evaporated milk
7 ☐ Other: _____
8 ☐ Don't know

3.During the past month, did you take any vitamin-mineral supplement(s)?

1 ☐ Yes
2 ☐ No

4.Do you smoke?

1 ☐ Yes
2 ☐ No

5.What is your current marital status? Are you: (read list)

1 ☐ Married or *Living common law
2 ☐ Divorced
3 ☐ Separated
4 ☐ Widowed
5 ☐ Never married

*Common law is defined as adults of the opposite sex or same sex living together in a sexual union of 3 months or more duration.– Statistics Canada

6.What is the highest level of education that you received:

1 ☐ No schooling
2 ☐ Some elementary or grade school
3 ☐ Completed elementary or grade school
4 ☐ Some secondary or high school
5 ☐ Completed secondary or grade school
6 ☐ Some trade/vocational training
7 ☐ Completed trade/vocational training
8 ☐ Some university
9 ☐ Certificate or diploma below bachelor's level
10 ☐ Bachelor's degree or above

These are examples of the study questions, would you like to reconsider participation in the study?

Thank you for your time.

INFORMED CONSENT FORM

[On University of Calgary, Department of Community Health Sciences Letterhead]

PARTICIPANT INFORMATION AND INFORMED CONSENT

TITLE: Determinants of Food Choice in Individuals with Mood Disorders

SPONSOR: Danone Institute

INVESTIGATORS: Dr. B.J. Kaplan, Ms. Karen Davison, Dr. Marja Verhoef, Dr. Scott Patten, Dr. Michael Eliasziw, Department of Community Health Sciences, University of Calgary

This consent form is only part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. If you would like more detail about something mentioned here, or information not included here, please ask. Take the time to read this carefully and to understand any accompanying information. You will receive a copy of this form. Please bring it with you to the interview.

BACKGROUND

We are inviting you to participate in a study conducted by researchers at the University of Calgary, Department of Community Health Sciences. The aim of the study is to investigate nutrition status in adults with mood disorders. You have been invited to participate because you apparently have a mood disorder and you are 18 years of age or older.

WHAT IS THE PURPOSE OF THE STUDY?

The main purpose of this study is to investigate nutrient status in adults with mood disorders.

WHAT WOULD I HAVE TO DO?

If you decide to participate, we are asking you to complete a food record and then attend an interview. As discussed over the phone, this consent form is part of the study.

First we are asking you to complete a 3-day food record (enclosed). The registered dietitian will have reviewed the procedures for completing this. If you have any questions, please feel free to contact her. You may have also agreed to provide a sample of blood, in which case you should also find a laboratory requisition enclosed. You can go to any BC Biomedical Laboratory to have this done.

At the interview, a trained interviewer will ask you questions about your mental health to verify if you have a mood disorder. At this same appointment, you will meet with the dietitian who will review your 3-day food record. She will also ask you other nutrition questions and will measure your height and weight. The interview will take about 90 minutes.

WHAT ARE THE RISKS?

There are no known risks associated with participation in this study. However, the recording of food intake for three days may seem burdensome. There is, of course, a small amount of discomfort from having blood drawn, but there is no danger from the loss of such a small amount of blood (about a teaspoon). If our data indicate that you are at any health risk we will inform you.

WILL I BENEFIT IF I TAKE PART?

If you agree to participate in this study there may or may not be a direct medical benefit to you. The information we get from this study may help us to develop nutrition education and intervention strategies for individuals with mood disorders. If you are interested, we will give you feedback about your own nutrient status.

DO I HAVE TO PARTICIPATE?

Participation in this study is voluntary and you may withdraw from the study at any time without jeopardizing your health care or your relationship with the Mood Disorders Association of British Columbia. To withdraw, simply inform us that you do not wish to go any further with the study. If you like, you can provide reasons for withdrawing but you are not required to do so.

In addition, the researcher can withdraw you from the study. This would most likely occur in the event that the criteria of the study for including participants were not met. For example, if it were discovered that a participant is pregnant, she would need to be

withdrawn from the study because pregnant women have different nutritional needs that are not being examined in the study.

WHAT ELSE DOES MY PARTICIPATION INVOLVE?

In summary, your participation will involve completing a 3-day food record, providing a blood sample (if you agree to it), and one appointment. Your name will be placed on a mailing list and you will be sent a copy of the results of the study, in approximately 2006.

WILL I BE PAID FOR PARTICIPATING, OR DO I HAVE TO PAY FOR ANYTHING?

The costs you may incur because of participation in this study include parking and travel. If you agree to participate, you will be offered $50 at the end of the interview as a honourarium to cover your time, travel and parking expenses. The provision of this honourarium is in recognition of the fact that we are asking you to do several things that will absorb some of your time.

WILL MY RECORDS BE KEPT PRIVATE?

The researchers of this study will have access to information collected according to identification numbers but the identity of the participant will not be disclosed. To ensure confidentiality, records will be kept on site at the Mood Disorders Association of British Columbia in a locked filing cabinet. All will be coded. Data in hard copy will also be stored in a locked filing cabinet. Information on computer disk will only be accessed by code. Your responses will be confidential and your name will not appear in any publications or presentations of the results from this study. The University of Calgary Conjoint Health Research Ethics Board will have access to the records. All hard copies of questionnaires and data for this study will be destroyed 5 years after the results are published.

IF I SUFFER A RESEARCH-RELATED INJURY, WILL I BE COMPENSATED?

In the event that you suffer injury as a result of participating in this research, the Mood Disorders Association of British Columbia, the Behavioural Research Unit of Alberta Children's Hospital, the University of Calgary, the Calgary Health Region or the researchers will provide no compensation to you. You still have all your legal rights. Nothing said in this consent form alters your right to seek damages.

SIGNATURES

Your signature on this form indicates that you have understood to your satisfaction the information regarding your participation in the research project and agree to participate as a subject. In no way does this waive your legal rights nor release the investigators, or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time without jeopardizing your health care.

If you have further questions concerning matters related to this research, please contact the researcher coordinator, Karen Davison at [redacted], or her primary supervisor Dr. Bonnie Kaplan [redacted].

If you have any questions concerning your rights as a possible participant in this research, please contact Pat Evans, Associate Director, Internal Awards, Research Services, University of Calgary, at [redacted].

Participant's Name	**Signature and Date**
Investigator/Delegate's Name	**Signature and Date**
Witness' Name	**Signature and Date**

The University of Calgary Conjoint Health Research Ethics Board has approved this research study. A copy of this consent form has been given to you to keep for your records and reference.

THREE-DAY FOOD RECORD

ID #:			

Date: _____

Time 00:01 23:59	**Rest/ Café ***	**Meal/ Snack ****	**Home Prep. *****	**FOOD DESCRIPTION**	**Amount/ Portion size**

These are the dates to record what you ate: _____ _____ _____

Please document the recipes of any food items indicated on the food record that were prepared from scratch.

Name of Recipe: _____

Cooking Time: _____

Temperature: _____

Description of the recipe and cooking procedures	Quantity

Total Yield: _____

FOOD FREQUENCY QUESTIONNAIRE

ID #:			

Part I

This part of the study involves reviewing a list of foods that we are interested in knowing whether you ate or not over the last month. If you did eat them, we would like to know how often you ate them and how much you ate at any one time. We are interested only in whether you have eaten them in the last month. So, if you have not eaten those foods at least once within the past month (that is before…give the date of one month ago) they are not important to this part of the study. Please think about the last 4 weeks. Were they work weeks or holiday times? Did they involve a special occasion such as wedding or party? Now I am going to read the list of foods.
"In the past month did you eat ___?" IF NO, MOVE TO NEXT FOOD. IF YES, THEN GO TO DAILY.

⇒DAILY

"Did you eat _____ everyday in the past 4 weeks?" IF NO, MOVE TO WEEKLY
IF YES, THEN ASK "How many times a day?" Using this model as a guide, would your usual serving size be the same, more, or less. How much more, how much less?"

⇒WEEKLY

"Did you eat _____ every week in the past month?" IF NO, MOVE TO MONTHLY
IF YES, THEN ASK "How many times a week? Using this model as a guide, would your usual serving size be the same, more, or less. How much more, how much less?"

⇒MONTHLY

"Then, how many times over the past month have you eaten _____ ? Using this model as a guide, would your usual serving size be the same, more, or less. How much more, how much less?"

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		**Day/D Week/W Month/M**	**Reference Portion Size or Model**	**How much/ How many?**	
HOW OFTEN DID YOU CONSUME <u>Vegetables and Fruits</u>							
1	Broccoli, dark leafy greens – cooked or raw				½ cup		
2	Cabbage, coleslaw, sauerkraut				½ cup		
3	Carrots or mixed vegetables and carrots				½ cup		
4	Squash (dark yellow)				½ cup		
5	Potatoes – French fries or panfried				½ cup		
6	Potatoes – baked, mashed or boiled				½ cup		
7	Fruit – all types				½ cup		
8	Fruit and vegetable juices – all types				½ cup		

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		**Day/D Week/W Month/M**	**Reference Portion Size or Model**	**How much/ How many?**	
HOW OFTEN DID YOU CONSUME <u>Meat, Poultry, Fish and Alternates</u>							
9	Poultry – fried				3 oz		
10	Poultry – cooked other ways				3 oz		
11	Beef – steaks, roasts, stews, and other cuts				3 oz		
12	Hamburgers and other ground beef				3 oz		
13	Pork/Ham – roasts, chops and other cuts				3 oz		
14	Pork – bacon				1 strip		
15	Liver				3 oz		
16	Fish – fried (excl. shellfish)				3 oz		
17	Fish – cooked other ways (excl. shellfish)				3 oz		
18	Wieners or Sausages (with/without a bun)				1 unit		

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		**Day/D Week/W Month/M**	**Reference Portion Size or Model**	**How much/ How many?**	
Meat/Alternatives continued..							
19	Luncheon meats (cold cuts)				1 slice		
20	Eggs or egg dishes				1 egg		
HOW OFTEN DID YOU CONSUME <u>Milk Products</u>							
21	Cheese – regular, hard (>25% MF)				1 slice or 1" cube		
22	Cheese – lower or reduced fat, part skim, processed or spreadable (10-24% MF)				1 slice or 1" cube or 2 tbsp		
23	Cottage or ricotta cheeses (all types)				¼ cup		
24	Yoghurt – all types				½ cup or 175 G		
25	Ice Cream – regular or rich				½ cup		
26	Ice Cream – low fat, frozen yoghurt, ice milk or sherbet				½ cup		

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		**Day/D Week/W Month/M**	**Reference Portion Size or Model**	**How much/ How many?**	
HOW OFTEN DID YOU CONSUME <u>Grain Products</u>							
27	White bread, buns, bagels				2 slices or 1 bun or 1 bagel		
28	100% or 60% whole wheat, multigrain, rye or pumpernickel bread, buns, bagels				2 slices or 1 bun or 1 bagel		
29	Cookies				1 cookie		
30	Crackers				1 cracker		
31	Donuts, cakes, pies, muffins, croissants, chocolate bars				1 unit		

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		**Day/D Week/W Month/M**	**Reference Portion Size or Model**	**How much/ How many?**	
HOW OFTEN DID YOU CONSUME <u>Grain Products</u>							
Other Foods							
32	Potato or tortilla chips				1 bowl		
33	Pizza				1 slice		
34	Rich gravy or pan drippings				¼ cup		
35	Cream or cheese sauce				¼ cup		
Alcohol							
36	Beer, coolers, ciders				1 bottle		
37	Wine				4 F oz		
38	Hard liquors – rye, whiskey, scotch, rum				1 F oz		

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		Day/D Week/W Month/M	Reference Portion Size or Model	How much/ How many?	
What kind of milk did you use in: (Do not read list. See codes below.)							
39	Tea or coffee				1 tbsp		
40	On Cereal				½ cup		
41	As a Beverage (white or chocolate)				1 cup		

A	Whole milk	I	Evaporated, light	Q	Rice milk
B	2% milk	J	Evaporated, 2%	R	Fortified rice milk
C	1% milk	K	Evaporated, skim	S	Other e.g. goat milk (specify)
D	Buttermilk	L	Evaporated, regular (whole), DILUTED	T	Used coffee whitener
E	Skim milk	M	Evaporated, light, DILUTED	U	Did not add milk or cream to tea or coffee
F	Dry skim milk powder	N	Evaporated, 2%, DILUTED	V	Ate cereals dry
G	Cream or creamers	O	Soy beverage		
H	Evaporated, regular (whole)	P	Fortified soy beverage		

FOOD		FREQUENCY			PORTION SIZE		COMMENTS
		Further Food Descriptions		Day/D Week/W Month/M	Reference Portion Size or Model	How much/ How many?	
HOW OFTEN DID YOU CONSUME Added Fats							
42	Vegetable oil				1 tsp		
43	Lard, bacon fat or other animal fat				1 tsp		
44	Shortening				1 tsp		
45	Butter				1 tsp		
46	Soft margarine				1 tsp		
47	Hard margarine				1 tsp		
48	Pam, non-stick spray or no oil				1 tsp		
49	Mayonnaise, mayonnaise-type and regular salad dressing				1 tsp		
50	Low calorie and calorie-reduced salad dressing				1 tsp		

Part II

This section deals with your intake of meat or poultry during the past month. Check (√) all that apply. If the person did not eat meat, fish or poultry in Part I, then check (√) "Did not eat meat/poultry/fish cooked by these methods this past month" below where appropriate.)

51. Of the meat, poultry and fish you ate last month, what was the most common method of cooking it? (Do NOT read list.)		
A		Broiled
B		Pan-fried with fat
C		Pan-fried without fat or with pan spray
D		Deep-fat fried
E		Oven-roasted (Baked)
F		Boiled
G		Microwaved
H		Barbequed
I		Steamed/Poached
J		Other method, specify:
K		Did not eat meat this past month

52. Of the meat you ate last month, did you eat the visible fat of the meat?		
A		Always
B		Sometimes
C		Never
D		Did not eat meat this past month
53. Of the poultry you ate last month, did you eat the skin of the poultry?		
A		Always
B		Sometimes
C		Never
D		Did not eat poultry this past month

NUTRIENT SUPPLEMENTS

1. Yesterday, did you take any of the following: nutritional supplements, vitamins, minerals, or herbal, botanical or homeopathic preparations? Yes No
2. In the past month, did you take any other nutritional supplements, vitamins, minerals or herbal, botanical or homeopathic preparations? Yes No
3. Please tell me the name of all these products with their DIN (when available) that you took yesterday or during the last month. (DIN is a Drug Id. #)
4. Yesterday, at what time did you take your supplements and how many pills (or tablets, capsules, teaspoons, etc.) were taken at each time?
5. In the last month, how often was each of these supplements taken? (Number of times per day, per week or per month)
6. How many pills (or tablets, capsules, teaspoons, etc.) were usually taken on each occasion?

SUPPLEMENT NAME	**DIN**	**YESTERDAY**		**DURING THE LAST MONTH**			
				How Often			**How Much**
		At what time?	# Pills, Tabs, Caps, Tsp…	Day	Week	Month	# Pills, Tabs, Caps, Tsp…

FOOD SELECTION QUESTIONNAIRE

ID #:			

These set of questions deal with why you choose certain foods. I will also ask you questions that will allow us to compare your answers with those provided by people of similar backgrounds. To begin I have a list of health related items that I will read out to you. Answer "yes" only to those reasons that influence what you do or don't eat. *(Check (√) all that apply. Briefly probe to confirm some action is being taken. Enter a "0" if not applicable.)*

1. Are you choosing or avoiding foods or types of foods because you are concerned about: (*read list and check (√) all that apply*).

1	☐	Maintaining or improving your health?
2	☐	Heart disease?
3	☐	Cancer?
4	☐	Osteoporosis (brittle bones)?
5	☐	High blood pressure?
6	☐	Diabetes?
7	☐	Your body weight?
8	☐	Food allergies or intolerances? (Specify: _____)
9	☐	None of the above
10	☐	Other (specify): _____

2. Are you choosing to eat foods or types of foods because of: (*read list and check (√) all that apply*).

1	☐	The lower fat content?
2	☐	The type of fat they contain?
3	☐	The fibre content?
4	☐	The iron content?
5	☐	The calcium content?
6	☐	The other vitamins or minerals they contain? Specify: _____
7	☐	None of the above
8	☐	Other nutrition components (Specify: _____)

3. Are you avoiding foods or types of foods because of: (*read list and check (√) all that apply*).

1 ☐ The fat content?
2 ☐ The salt content?
3 ☐ The cholesterol content?
4 ☐ The sugar content?
5 ☐ The saturated fat content?
6 ☐ None of the above
7 ☐ Other (Specify: _____)

4. Do you consider yourself a vegetarian?

1 ☐ Yes ⇒ **go to question 5**
2 ☐ No ⇒ **go to question 6**
3 ☐ Don't know

5. Do you ever eat:

A	Dairy Products	1	☐	Yes	2	☐ No
B	Eggs	1	☐	Yes	2	☐ No
C	Fish/Seafood	1	☐	Yes	2	☐ No
D	Poultry	1	☐	Yes	2	☐ No
E	Red meat	1	☐	Yes	2	☐ No

6. I'm going to read a list of conditions. Please indicate if you have this condition or if you have had this condition in the past. (*Read list. Check (√) all that apply.)*

1 ☐ High Cholesterol
2 ☐ Heart Disease
3 ☐ Stroke
4 ☐ Cancer
5 ☐ Osteoporosis
6 ☐ Diabetes (Read – does NOT include diabetes during pregnancy)
7 ☐ High Blood Pressure (Read – does NOT include high blood pressure during pregnancy)
8 ☐ Previous mental health diagnoses. Specify: _____
9 ☐ Other (specify: _____)
10 ☐ None of the above

7. Are you following any special diet?

1 ☐ Yes (specify _____)
2 ☐ No ⇒ **go to question 9**
3 ☐ Don't know

8. Who prescribed this special diet?

1 ☐ Doctor
2 ☐ Dietitian
3 ☐ Naturopath
4 ☐ Chiropractor
5 ☐ Relative/Friend (not a doctor, dietitian, naturopath or chiropractor)
6 ☐ Self-prescribed
7 ☐ Don't know
8 ☐ Refused
9 ☐ Other (specify _____)

9. Have you ever seen or heard about the Canada's Food Guide to Healthy Eating? (Show Food Guide)

1 ☐ Yes
2 ☐ No ⇒ **go to question 12**
3 ☐ Don't know

10. Do you use it?

1 ☐ Yes ⇒ **go to question 11**
2 ☐ No ⇒ **go to question 12**
3 ☐ Don't know

11. How do you use it? *(Do not read. Check (√) all that apply.)*

1 ☐ For shopping (e.g. to prepare my shopping list)
2 ☐ For planning/choosing meals (at home)
3 ☐ For choosing foods in restaurants
4 ☐ Other (specify _____)

For the next set of questions, please indicate the response which best describes your eating behaviour.

12. How often are you restricting your food intake in a conscious effort to control your weight? Would you say this is: (show card)

1 ☐ Rarely
2 ☐ Sometimes
3 ☐ Usually
4 ☐ Always

13. Do you go on eating binges even though you are not hungry? Would you say you do this (show card): (Note: binge eating means eating a very large amount of food in a very short period of time, and feeling that you can't control how much you're eating).

1 ☐ Never
2 ☐ Rarely
3 ☐ Sometimes
4 ☐ At least weekly

14. How likely are you to consciously eat less than you want. Would you say you are:

1 ☐ Unlikely
2 ☐ Slightly likely
3 ☐ Moderately likely
4 ☐ Very likely

15. On a scale of 0-5, where 0 means no restraint in eating (eating whatever you want, whenever you want), and 5 means total restraint (constantly limiting food intake and never 'giving in'), how you rate yourself? (Show cared. Circle response).

0	1	2	3	4	5
Eat whatever you want, whenever you want	Usually eat whatever you want, whenever you want	Often eat whatever you want, whenever you want	Often limit food intake, but often 'give in'	Usually limit food intake, rarely 'give in'	Constantly limit food intake, never 'give in'

The next sets of questions are about your weight and the way you feel about your body.

16. On a scale of 0 to 5, where 0 is very uncomfortable and 5 is very comfortable, how comfortable do you feel about your body when you see yourself in a mirror? (show card with scale on it)

0 ☐ Very uncomfortable
1 ☐ Somewhat uncomfortable
2 ☐ Neutral (neither comfortable nor uncomfortable)
3 ☐ Somewhat comfortable
4 ☐ Very comfortable
5 ☐ Not sure

17. I am going to read out some statements. Please state whether the statement is true (T) or false (F) for you. (If respondent cannot decide whether a statement is true or false for them, ask them to identify whether it is true or false most of the time). (circle T or F)

		1	2
A	I do not eat some foods because they make me fat…….……	T	F
B	When I feel 'down' or sad, I often overeat…….	T	F
C	I deliberately take small helpings as a means of controlling my weight…….	T	F
D	Sometimes when I start eating, I just can't seem to stop…….……	T	F
E	When I feel lonely, I console myself by eating……..	T	F

Next, I am going to ask some questions about your food situation.

18. In the past 12 months did you:

	1	2	3
A Worry that there would not be enough to eat because of a lack of money?	☐ Yes	☐ No	☐ Don't know
B Not have enough food to eat because of a lack of money?	☐ Yes	☐ No	☐ Don't know
C Access the services of any food assistance programs?	☐ Yes	☐ No	☐ Don't know

19. Do you smoke?

1 ☐ Yes
2 ☐ No

20. How would you describe your current activity level?

1 ☐ Active
2 ☐ Moderately active
3 ☐ Inactive

The following questions identify particular groups of people and allow us to make comparisons.

21. Are you:

1 ☐ Male
2 ☐ Female

22. What is your current marital status? Are you: *(read list)*

1 ☐ Married or *Living common law
2 ☐ Divorced
3 ☐ Separated
4 ☐ Widowed
5 ☐ Never married

*Common law is defined as adults of the opposite sex or same sex living together in a sexual union of 3 months or more duration. – Statistics Canada

23. How many people are in your household, including non-family members (include self)?

☐☐ # people

24. What is the highest level of education that you received?

1 ☐ No schooling
2 ☐ Some elementary or grade school
3 ☐ Completed elementary or grade school
4 ☐ Some secondary or high school
5 ☐ Completed secondary or grade school
6 ☐ Some trade/vocational training
7 ☐ Completed trade/vocational training
8 ☐ Some university
9 ☐ Certificate or diploma below bachelor's level
10 ☐ Bachelor's degree or above

25. What is your date of birth?

day		month		year	

Current age? _____

26. How old were you when your mood disorder was diagnosed? _____

27. I am going to read a list of treatments for mood disorders, please indicate which ones you are currently taking? (Check (√) *all that apply and indicated types)*

1 ☐ Prescription medication. Types: _____

2 ☐ Herbal or natural remedies Types: _____

3 ☐ Other. Specify: _____

28. The following questions concern any problems you might have had with taking psychiatric pills. If you are currently bothered by any of the following please indicate so (Read list and check (√) *all that apply*)

1 ☐ Poor appetite

2 ☐ Nausea

3 ☐ Heartburn

4 ☐ Vomiting

6 ☐ Diarrhea

7 ☐ Constipation

8 ☐ Weight loss: Amount of weight lost/time period _____/_____

9 ☐ Weight gain: Amount of weight gained/time period _____/_____

10 ☐ Dry mouth

11 ☐ Altered taste

12 ☐ Other nutrition-related problems (specify): _____

This following question will help us interpret the results of this study.

29. Please look at this card and tell me, as close as you possibly can, the code that corresponds to your family income before taxes in 2002. (Family is a group of individuals related by blood, marriage including common-law, or adoption, who share a common dwelling unit at the time of the study. – Statistics Canada)

1	☐	≤ $12,000
2	☐	$12,001 to 15,000
3	☐	$15,001 to 20,000
4	☐	$20,001 to 25,000
5	☐	$25,001 to 30,000
6	☐	$30,001 to 35,000
7	☐	$35,001 to 40,000
8	☐	$40,001 to 45,000
9	☐	$45,001 to 55,000
10	☐	>$55,000
11	☐	Don't know
12	☐	Refused

We have reached the end of this set of questions. If possible I would like to measure your weight and height. Interviewer to indicate weight calibration at beginning of each week in following:

Weight used for calibration: ☐☐☐ . ☐ kg

Weight on scale of calibration weight: ☐☐☐ . ☐ kg

30. Respondent's weight:

☐☐☐ . ☐ kg

OR

☐☐☐ . ☐ lbs

1 ☐ Measured

2 ☐ Self-Reported (specify reason) _____

3 ☐ Refused

Comments: _____

31. Respondent's height: (note on comment line if measurement may not be accurate and why)

☐☐☐.☐ inches	1 ☐	Measured
OR	2 ☐	Self-Reported (specify reason) _____
☐☐☐.☐☐ cm	3 ☐	Refused

Comments: _____

The following are the last set of questions of this survey.

32. Do you believe that what people eat and drink has an effect on or can prevent major diseases?

1 ☐ Yes
2 ☐ No
3 ☐ Don't know

33. Which of the following nutritional issues are important to you?

1 ☐ Avoiding salt
2 ☐ Avoiding sugar
3 ☐ Lowering cholesterol
4 ☐ Avoiding being overweight
5 ☐ None of these

34.Do you think your diet is:

1 ☐ High in fiber?
2 ☐ Low in fiber?
3 ☐ High in fat?
4 ☐ Low in fat?
5 ☐ Don't know?

35. Which of the following foods are high in fat?

1 ☐ White bread
2 ☐ Soda
3 ☐ Broiled fish
4 ☐ Bananas
5 ☐ Cold cuts or ham
6 ☐ Don't know

36. Which of the following foods are high in fiber?

1 ☐ Red meat
2 ☐ Lettuce
3 ☐ Carrots
4 ☐ White rice
5 ☐ Apples

Thank you for answering this set of questions.

APPENDIX C: NUTRIENT INTAKE DATA

Table C.0.1: Energy intakes (kcal) by age and gender compared to the British Columbia Nutrition Survey (BCNS)

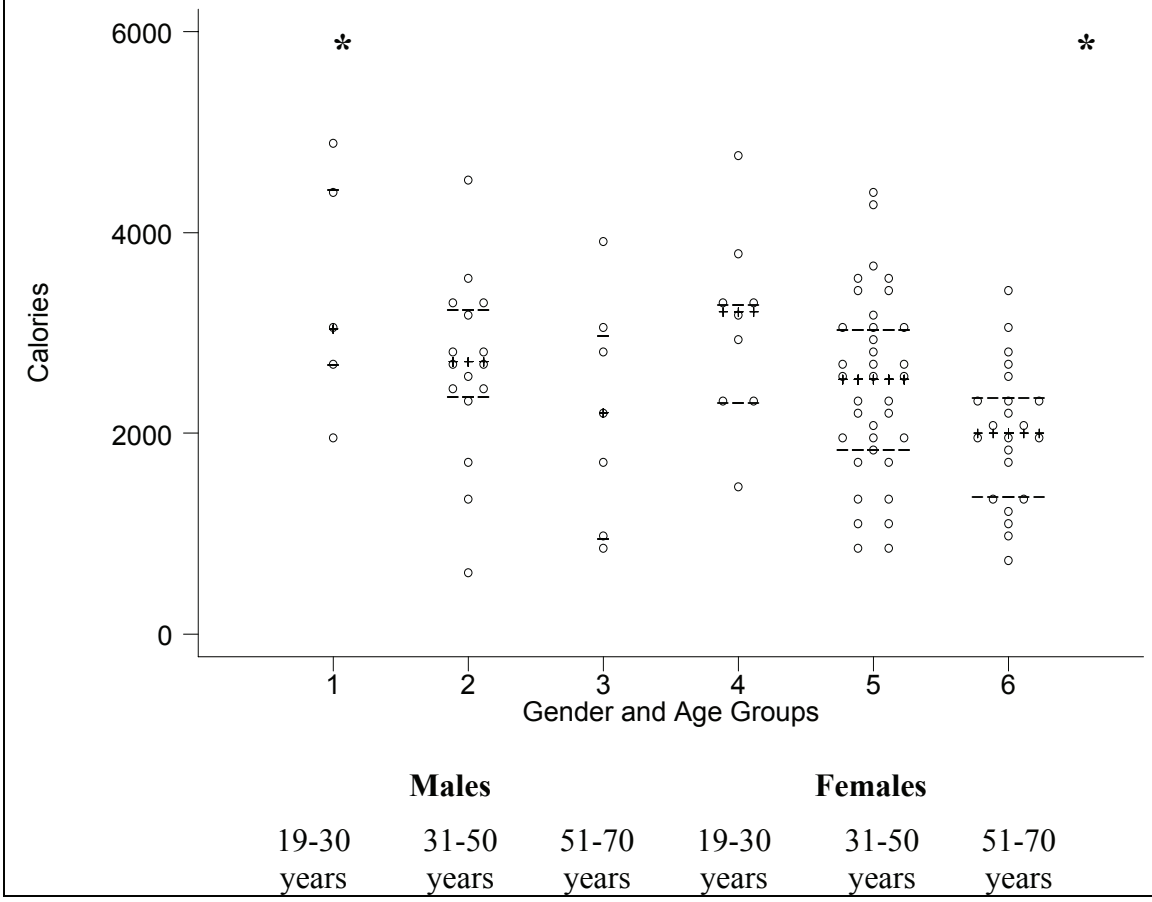
Sex	Age (years)	Sample	n	Mean ± SD[1]	50th percentile (25th; 75th)	TEE Mean ± SD[2,3]
Total Study Sample			97	2482 ± 945	2521 (1918; 3051)	2214 ± 415
Males	19-30	Study	4	3506 ± 1413	3580 (2325; 690)	3055 ± 264
		BCNS	142	2883 ± 1251	2824 (2167; 3452)	2907 ± 179
	31-50	Study	17	2657 ± 894	2717 (2357; 3213)	2782 ± 378
		BCNS	205	2624 ± 1017	2644 (2155; 2912)	2837 ± 243
	51-70	Study	7	2255 ± 1048	2464 (975; 2811)	2456 ± 261
		BCNS	249	2342 ± 1168	2334 (1898’ 2703)	2588 ± 103
Females	19-30	Study	9	2754 ± 780	2952 (2315; 3308)	2401 ± 487
		BCNS	176	1971 ± 955	1875 (1667; 2125)	2282 ± 172
	31-50	Study	39	2504 ± 974	2521 (1802; 3182)	2307 ± 402
		BCNS	266	1812 ± 799	1763 (1515; 2012)	2155 ± 196
	51-70	Study	21	2061 ± 746	2018 (1396; 2683)	2239 ± 262
		BCNS	282	1669 ± 705	1610 (1422; 1826)	2029 ± 252

[1]SD = Standard Deviation

[2]TEE low activity for men = Total Energy Expenditure for those whose activity is low (defined as TEE/BEE of 1.4-<1.6), estimated using the following formula=662 - 9.53 x Age (yr) + PA x (15.91 x Weight [kg] + 539.6 x Height [m]), where PA is the physical activity coefficient and equal to 1.11

[3]TEE low activity for women = Total Energy Expenditure for those whose activity is low, estimated using the following formula=354 – 6.91 x Age (yr) + PA x (9.36 x Weight [kg] + 726 x Height [m]), where PA is the physical activity coefficient and equal to 1.12

Figure C.0.1: Dot plots of calorie intake (kcal/day) by age and gender



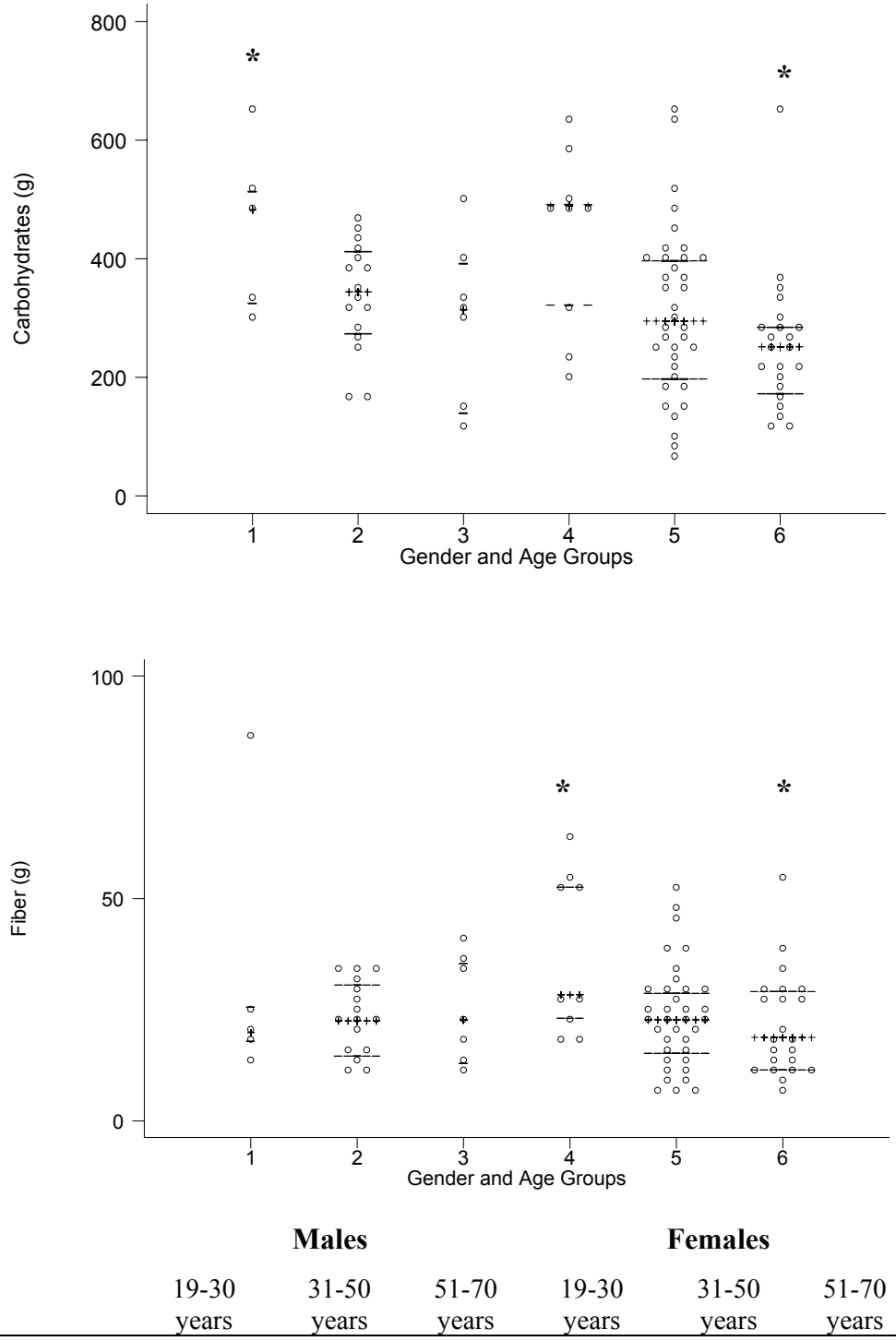
*Significant differences between these groups ($p < 0.05$)

Table C.0.2: Carbohydrate and fibre intakes by age and gender compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams per day)

Sex	Age (years)	Sample	n	Mean ± SD[1]	50th percentile (25th; 75th)	EAR[2]	% < EAR[2]
Total Study Sample			97	323 ± 138	305 (215; 405)	100	0
Males	19-30	Study	4	448 ± 163	422 (315; 581)	100	0
		BCNS	142	365 ± 203	342 (272; 423)	100	0
	31-50	Study	17	347 ± 94	332 (301; 425)	100	0
		BCNS	205	323 ± 143	316 (251; 381)	100	0
	51-70	Study	7	291 ± 135	313 (143; 385)	100	0
		BCNS	249	293 ± 158	288 (222; 343)	100	0
Females	19-30*	Study	9	400 ± 147	488 (241; 491)	100	0
		BCNS	176	265 ± 119	257 (231; 276)	100	0
	31-50*	Study	39	330 ± 146	326 (209; 411)	100	0
		BCNS	266	226 ± 114	219 (189; 249)	100	0
	51-70	Study	21	250 ± 123	245 (167; 288)	100	2
		BCNS	282	213 ± 101	210 (178; 244)	100	<1
						AI[3]	
Total Study Sample			97	25 ± 14	23 (15; 30)	--	
Males	19-30	Study	4	38 ± 33	23 (19; 56)	38	
		BCNS	142	21 ± 12	16 (14; 23)	38	
	31-50	Study	17	21 ± 8	24 (15; 27)	38	
		BCNS	205	20 ± 14	17 (14; 23)	38	
	51-70	Study	7	26 ± 11	23 (14; 36)	30	
		BCNS	249	20 ± 16	18 (13; 23)	30	
Females	19-30	Study	9	38 ± 19	30 (18; 53)	25	
		BCNS	176	16 ±13	13 (11; 17)	25	
	31-50	Study	39	25 ± 13	23 (17; 29)	25	
		BCNS	266	15 ± 16	13 (10; 18)	25	
	51-70	Study	21	22 ± 12	21 (12; 30)	21	
		BCNS	282	17 ± 17	15 (12; 19)	21	

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirement
[3]AI = Adequate Intakes
*Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.05$

Figure C.0.2: Dot plots of carbohydrate and fiber distributions (grams/day) by age and gender



*Significant differences between these groups ($p < 0.05$)

Table C.0.3: Total, saturated, monounsaturated, polyunsaturated, α-linolenic and linoleic fat intakes by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams per day)

Sex	Age (years)	Sample	n	Mean ± SD[2]	50th percentile (25th; 75th)
Total Fat[1]					
Total Study Sample			97	98 ± 50	90 (64; 123)
Males	19-30	Study	4	143 ± 85	133 (83; 202)
		BCNS	142	100 ± 60	97 (70; 120)
	31-50	Study	17	114 ± 46	98 (90; 141)
		BCNS	205	96 ± 57	89 (71; 113)
	51-70	Study	7	81 ± 46	67 (27; 122)
		BCNS	249	84 ± 47	82 (62; 99)
Females	19-30	Study	9	85 ± 18	90 (65; 95)
		BCNS	176	69 ± 53	63 (51; 80)
	31-50	Study	39	96 ± 51	82 (57; 125)
		BCNS	266	69 ± 49	63 (50; 79)
	51-70	Study	21	86 ± 52	83 (54; 111)
		BCNS	282	60 ± 34	55 (46; 64)
Saturated Fat[1]					
Total Study Sample			97	30 ± 17	28 (18; 38)
Males	19-30	Study	4	41 ± 22	44 (24; 57)
		BCNS	142	33 ± 24	30 (21; 42)
	31-50	Study	17	24 ± 17	30 (25; 36)
		BCNS	205	31 ± 29	28 (23; 37)
	51-70	Study	7	24 ± 14	27 (10; 33)
		BCNS	249	27 ± 16	25 (18; 32)
Females	19-30	Study	9	25 ± 13	24 (21; 29)
		BCNS	176	23 ± 27	19 (13; 25)
	31-50	Study	39	31 ± 18	28 (17; 40)
		BCNS	266	23 ± 16	20 (14; 26)
	51-70	Study	21	25 ± 13	24 (16; 32)
		BCNS	282	20 ± 17	18 (14; 23)

[1]There is no *Dietary Reference Intake* for these nutrients
[2]SD = Standard Deviation

Table C.0.3: Total, saturated, monounsaturated, polyunsaturated, α-linolenic and linoleic fat intakes by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams per day) /continued

Sex	Age (years)	Sample	N	Mean ± SD[2]	50th percentile (25th; 75th)
Monounsaturated fat[1]					
Total Sample			97	26 ± 19	22 (16; 29)
Males	19-30	Study	4	26 ± 17	30 (13; 40)
		BCNS	142	41 ± 24	39 (25; 50)
	31-50	Study	17	28 ± 13	28 (20; 34)
		BCNS	205	39 ± 29	36 (29; 43)
	51-70	Study	7	18 ± 11	21 (5; 27)
		BCNS	249	33 ± 16	32 (24; 39)
Females	19-30	Study	9	22 ± 5	21 (18; 27)
		BCNS	176	27 ± 27	25 (20; 30)
	31-50	Study	39	25 ± 19	21 (18; 26)
		BCNS	266	27 ± 16	24 (20; 30)
	51-70	Study	21	27 ± 27	19 (16; 27)
		BCNS	282	24 ± 17	22 (18; 26)
Polyunsaturated Fat[1]					
Total Sample			97	13 ± 10	11 (7; 16)
Males	19-30	Study	4	10 ± 6	11 (7; 14)
		BCNS	142	17 ± 12	16 (11; 20)
	31-50	Study	17	15 ± 12	12 (7; 19)
		BCNS	205	17 ± 14	15 (12; 20)
	51-70	Study	7	9 ± 7	7 (2; 15)
		BCNS	249	15 ± 16	14 (10; 18)
Females	19-30	Study	9	13 ± 6	13 (9; 18)
		BCNS	176	11 ± 13	10 (9; 13)
	31-50	Study	39	14 ± 10	12 (8; 17)
		BCNS	266	12 ± 16	10 (8; 13)
	51-70	Study	21	13 ± 11	11 (8; 14)
		BCNS	282	10 ± 17	9 (7; 11)
		BCNS	142	2.4 ± 2.4	2 (1; 3)

[1]There is no *Dietary Reference Intake* for these nutrients
[2]SD = Standard Deviation

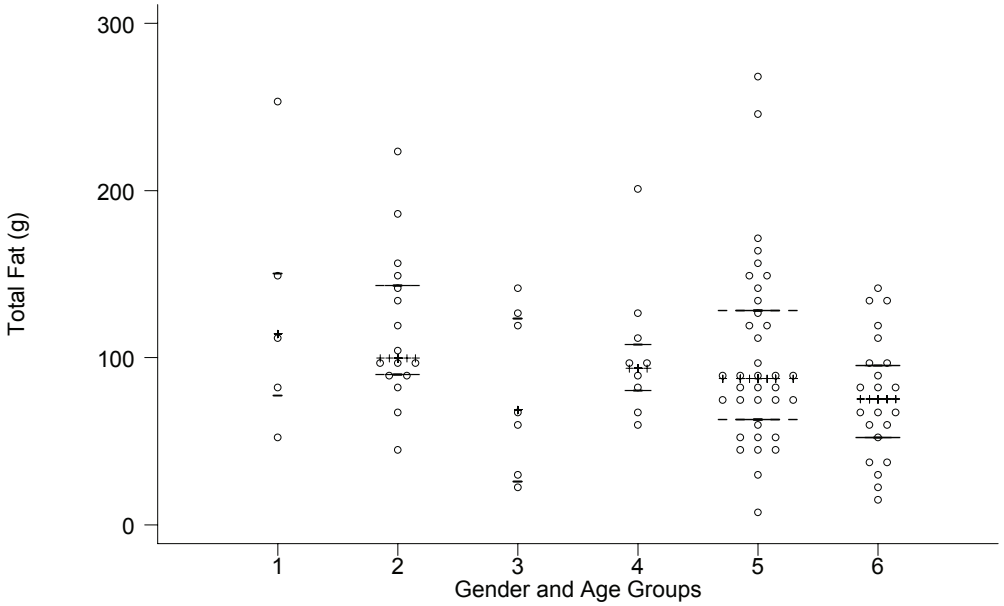
Table C.0.3: Total, saturated, monounsaturated, polyunsaturated, α-linolenic and linoleic fat intakes by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams per day) /continued

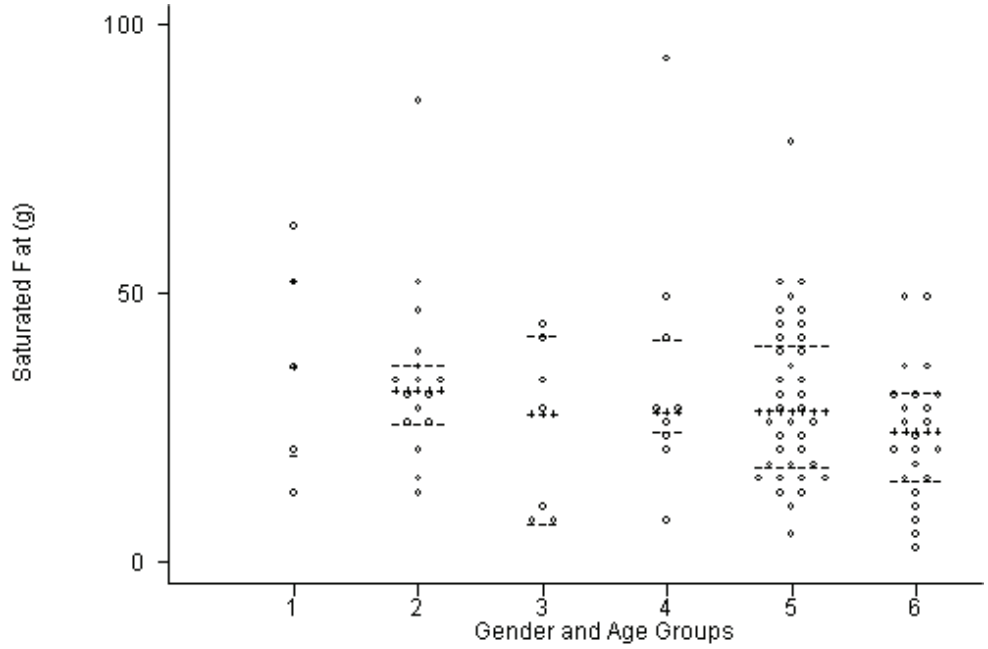
Sex	Age (years)	Sample	n	Mean ± SD[2]	50th percentile (25th; 75th)
α-Linolenic acid[1]					
Total Study Sample			97	0.4 ± 0.5	0.3 (0.1; 0.4)
Males	19-30	Study	4	0.2 ± 0.1	0.3 (0.2; 0.3)
		BCNS	142	2.4 ± 2.4	1.9 (1.3; 2.7)
	31-50	Study	17	0.3 ± 0.2	0.2 (0.1; 0.4)
		BCNS	205	2.3 ± 2.9	1.9 (1.4; 2.6)
	51-70	Study	7	0.5 ± 0.4	0.5 (0.1; 0.9)
		BCNS	249	2.7 ± 6.3	1.8 (1.2; 2.6)
Females	19-30	Study	9	0.2 ± 0.1	0.1 (0.1; 0.3)
		BCNS	176	1.6 ± 1.3	1.3 (1.0; 1.6)
	31-50	Study	39	0.3 ± 0.4	0.2 (0.1; 0.3)
		BCNS	266	1.8 ± 1.6	1.3 (1.1; 1.6)
	51-70	Study	21	0.7 ± 1.0	0.4 (0.2; 0.6)
		BCNS	282	2.0 ± 3.4	1.3 (1.0; 1.6)
Linoleic Acid[1]					
Males	19-30	Study	4	2.7 ± 1.6	2.6 (1.6; 3.7)
		BCNS	142	13.3 ± 9.5	12.0 (9.1; 15.0)
	31-50	Study	17	3.4 ± 3.1	2.5 (1.1; 6.5)
		BCNS	205	14.2 ± 10.0	12.0 (9.3; 16.0)
	51-70	Study	7	4.8 ± 6.3	2.5 (1.0; 6.7)
		BCNS	249	13.2 ± 12.6	11.0 (7.7; 15.0)
Females	19-30	Study	9	3.9 ± 3.0	4.9 (0.7; 6.5)
		BCNS	176	9.2 ± 8.0	7.9 (6.4; 9.7)
	31-50	Study	39	4.4 ± 5.3	3.0 (2.1; 4.5)
		BCNS	266	9.5 ± 8.2	8.1 (6.3; 9.9)
	51-70	Study	21	5.4 ± 5.5	4.1 (2.1; 6.1)
		BCNS	282	8.6 ± 8.4	7.3 (6.1; 8.7)

[1]Adequate Intake (AI) for α-linolenic acid is 1.6 g/day for males and 1.1 g/day for females. Adequate Intake (AI) for linoleic acid is 17 g/day for males and 12 g/day for females aged 19-50 years, and 14 g/d and 11 g/d for older males and females, respectively

[2]SD = Standard Deviation

Figure C.0.3: Dot plots of total and saturated fat intake (grams/day) by age and gender





Males **Females**

19-30 years	31-50 years	51-70 years	19-30 years	31-50 years	51-70 years

Figure C.0.4: Dot plots of monounsaturated and polyunsaturated fat intake (grams/day) by age and gender

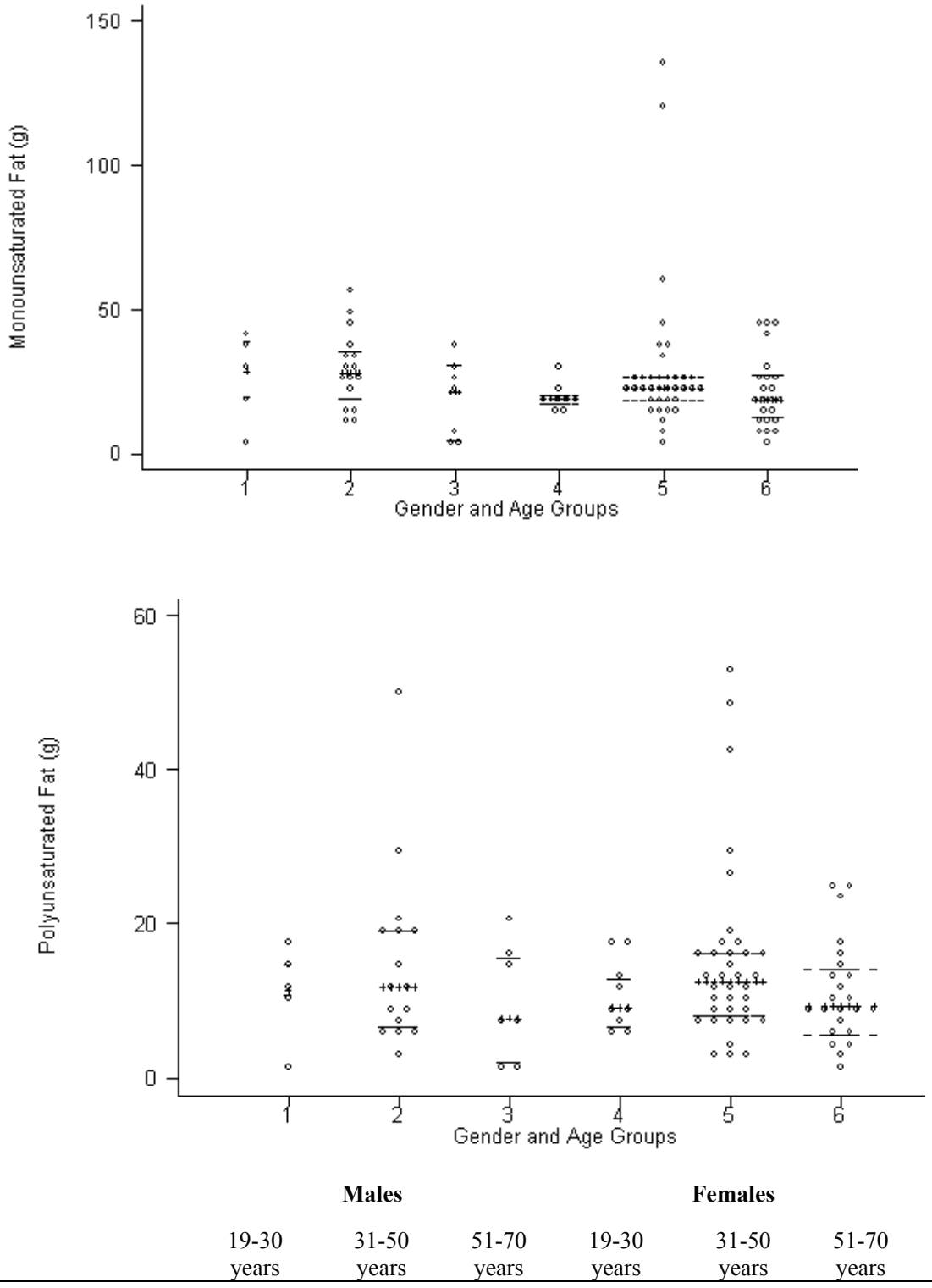
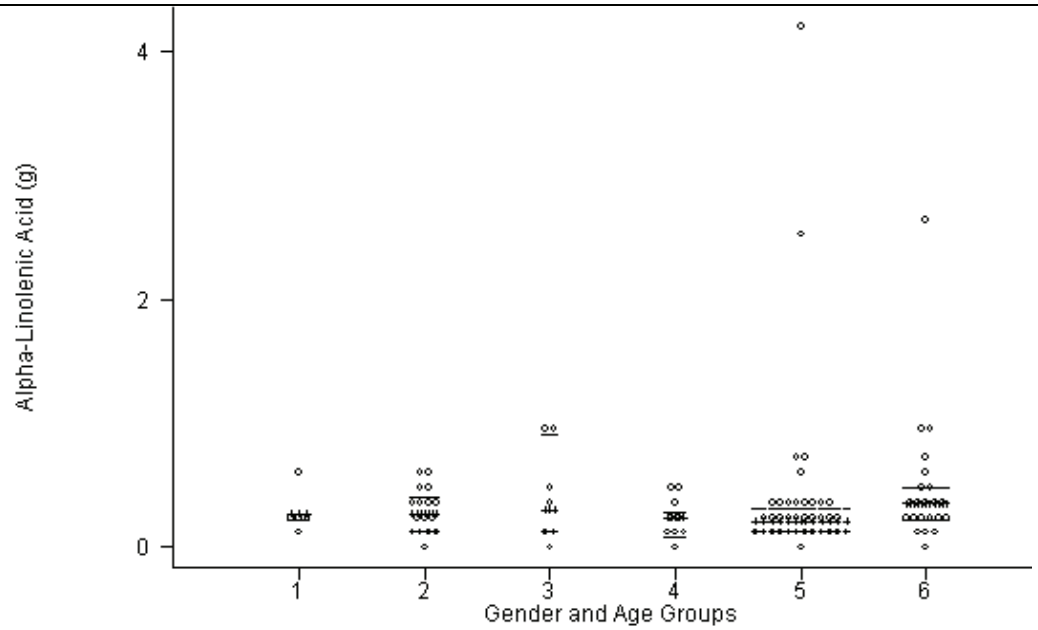
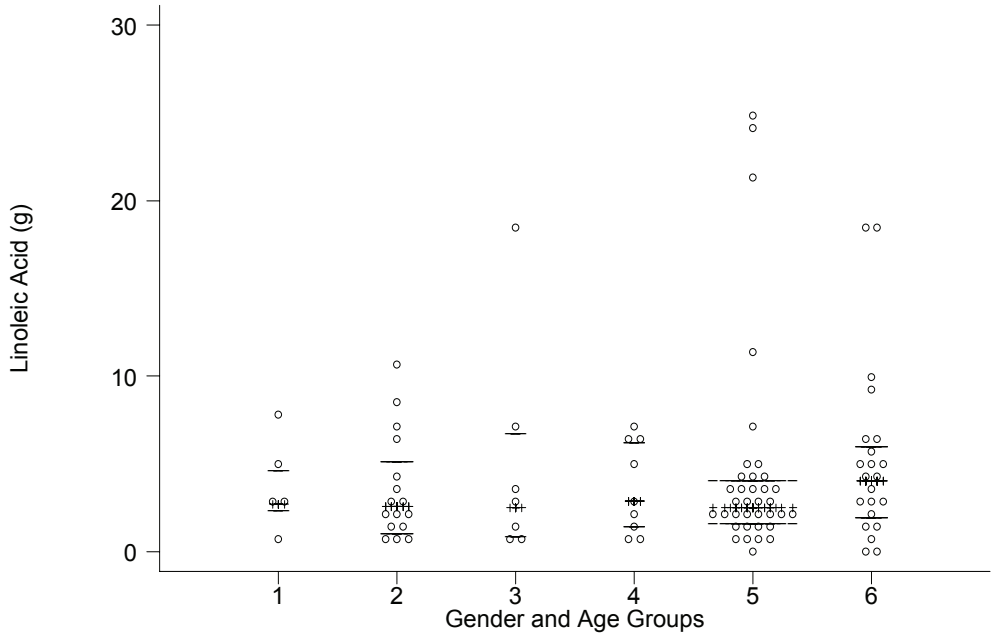


Figure C.0.5: Dot plots of α-linolenic and linoleic fat intake (grams/day) by age and gender





Males **Females**

19-30 years	31-50 years	51-70 years	19-30 years	31-50 years	51-70 years

Table C.0.4: Cholesterol intakes by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)[1]

Sex	Age (years)	Sample	n	Mean ± SD[2]	50th percentile (25th; 75th)
Total Study Sample			97	294 ± 224	219 (149; 405)
Males	19-30	Study	4	458 ± 149	453 (346; 570)
		BCNS	142	383 ± 346	294 (209; 425)
	31-50	Study	17	429 ± 306	295 (215; 638)
		BCNS	205	344 ± 315	258 (196; 351)
	51-70	Study	7	224 ± 114	209 (137; 285)
		BCNS	249	326 ± 363	247 (177; 374)
Females	19-30	Study	9	186 ± 203	147 (74; 199)
		BCNS	176	243 ± 332	166 (115; 258)
	31-50	Study	39	247 ± 192	183 (112; 345)
		BCNS	266	252 ± 277	195 (139; 254)
	51-70	Study	21	315 ± 223	219 (176; 407)
		BCNS	282	224 ± 218	175 (141; 213)

[1]There is no *Dietary Reference Intake* set for cholesterol [2]SD = Standard Deviation

Figure C.0.6: Dot plots of cholesterol intake by age and gender (expressed as milligrams/day)

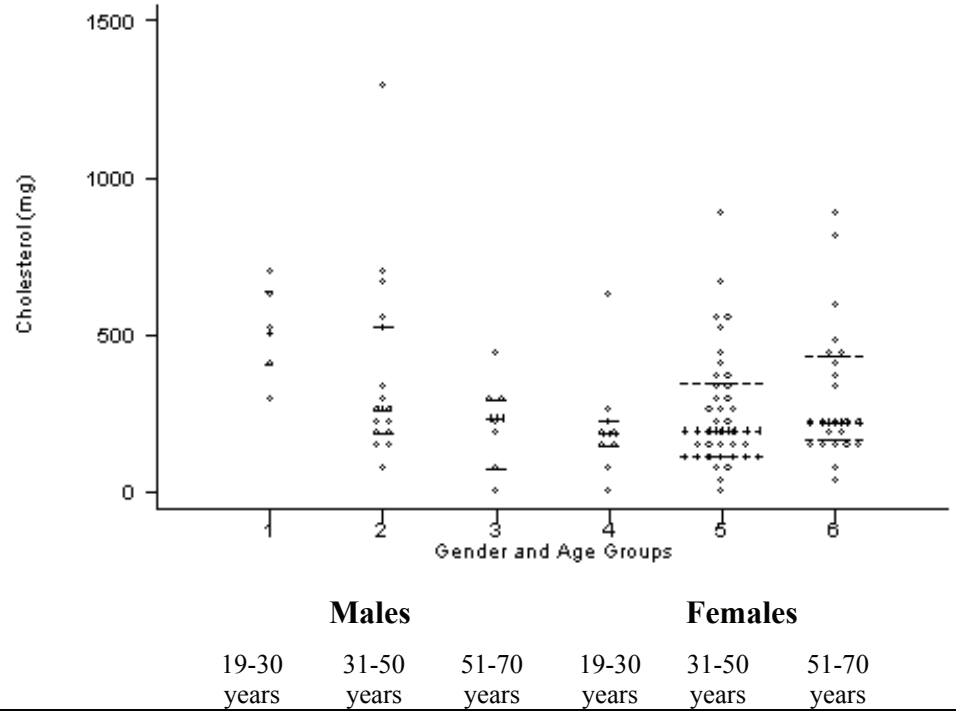


Table C.0.5: Protein intakes by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as grams per kilogram per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% < EAR[2] (0.66 g/kg/d)
Total Study Sample			97	1.21 ± 0.44	1.16 (0.87; 1.53	10
Males	19-30	Study	4	1.22 ± 0.63	1.16 (0.78; 1.66)	25
		BCNS	142	1.50 ± 0.48	1.44 (1.13; 1.76)	4
	31-50	Study	17	1.13 ± 0.33	1.09 (0.90; 1.41)	0
		BCNS	205	1.19 ± 0.29	1.17 (0.94; 1.37)	4
	51-70	Study	7	1.06 ± 0.43	1.13 (0.58; 1.22)	29
		BCNS	249	1.17 ± 0.63	1.09 (0.81; 1.29)	9
Females	19-30	Study	9	1.65 ± 0.57	1.93 (1.27; 1.98)	14
		BCNS	176	1.04 ± 0.27	0.06 (0.82; 1.28)	10
	31-50	Study	39	1.24 ± 0.47	1.23 (1.02; 1.56)	15
		BCNS	266	1.05 ± 0.33	1.04 (0.85; 1.19)	9
	51-70	Study	21	1.10 ± 0.32	0.98 (0.82; 1.32)	0
		BCNS	282	1.00 ± 0.34	0.96 (0.82; 1.17)	10

[1]SD = Standard Deviation [2]EAR = Estimated Average Requirement

Figure C.0.7: Dot plots of protein intake by age and gender (expressed as grams/kilogram body weight/day)

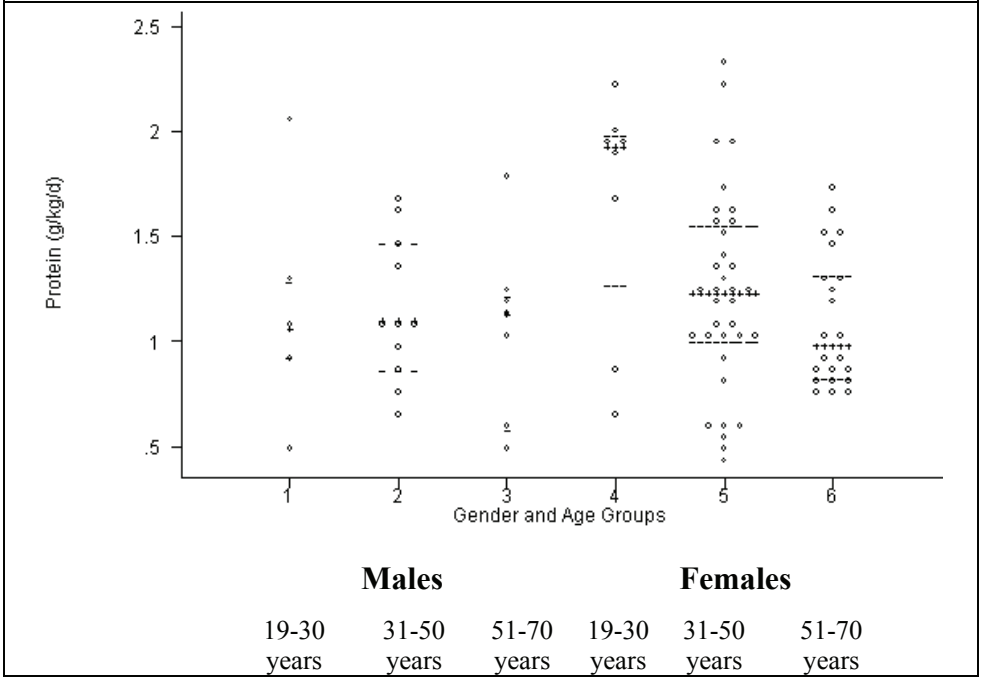
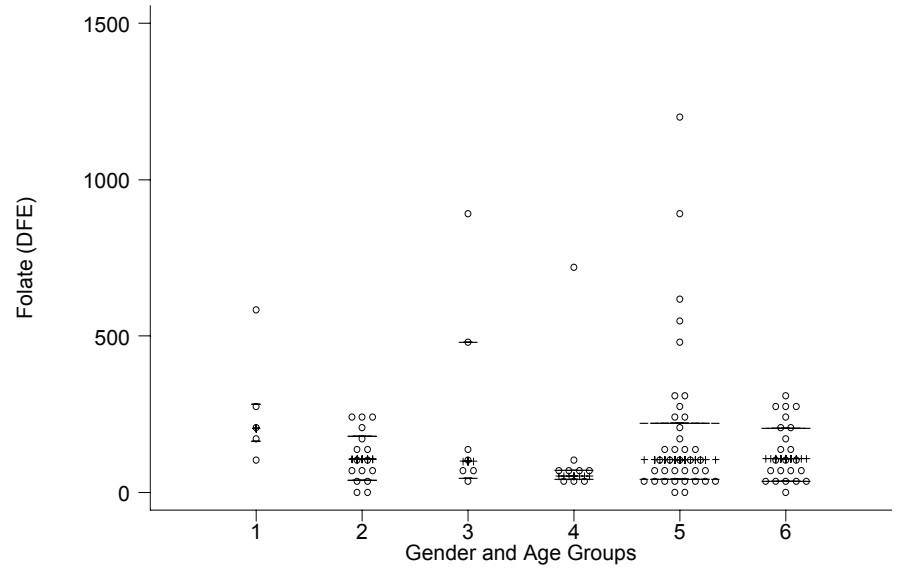


Table C.0.6: Folate intakes from food sources (including fortified foods) and food sources (including fortified foods) and supplements by age and gender compared to the British Columbia Nutrition Survey (BCNS) (expressed as dietary folate equivalents (DFE/d)[1]

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% < EAR[2] (320 DFE/d)
Food Sources Only (including fortified foods)						
Total Study Sample			97	321 ± 181	266 (194; 402)	64
Males	19-30	Study	4	465 ± 184	553 (369; 561)	25
		BCNS	142	617 ± 405	560 (453; 647)	2
	31-50	Study	17	238 ± 519	92 (57; 171)	94
		BCNS	205	553 ± 344	500 (406; 572)	5
	51-70	Study	7	385 ± 300	266 (207; 414)	57
		BCNS	249	504 ± 410	466 (335; 593)	20
Females	19-30	Study	9	152 ± 250	54 (48; 87)	86
		BCNS	176	393 ± 226	368 (300; 418)	32
	31-50	Study	39	332 ± 190	282 (202; 402)	56
		BCNS	266	385 ± 245	344 (279; 437)	40
	51-70	Study	21	280 ± 142	245 (194; 346)	67
		BCNS	282	364 ± 269	327 (266; 386)	47
Food (including fortified foods) and Supplement Sources						
Total Study Sample			97	478 ± 368	365 (202; 641)	39
Males	19-30	Study	4	465 ± 184	553 (369; 561)	25
		BCNS	142	729 ± 357	591 (483; 764)	2
	31-50	Study	17	435 ± 388	321 (192; 577)	41
		BCNS	205	796 ± 859	535 (436; 748)	3
	51-70	Study	7	834 ± 908	532 (380; 667)	29
		BCNS	249	663 ± 552	524 (370; 745)	16
Females	19-30	Study	9	416 ± 236	331 (226; 665)	57
		BCNS	176	607 ± 385	419 (342; 662)	19
	31-50	Study	39	554 ± 411	454 (240; 727)	31
		BCNS	266	621 ± 440	427 (330; 764)	23
	51-70	Study	21	471 ± 344	331 (195; 640)	52
		BCNS	282	671 ± 655	429 (313; 942)	29

[1]Dietary Folate Equivalents (DFE) = values that adjust for the differences in absorption of food folate and synthetic folic acid.1 mcg of DFE = 0.6 mcg of folic acid from fortified food or as a supplement taken with a meal = 1 mcg food folate = 0.5 mcg of folic acid from a supplement taken on an empty stomach
[2]SD = Standard Deviation [3]EAR = Estimated Average Requirement

Figure C.0.8: Dot plots of folate intake from food sources (including fortified foods) and food plus supplements (expressed as Dietary Folate Equivalents/day) by age and gender



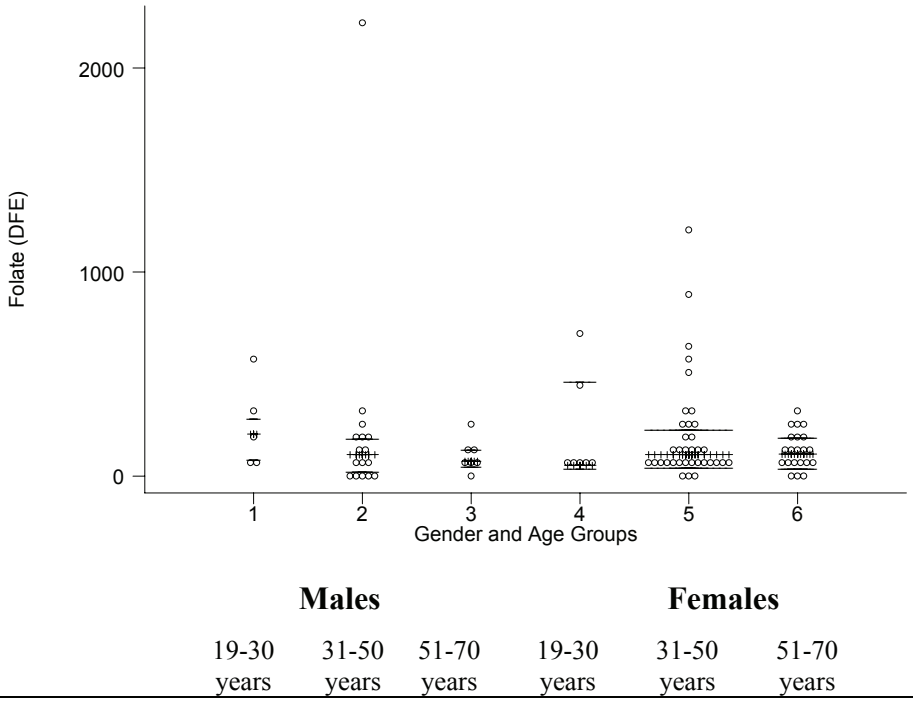


Table C.0.7: Folic acid[1] intakes from fortified foods and supplements by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as micrograms per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% > UL[2] (1000 mcg/d)
Total Study Sample			97	431 ± 211	395 (113; 740)	17
Males	19-30*	Study	4	624 ± 585	513 (148; 1099)	25
		BCNS	142	264 ± 203	176 (129; 263)	3
	31-50	Study	17	254 ± 365	83 (19; 491)	5
		BCNS	205	305 ± 515	149 (112; 227)	4
	51-70	Study	7	597 ± 927	292 (59; 536)	14
		BCNS	249	284 ± 430	92 (55; 254)	7
Females	19-30	Study	9	172 ± 177	101 (27; 336)	0
		BCNS	176	244 ± 226	126 (96; 294)	4
	31-50*	Study	39	356 ± 375	158 (50; 689)	10
		BCNS	266	244 ± 245	119 (80; 324)	4
	51-70	Study	21	281 ± 296	160 (60; 432)	5
		BCNS	282	311 ± 697	86 (51; 469)	5

[1]Folic acid represents the synthetic form of the vitamin. 1 mcg of DFE = 0.6 mcg of folic acid from fortified food or as a supplement taken with a meal = 1 mcg food folate = 0.5 mcg of folic acid from a supplement taken on an empty stomach

[2]SD = Standard Deviation

[3]UL = Tolerable Upper Intake Level

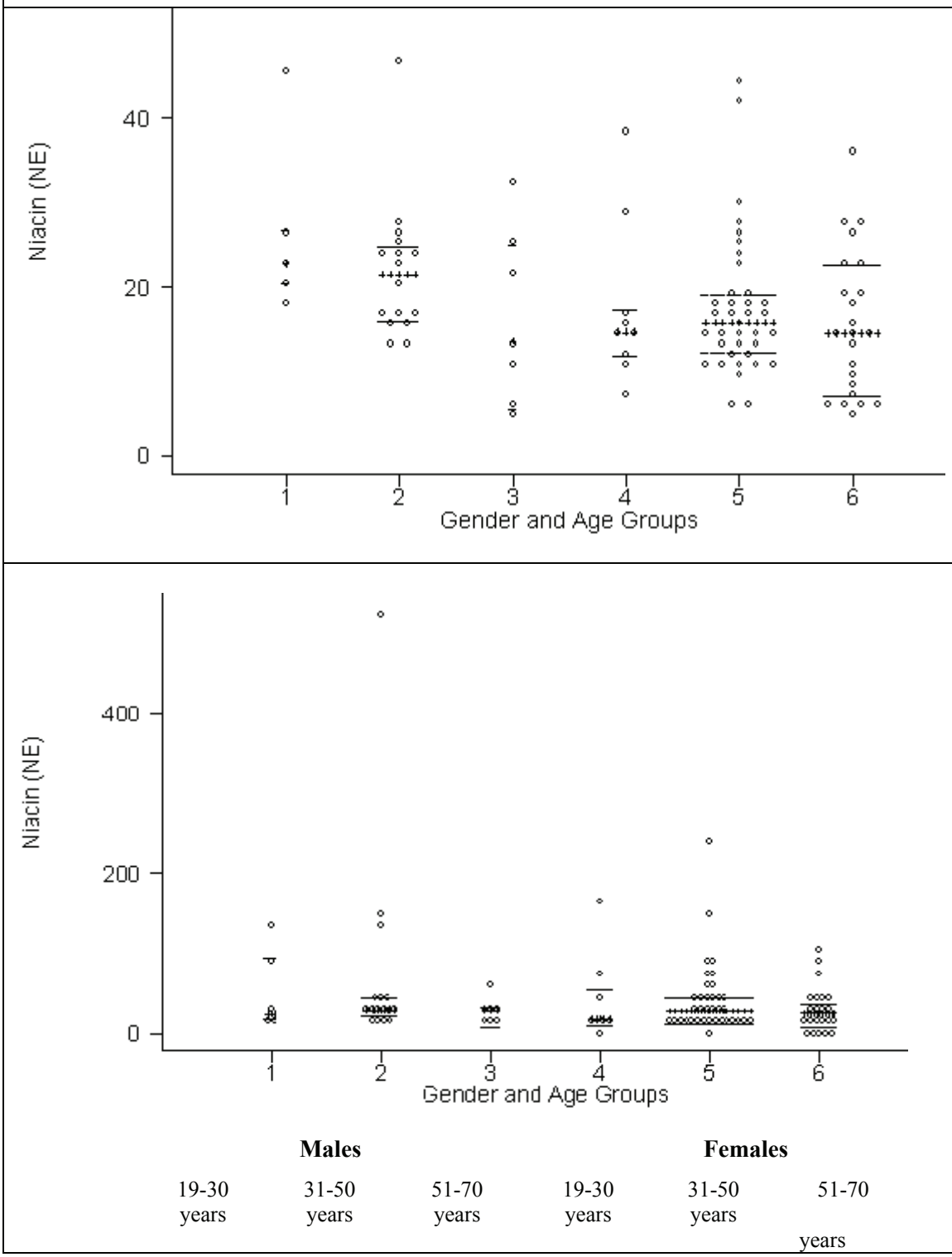
*Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.05$

Table C.0.8: Niacin intakes from food and from food and supplements by age and gender compared to the British Columbia Nutrition Survey (BCNS) (expressed as Niacin Equivalents (NE/d))[1]

Sex	Age (years)	Sample	N	Mean ± SD[2]	50th percentile (25th; 75th)	EAR[3]	% < EAR
Food Sources							
Total Study Sample			97	27 ± 12	26 (20; 34)	--	8
Males	19-30	Study	4	40 ± 19	35 (28; 52)	12	0
		BCNS	142	55 ± 24	50 (41; 63)	12	0
	31-50	Study	17	22 ± 8	23 (16; 24)	12	0
		BCNS	205	47 ± 29	45 (37; 53)	12	0
	51-70	Study	7	29 ± 16	34 (10; 38)	12	29
		BCNS	249	44 ± 32	41 (33; 51)	12	0
Females	19-30	Study	9	31 ± 13	27 (22; 39)	11	0
		BCNS	176	31 ± 13	29 (26; 35)	11	0
	31-50	Study	39	26 ± 11	26 (19; 30)	11	10
		BCNS	266	32 ± 16	30 (26; 34)	11	0
	51-70	Study	21	24 ± 11	22 (15; 32)	11	10
		BCNS	282	32 ± 17	30 (25; 34)	11	0
Food Plus Supplements						**UL[4]**	**% > UL[5]**
Total Study Sample			97	43 ± 64	27 (14; 45)	35	28
Males	19-30	Study	4	40 ± 20	35 (28; 52)	35	50
		BCNS	142	61 ± 23	57 (44; 72)	35	10
	31-50	Study	17	26 ± 11	26 (19; 31)	35	32
		BCNS	205	55 ± 14	50 (38; 64)	35	12
	51-70	Study	7	30 ± 14	27 (17; 34)	35	29
		BCNS	249	60 ± 7	48 (36; 61)	35	12
Females	19-30	Study	9	32 ± 13	27 (22; 39)	35	29
		BCNS	176	48 ± 13	35 (28; 48)	35	12
	31-50	Study	39	27 ± 11	26 (19; 31)	35	29
		BCNS	266	50 ± 33	36 (29; 59)	35	20
	51-70	Study	21	24 ± 11	22 (15; 32)	35	19
		BCNS	282	79 ± 21	37 (29; 63)	35	27

[1]Niacin equivalents = the amount of niacin present in food, including the niacin that can theoretically be made from its precursor, tryptophan, present in the food. To make 1 mg of niacin requires approximately 60 mg of dietary tryptophan
[2]SD = Standard Deviation [3]EAR = Estimated Average Requirement.
[4]UL = Tolerable Upper Intake Level, based on the consumption of synthetic niacin in fortified foods, supplements and pharmacologic agents
[5]Reflects only intake from supplements

Figure C.0.9: Dot plots of niacin intake from food sources and food plus supplement sources by age and gender (expressed as Niacin Equivalents/day)



Niacin equivalents = the amount of niacin present in food, including the niacin that can theoretically be made from its precursor, tryptophan, present in the food. To make 1 mg of niacin requires approximately 60 mg of dietary tryptophan.

Table C.0.9: Pantothenic acid intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	AI[2]
Food Sources						
Total Study Sample			97	4.0 ± 2.0	3.8 (2.6; 5.5)	5
Males	19-30	Study	4	5.7 ± 1.4	6.0 (4.6; 6.7)	5
		BCNS	142	7.2 ± 4.8	6.7 (5.3; 7.7)	5
	31-50	Study	17	4.7 ± 2.3	4.2 (3.3; 5.6)	5
		BCNS	205	6.0 ± 2.9	5.6 (4.9; 6.5)	5
	51-70	Study	7	4.1 ± 2.0	4.2 (1.9; 5.7)	5
		BCNS	249	5.6 ± 3.2	5.3 (4.4; 6.3)	5
Females	19-30	Study	9	4.3 ± 1.8	4.2 (3.1; 6.1)	5
		BCNS	176	4.5 ± 2.7	4.0 (3.4; 4.8)	5
	31-50	Study	39	3.9 ± 2.1	3.6 (2.4; 5.5)	5
		BCNS	266	4.3 ± 3.3	4.0 (3.2; 4.6)	5
	51-70	Study	21	3.4 ± 1.6	3.5 (1.9; 4.6)	5
		BCNS	282	4.3 ± 1.7	4.2 (3.4; 4.7)	5
Food Plus Supplements						
Total Study Sample			97	45.1 ± 31.2	6.0 (3.3; 54)	5
Males	19-30	Study	4	76.9 ± 125.2	19.2 (6.1; 147.7)	5
		BCNS	142	9.8 ± 0.5	7.1 (5.5; 11.5)	5
	31-50	Study	17	20.8 ± 38.5	4.3 (2.9; 14.5)	5
		BCNS	205	10.4 ± 0.7	5.9 (5.2; 8.0)	5
	51-70	Study	7	6.4 ± 5.4	4.7 (3.7; 6.0)	5
		BCNS	249	11.6 ± 2.4	6.0 (4.6; 9.5)	5
Females	19-30	Study	9	24.4 ± 40.7	4.8 (3.6; 31.3)	5
		BCNS	176	8.8 ± 0.7	4.9 (3.5; 10.6)	5
	31-50	Study	39	41.0 ± 109.0	6.0 (2.8; 15.7)	5
		BCNS	266	14.8 ± 1.7	5.0 (3.5; 12.7)	5
	51-70	Study	21	18.3 ± 25.4	4.8 (2.2; 26.2)	5
		BCNS	282	16.6 ± 2.1	5.0 (3.9; 14.3)	5

[1]SD = Standard Deviation
[2]AI = Adequate Intake

Figure C.0.10: Dot plots of pantothenic acid intake from food and food plus supplement sources by age and gender (expressed as milligrams per day)

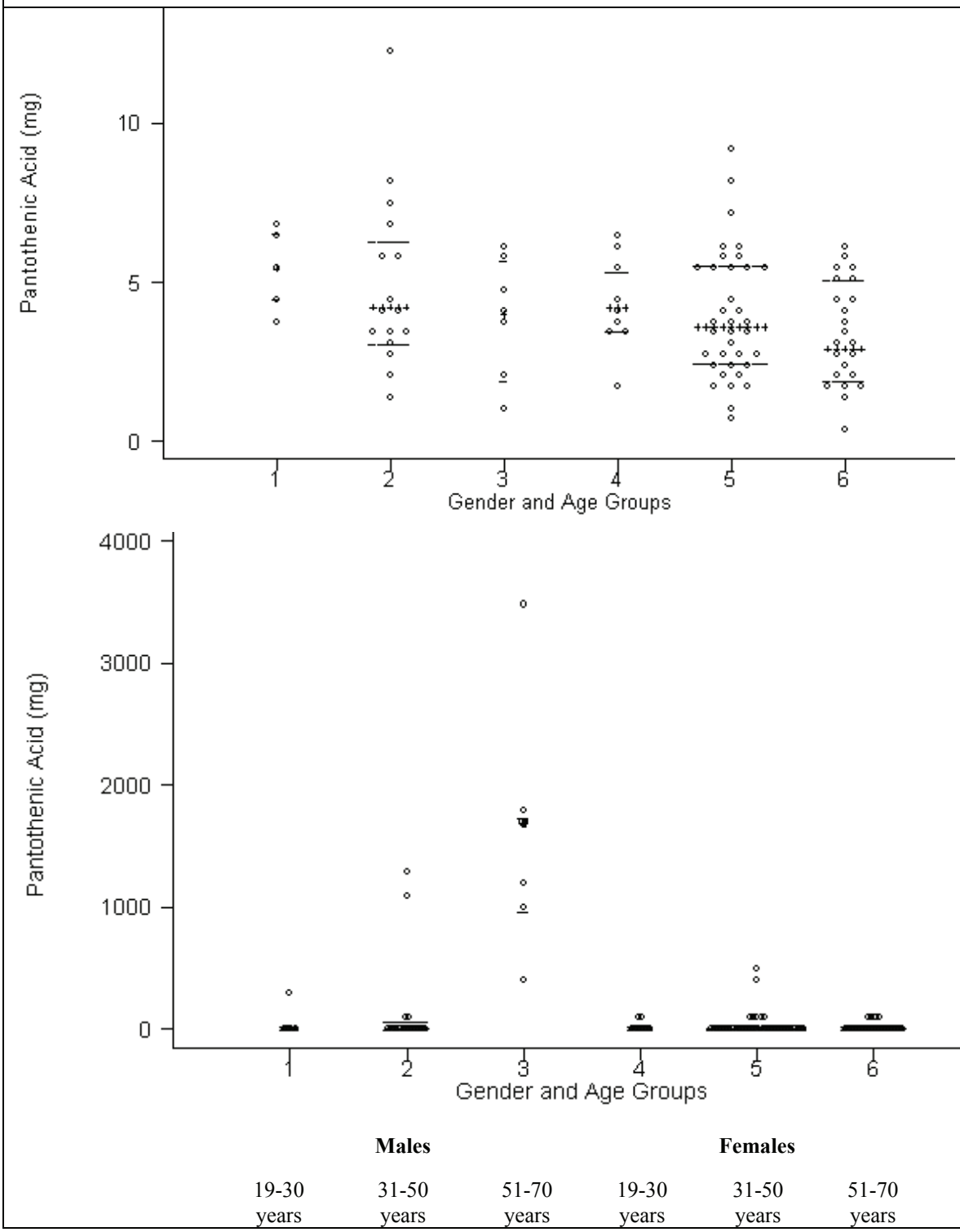


Table C.0.10: Riboflavin intakes from food sources and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	EAR[2]	% < EAR
Food Sources							
Total Study Sample			97	1.7 ± 0.8	1.5 (1.1; 2.2)	--	21
Males	19-30	Study	4	1.7 ± 0.7	1.8 (1.1; 2.2)	1.1	25
		BCNS	142	2.6 ± 1.2	2.3 (1.8; 3.1)	1.1	3
	31-50	Study	17	1.8 ± 0.9	1.6 (1.3; 2.1)	1.1	18
		BCNS	205	2.3 ± 1.4	2.1 (1.8; 2.5)	1.1	3
	51-70	Study	7	1.7 ± 1.0	1.8 (0.5; 2.4)	1.1	29
		BCNS	249	2.2 ± 1.6	2.0 (1.6; 2.4)	1.1	3
Females	19-30	Study	9	1.9 ± 1.2	1.7 (0.9; 3.0)	0.9	1
		BCNS	176	1.8 ± 1.3	1.6 (1.4; 2.0)	0.9	2
	31-50	Study	39	1.8 ± 0.8	1.6 (1.1; 2.6)	0.9	13
		BCNS	266	1.6 ± 1.6	1.5 (1.3; 1.8)	0.9	8
	51-70	Study	21	1.4 ± 0.7	1.5 (0.9; 1.9)	0.9	33
		BCNS	282	1.6 ± 1.7	1.5 (1.3; 1.8)	0.9	4
Food Plus Supplements							
Total Study Sample			97	27.8 ± 116.2	2.8 (1.4; 11.4)	--	12
Males	19-30	Study	4	1.7 ± 0.7	1.8 (1.1; 2.2)	1.1	20
		BCNS	142	4.6 ± 0.5	2.8 (1.9; 4.2)	1.1	2
	31-50	Study	17	12.6 ± 28.5	2.0 (1.3; 3.6)	1.1	13
		BCNS	205	5.9 ± 0.6	2.4 (1.9; 3.4)	1.1	3
	51-70	Study	7	35.8 ± 50.4	2.4 (1.8; 100.5)	1.1	11
		BCNS	249	4.6 ± 0.6	2.2 (1.7; 3.6)	1.1	3
Females	19-30	Study	9	9.3 ± 18.1	1.8 (1.2; 7.9)	0.9	13
		BCNS	176	5.8 ± 0.6	2.2 (1.5; 5.1)	0.9	2
	31-50	Study	39	13.9 ± 26.6	2.9 (1.5; 5.0)	0.9	8
		BCNS	266	10.3 ± 1.3	2.0 (1.4; 5.3)	0.9	6
	51-70	Study	21	11.6 ± 21.2	2.0 (0.9; 6.9)	0.9	19
		BCNS	282	10.0 ± 1.7	2.0 (1.5; 5.2)	0.9	1

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirement

Figure C.0.11: Dot plots of riboflavin intake from food and food plus supplement sources by age and gender (expressed as milligrams/day)

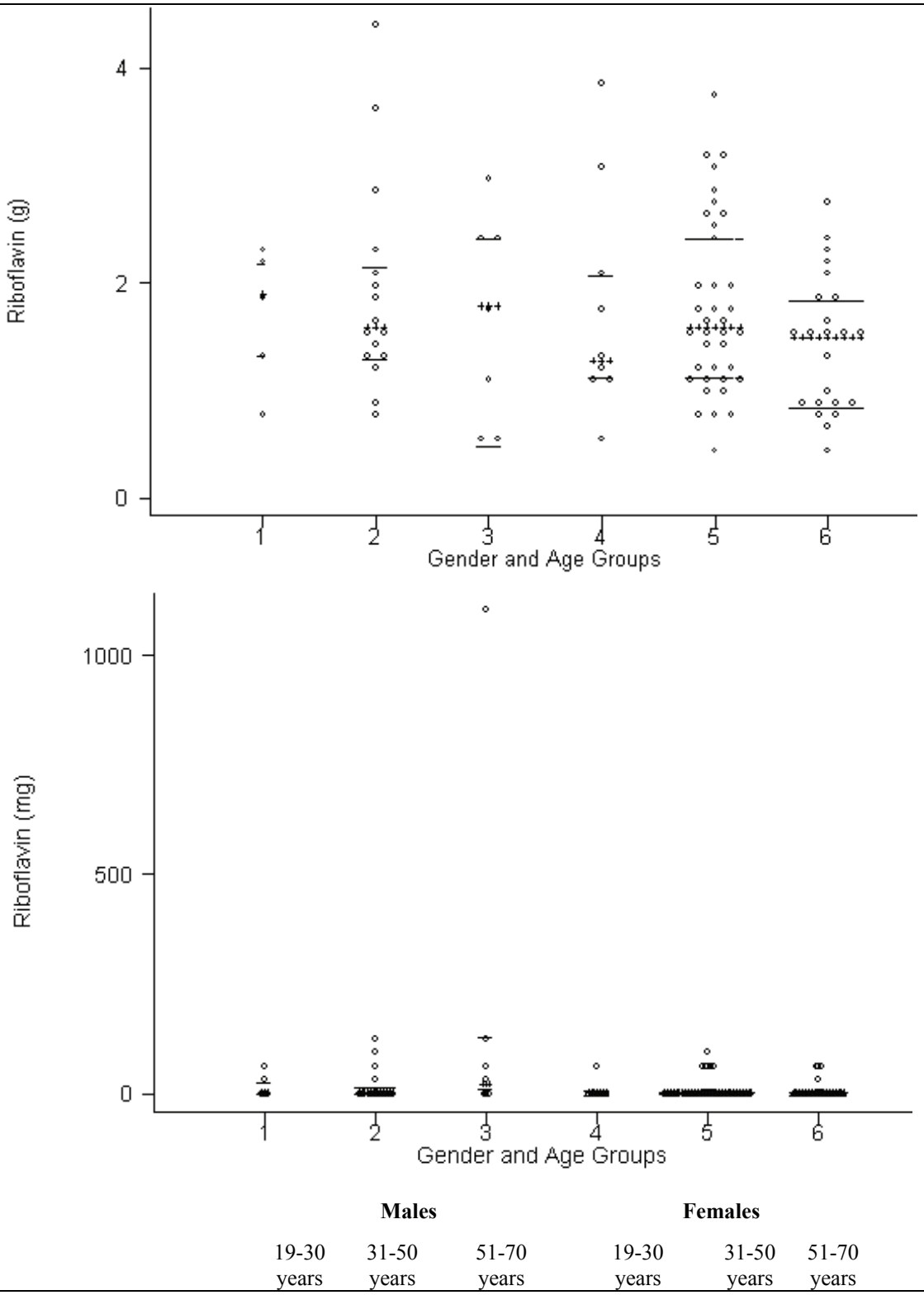


Table C.0.11: Thiamin intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	EAR[2]	% < EAR
Food Sources							
Total Study Sample			97	1.5 ± 0.8	1.3 (1.0; 2.0)	--	26
Males	19-30	Study	4	1.8 ± 0.8	2.0 (1.3; 2.3)	1.0	25
		BCNS	142	2.4 ± 1.2	2.2 (1.6; 2.8)	1.0	3
	31-50	Study	17	1.7 ± 0.8	1.7 (1.3; 2.2)	1.0	12
		BCNS	205	2.1 ± 1.4	1.9 (1.6; 2.3)	1.0	<1
	51-70	Study	7	1.6 ± 1.0	1.7 (1.3; 2.2)	1.0	12
		BCNS	249	2.1 ± 1.6	1.9 (1.4; 2.4)	1.0	5
Females	19-30	Study	9	2.3 ± 1.6	2.0 (1.1; 3.4)	0.9	14
		BCNS	176	1.6 ± 1.3	1.3 (1.1; 1.7)	0.9	7
	31-50	Study	39	1.6 ± 1.0	1.3 (0.9; 2.1)	0.9	36
		BCNS	266	1.4 ± 1.6	1.3 (1.1; 1.6)	0.9	14
	51-70	Study	21	1.2 ± 0.7	1.0 (0.5; 1.5)	0.9	29
		BCNS	282	1.5 ± 1.7	1.3 (1.1; 1.7)	0.9	14
Food Plus Supplements							
Total Study Sample			97	13.8 ± 26.8	2.3 (1.2; 4.7)	--	11
Males	19-30	Study	5	21.1 ± 24.2	14.7 (2.3; 39.9)	1.0	0
		BCNS	142	4.1 ± 0.4	2.4 (1.8; 3.8)	1.0	2
	31-50	Study	16	5.6 ± 13.3	2.2 (1.4; 3.3)	1.0	6
		BCNS	205	5.7 ± 0.6	2.1 (1.7; 3.2)	1.0	<1
	51-70	Study	7	35.5 ± 50.6	1.9 (1.1; 100.5)	1.0	11
		BCNS	249	5.2 ± 0.9	2.1 (1.5; 3.3)	1.0	4
Females	19-30	Study	9	9.8 ± 18.8	2.1 (1.2; 8.1)	0.9	11
		BCNS	176	5.5 ± 0.6	1.9 (1.2; 3.8)	0.9	5
	31-50	Study	39	13.4 ± 26.6	2.3 (1.2; 4.5)	0.9	10
		BCNS	266	10.4 ± 1.3	1.7 (1.2; 4.1)	0.9	11
	51-70	Study	21	11.2 ± 21.1	1.8 (1.0; 5.0)	0.9	19
		BCNS	282	11.2 ± 1.8	1.9 (1.2; 4.4)	0.9	7

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirement

Figure C.0.12: Dot plots of thiamin intake from food and food plus supplement sources by age and gender (expressed as milligrams/day)

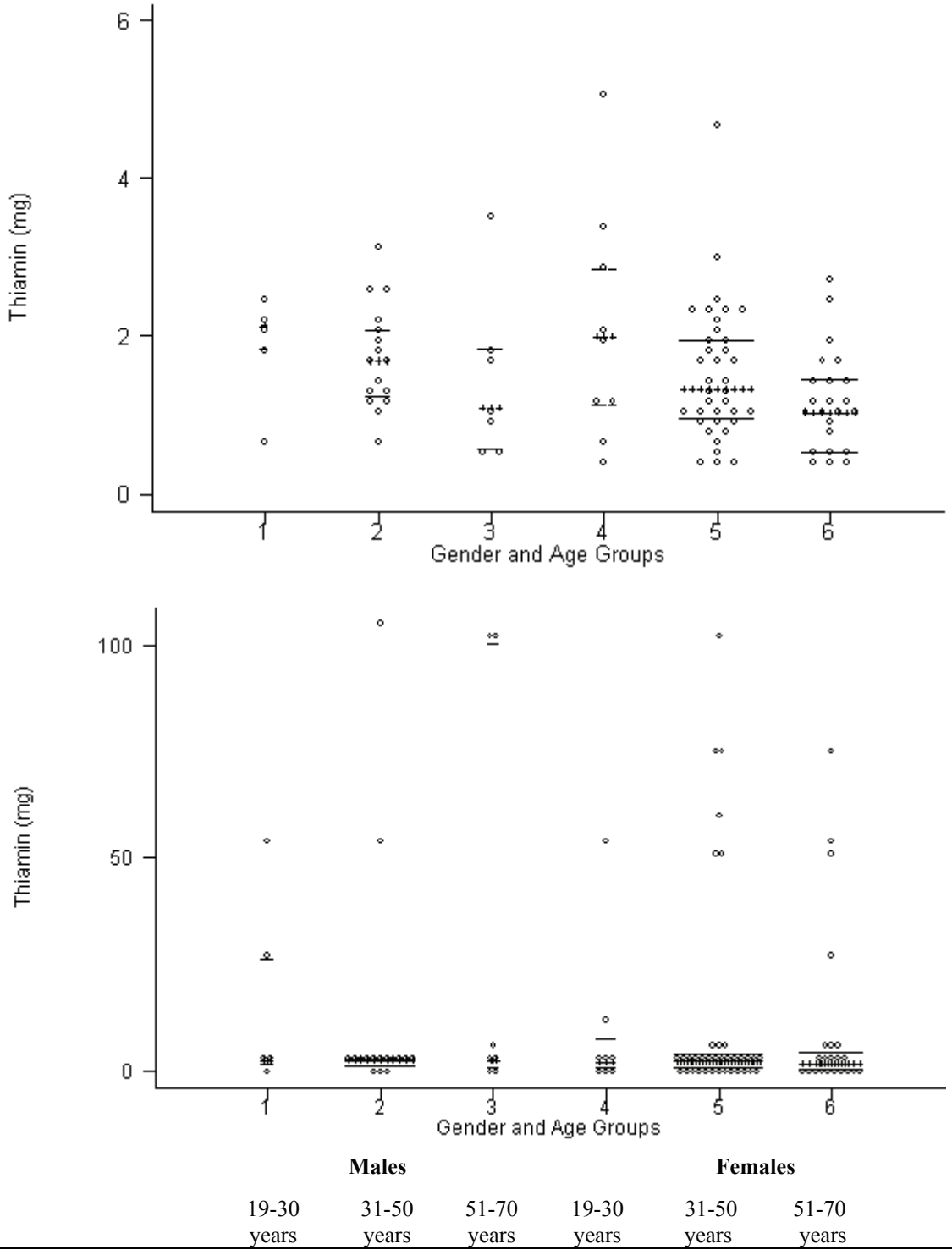


Table C.0.12: Vitamin B_6 intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	EAR[2]	% < EAR	
Food Sources								
Total Study Sample			97	1.7 ± 1.0	1.5 (1.1; 2.1)	--	26	
Males	19-30	Study	4	2.6 ± 0.5	2.4 (2.3; 2.9)	1.1	0	
		BCNS	142	2.6 ± 2.4	2.4 (2.0; 2.9)	1.1	2	
	31-50	Study	17	1.8 ± 0.8	1.6 (1.4; 2.2)	1.1	24	
		BCNS	205	2.2 ± 1.4	2.1 (1.7; 2.3)	1.1	0	
	51-70	Study	7	1.6 ± 0.7	1.9 (0.8; 2.0)	1.4	28	
		BCNS	249	2.0 ± 1.6	2.0 (1.5; 2.3)	1.4	21	
Females	19-30	Study	9	2.1 ± 1.0	2.0 (1.5; 2.5)	1.1	14	
		BCNS	176	1.5 ± 1.3	1.4 (1.2; 1.6)	1.1	16	
	31-50	Study	39	1.8 ± 1.3	1.5 (1.1; 2.2)	1.1	21	
		BCNS	266	1.5 ± 1.3	1.4 (1.2; 1.6)	1.1	19	
	51-70	Study	21	1.4 ± 0.6	1.3 (0.9; 1.7)	1.3	48	
		BCNS	282	1.7 ± 1.7	1.5 (1.3; 1.9)	1.3	29	
Food Plus Supplements								**% > UL**
Total Study Sample			97	23.0 ± 49.4	2.9 (1.4; 7.1)	--	20	8
Males	19-30*	Study	4	22.1 ± 24.9	15.4 (2.9; 41.3)	1.1	0	0
		BCNS	142	4.5 ± 0.4	2.8 (2.0; 3.9)	1.1	2	0
	31-50	Study	17	21.8 ± 55.9	1.9 (1.0; 3.9)	1.1	19	6
		BCNS	205	5.9 ± 0.7	2.2 (1.8; 3.3)	1.1	0	0
	51-70	Study	7	35.7 ± 50.3	2.2 (1.9; 100.8)	1.4	22	33
		BCNS	249	6.5 ± 1.1	2.2 (1.6; 3.6)	1.4	19	0
Females	19-30	Study	9	22.2 ± 41.0	2.0 (1.1; 29.2)	1.1	11	11
		BCNS	176	6.1 ± 0.7	1.7 (1.3; 4.8)	1.1	13	0
	31-50	Study	39	16.8 ± 30.6	3.3 (1.4; 5.7)	1.1	13	5
		BCNS	266	10.5 ± 1.4	1.7 (1.3; 5.3)	1.1	15	0
	51-70	Study	21	27.2 ± 72.6	2.3 (1.1; 5.1)	1.3	38	10
		BCNS	282	11.8 ± 2.0	2.1 (1.5; 6.8)	1.3	16	0

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirement
[3]UL = Tolerable Upper Intake Level
*Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.0001$

Figure C.0.13: Dot plots of vitamin B_6 intake from food and food plus supplement sources by age and gender (expressed as milligrams/day)

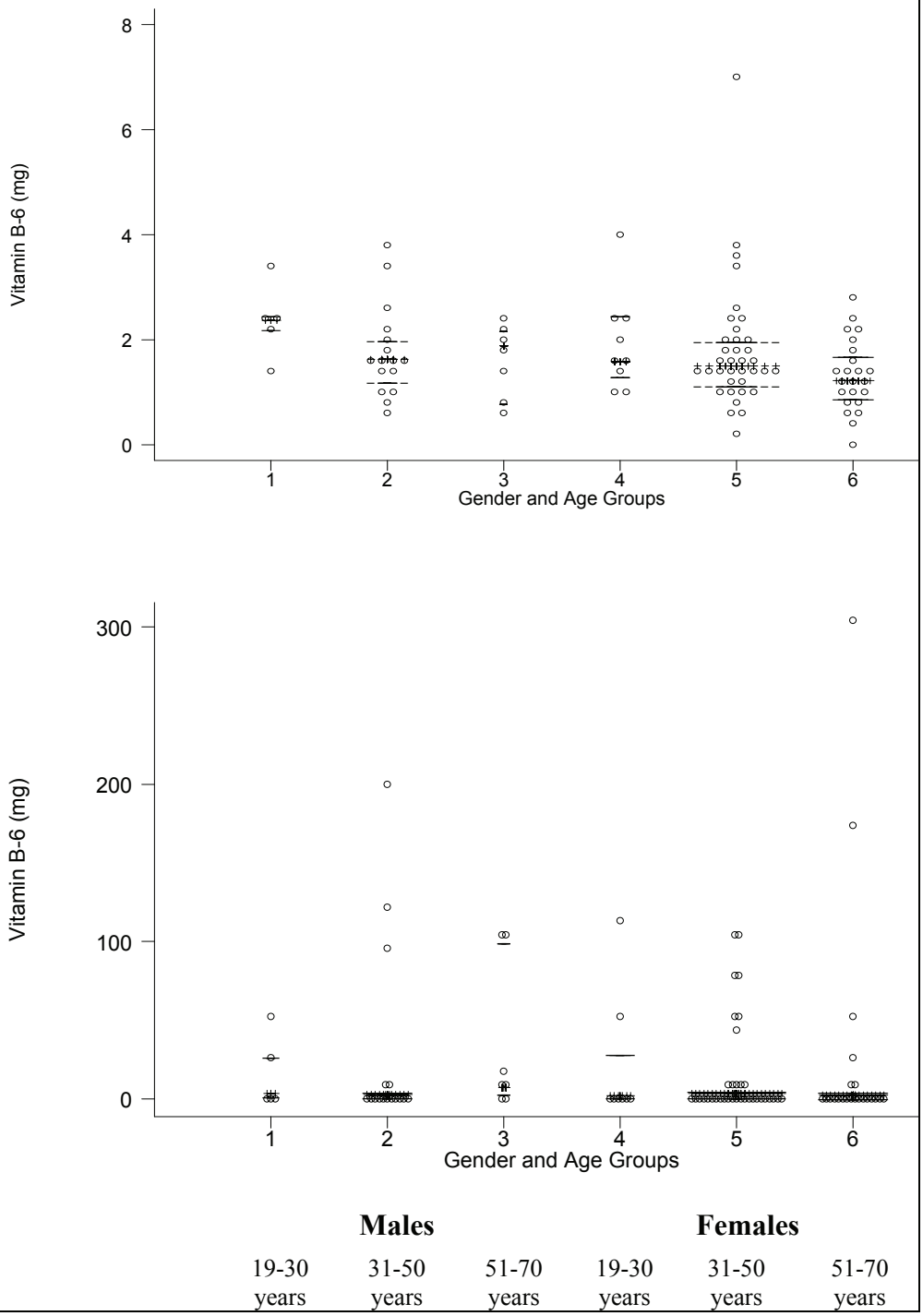


Table C.0.13: Vitamin B_{12} intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as micrograms per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% < EAR[2] (2 mcg/d)
Food Sources						
Total Study Sample			97	4.0 ± 3.6	3.4 (1.9; 3.8)	27
Males	19-30	Study	4	3.7 ± 3.3	3.0 (1.5; 5.0)	25
		BCNS	142	5.9 ± 7.1	4.3 (3.0; 6.2)	7
	31-50	Study	17	3.6 ± 2.1	3.4 (1.7; 4.9)	29
		BCNS	205	6.4 ± 10.0	3.9 (3.0; 5.2)	10
	51-70	Study	7	3.6 ± 2.1	3.5 (2.9; 4.6)	14
		BCNS	249	6.6 ± 17.4	3.4 (2.6; 4.6)	8
Females	19-30	Study	9	4.3 ± 2.5	5.0 (1.9; 6.7)	29
		BCNS	176	4.1 ± 13.3	2.3 (1.8; 3.2)	38
	31-50	Study	39	4.0 ± 4.0	3.3 (1.6; 4.6)	33
		BCNS	266	4.1 ± 9.8	2.5 (2.0; 3.2)	25
	51-70	Study	21	4.5 ± 4.8	3.9 (2.6; 4.4)	14
		BCNS	282	3.9 ± 8.4	2.6 (2.1; 3.2)	20
Food Plus Supplements						
Total Study Sample			97	45.0 ± 139.8	6.7 (2.6; 27.4)	19
Males	19-30*	Study	4	82.2 ± 124.4	30.9 (3.0; 161.4)	0
		BCNS	142	9.9 ± 1.1	5.4 (3.5; 9.1)	5
	31-50	Study	17	26.3 ± 58.3	4.9 (2.3; 8.6)	19
		BCNS	205	33.2 ± 13.8	4.9 (3.3; 9.2)	9
	51-70	Study	7	39.7 ± 50.7	7.2 (4.6; 100.2)	14
		BCNS	249	12.7 ± 2.5	4.3 (2.8; 11.0)	7
Females	19-30	Study	9	26.2 ± 45.8	5.2 (2.1; 34.2)	22
		BCNS	176	11.0 ± 1.4	3.7 (2.0; 11.5)	21
	31-50	Study	39	38.2 ± 92.0	8.7 (3.0; 17.9)	22
		BCNS	266	26.5 ± 13.0	3.5 (2.2; 13.0)	16
	51-70	Study	21	69.4 ± 254.5	4.0 (2.1; 17.9)	14
		BCNS	282	109.5 ± 60	3.9 (2.3; 22.5)	13

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirement
*Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.0001$

Figure C.0.14: Dot plots of vitamin B_{12} intake from food sources and food plus supplement sources by age and gender (expressed as micrograms/day)

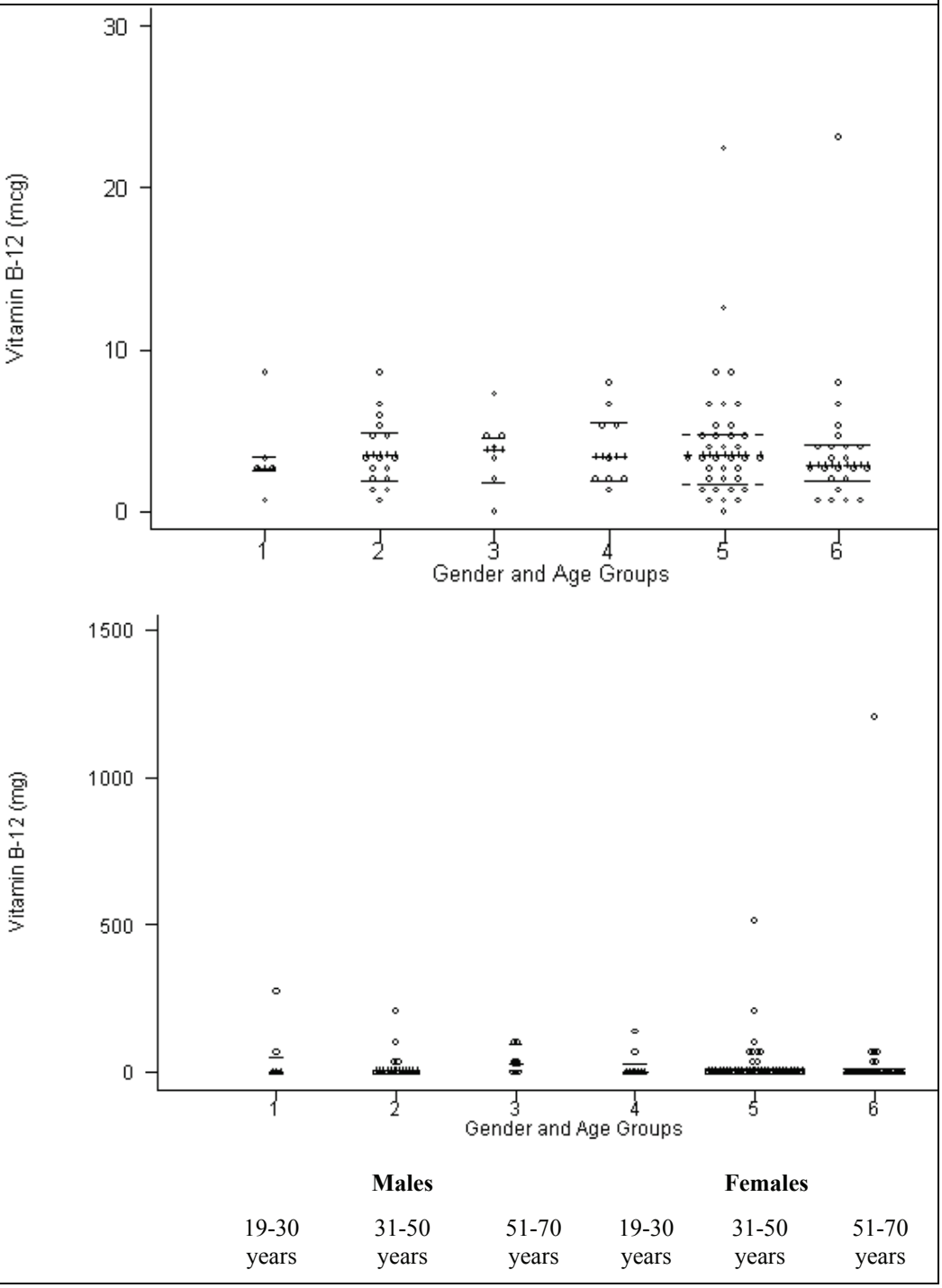
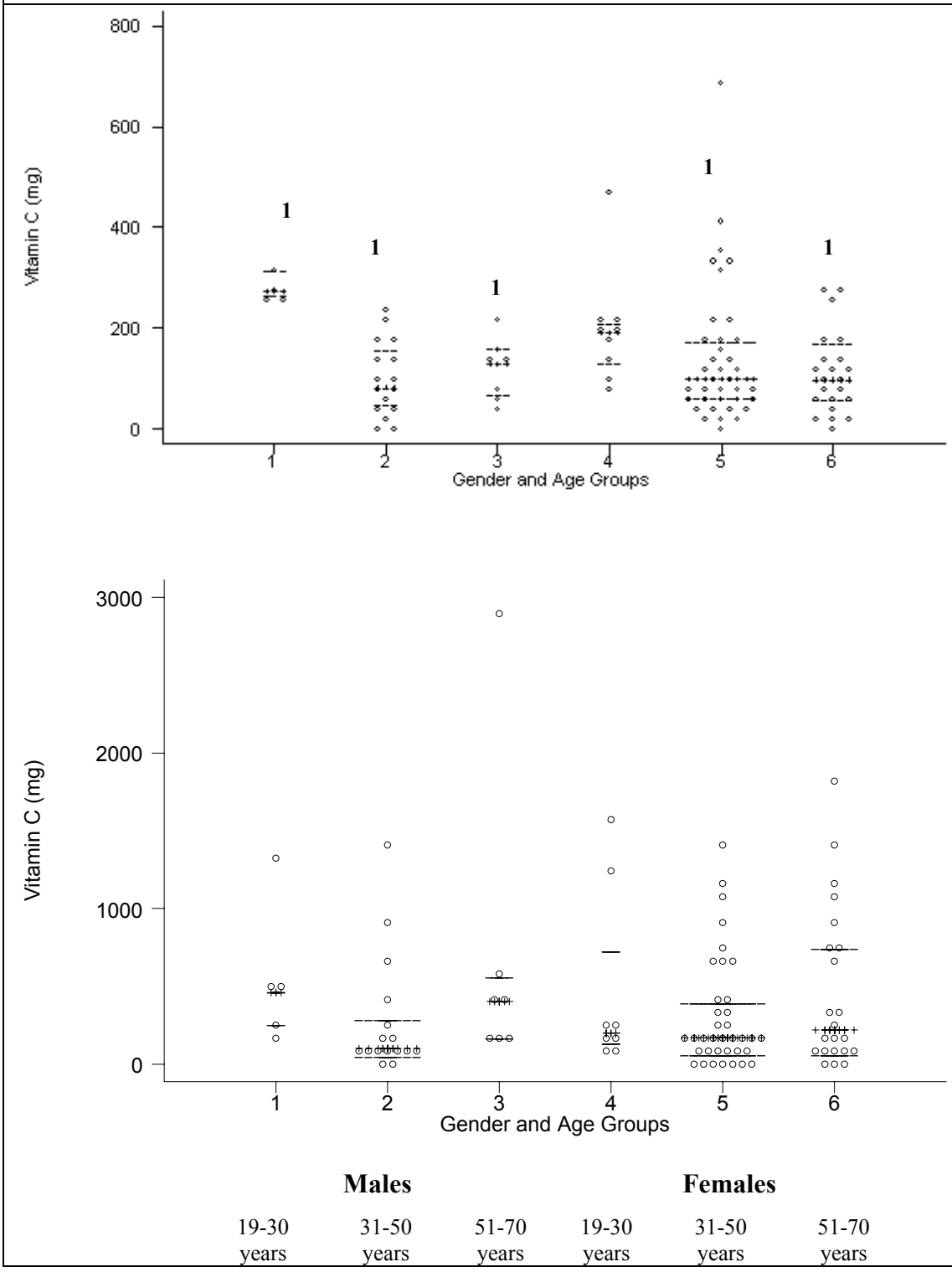


Table C.0.14: Vitamin C intakes from food sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	EAR[2]	% < EAR	
Food Sources								
Total Study Sample			97	142 ± 118	110 (63/ 190)	--	24	
Males	19-30**	Study	4	324 ± 101	288 (257; 391)	75	0	
		BCNS	142	145 ± 238	92 (71; 132)	75	31	
	31-50	Study	17	143 ± 108	100 (48; 217)	75	41	
		BCNS	205	131 ± 143	94 (55; 155)	75	42	
	51-70	Study	7	127 ± 57	131 (71; 160)	75	29	
		BCNS	249	122 ± 142	98 (60; 137)	75	34	
Females	19-30	Study	9	205 ± 130	190 (100; 219)	60	0	
		BCNS	176	105 ± 119	74 (52; 110)	60	33	
	31-50	Study	39	141 ± 132	107 (61; 182)	60	23	
		BCNS	266	110 ± 179	76 (53; 118)	60	32	
	51-70	Study	21	115 ± 87	96 (63; 145)	60	24	
		BCNS	282	121 ± 101	101 (68; 130)	60	20	
Food Plus Supplements								**% > UL[3]**
Total Study Sample			97	397 ± 498	184 (78; 596)	--	18	1
Males	19-30***	Study	4	634 ± 480	466 (363; 904)	75	0	0
		BCNS	142	214 ± 22	119 (75; 208)	75	25	2
	31-50	Study	17	253 ± 397	100 (48; 217)	75	41	0
		BCNS	205	272 ± 19	120 (61; 276)	75	34	<1
	51-70	Study	7	245 ± 305	160 (128; 220)	75	29	14
		BCNS	249	255 ± 26	118 (72; 229)	75	26	2
Females	19-30	Study	9	476 ± 580	198 (141; 736)	60	0	0
		BCNS	176	207 ± 18	126 (63; 219)	60	23	1
	31-50	Study	39	314 ± 358	168 (70; 402)	60	18	0
		BCNS	266	352 ± 33	129 (67; 370)	60	21	3
	51-70	Study	21	470 ± 528	221 (70; 752)	60	14	0
		BCNS	282	356 ± 33	185 (98; 427)	60	11	2

[1]SD = Standard Deviation [2]EAR = Estimated Average Requirement [3]UL = Tolerable Upper Intake Level (2000 mg/day)
**Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.001$
***Differences between study sample and BCNS for this age/gender group were significantly different at $p < 0.0001$

Figure C.0.15: Dot plots of vitamin C intake from food and food plus supplement sources by age and gender (expressed as milligrams/day)



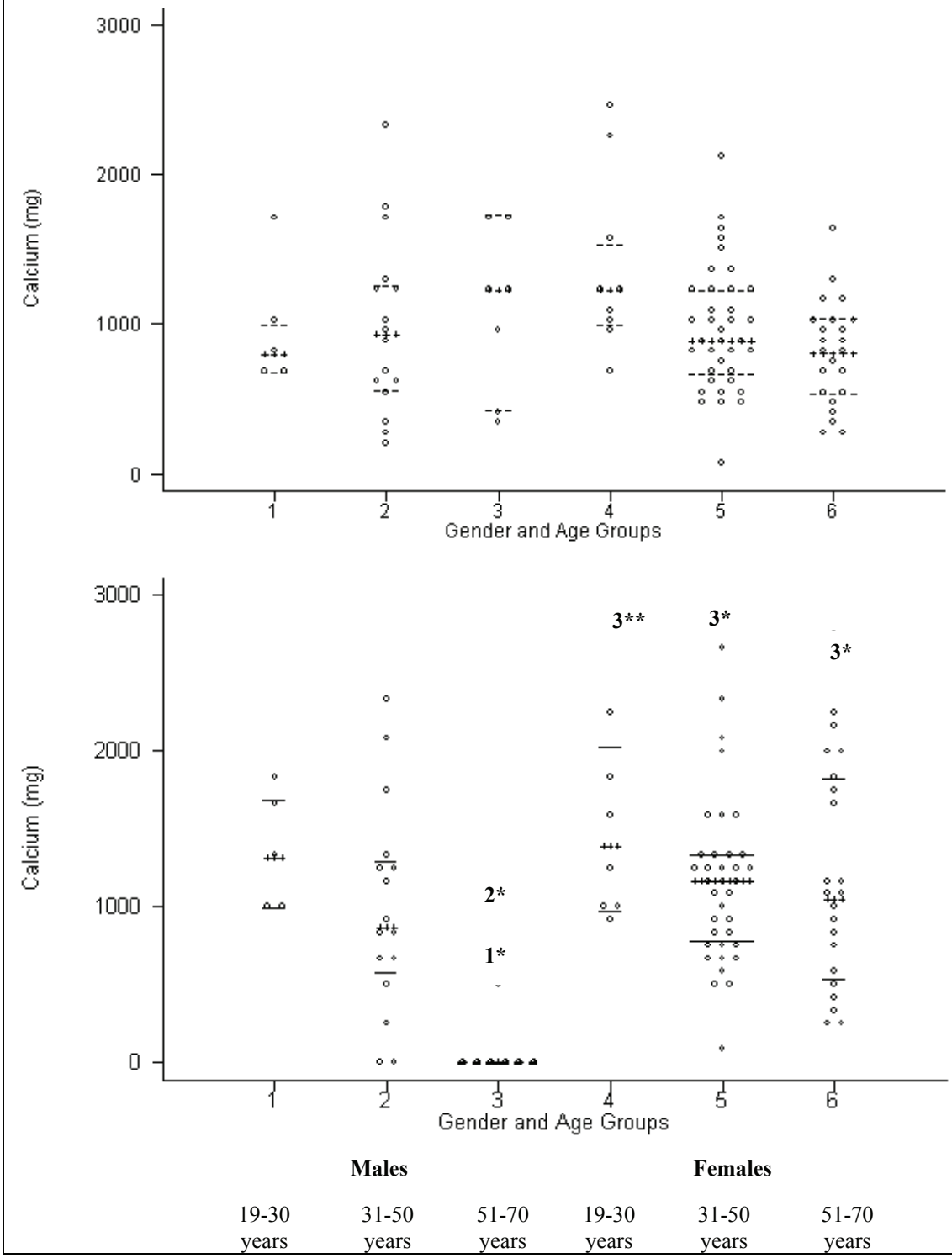
[1]Significant differences found between group 1 and others noted at $p < 0.05$

Table C.0.15: Calcium intakes from food sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	AI[2]	% > UL[3] (> 2500 mg/d)
Food Sources							
Total Study Sample			97	980 ± 477	938 (652; 1231)	--	0
Males	19-30	Study	4	1043 ± 452	898 (740; 1346)	1000	0
		BCNS	142	1193 ± 881	1030 (724; 1377)	1000	1
	31-50	Study	17	957 ± 562	885 (647; 1211)	1000	0
		BCNS	205	980 ± 687	883 (652; 1143)	1000	0
	51-70	Study	7	983 ± 516	1220 (434; 1256)	1200	0
		BCNS	249	858 ± 552	771 (618; 1062)	1200	0
Females	19-30	Study	9	1419 ± 696	1217 (823; 2244)	1000	0
		BCNS	176	876 ± 637	758 (589; 1007)	1000	0
	31-50	Study	39	1018 ± 477	991 (601; 1244)	1000	0
		BCNS	266	750 ± 506	679 (526; 876)	1000	0
	51-70	Study	21	804 ± 345	800 (654; 1001)	1200	0
		BCNS	282	726 ± 537	666 (489; 863)	1200	0
Food Plus Supplements							
Total Study Sample			97	1300 ± 592	1212 (885; 1627)	--	6
Males	19-30	Study	4	1449 ± 367	1499 (1154; 1744)	1000	0
		BCNS	142	1179 ± 34	1049 (739; 1466)	1000	1
	31-50	Study	17	1111 ± 572	957 (687; 1316)	1000	12
		BCNS	205	994 ± 21	906 (721; 1244)	1000	0
	51-70	Study	7	1515 ± 849	1256 (1120; 1739)	1200	14
		BCNS	249	917 ± 25	885 (661; 1123)	1200	0
Females	19-30	Study	9	1580 ± 710	1385 (984; 2031)	1000	11
		BCNS	176	889 ± 27	852 (619; 1067)	1000	<1
	31-50	Study	39	1160 ± 531	2263 (782; 1340)	1000	3
		BCNS	266	873 ± 20	827 (606; 1096)	1000	0
	51-70	Study	21	1207 ± 725	1043 (542; 1829)	1200	5
		BCNS	282	1012 ± 43	863 (593; 1272)	1200	3

[1]SD = Standard Deviation [2]AI = Adequate Intakes [3]UL = Tolerable Upper Intake Level

Figure C.0.16: Dot plots of calcium intake from food and food plus supplements by age and gender (expressed as milligrams/day)



*Significant differences with group number indicated at $p < 0.05$
**Significant differences with the group number indicated at $p < 0.001$

Table C.0.16: Iron intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% < EAR[2]	% > UL[3]
Food Sources							
Total Study Sample			97	18 ± 9	15 (11; 22)	9	3
Males	19-30	Study	4	21 ± 9	19 (14; 28)	0	0
		BCNS	142	20 ± 1	19 (15; 23)	0	0
	31-50	Study	17	18 ± 7	15 (14; 23)	0	0
		BCNS	205	18 ± 1	17 (13; 21)	0	0
	51-70	Study	7	17 ± 9	15 (7; 27)	14	0
		BCNS	249	17 ± 1	16 (13; 20)	1	<1
Females	19-30	Study	9	25 ± 16	21 (12; 38)	33	14
		BCNS	176	13 ± 1	12 (10; 15)	14	0
	31-50	Study	39	19 ± 10	17 (12; 25)	13	3
		BCNS	266	13 ± 0.4	12 (10; 15)	20	0
	51-70	Study	21	14 ± 9	11 (10; 15)	0	5
		BCNS	282	13 ± 1	12 (10; 14)	1	0
Food Plus Supplements							
Total Study Sample			97	70 ± 36	21 (13; 35)	3	7
Males	19-30	Study	5	26 ± 9	28 (19; 33)	0	0
		BCNS	142	21 ± 6	19 (15; 24)	0	<1
	31-50	Study	16	41 ± 79	15 (13; 35)	0	6
		BCNS	205	19 ± 6	17 (14; 23)	0	<1
	51-70	Study	7	19 ± 9	16 (15; 27)	0	0
		BCNS	249	18 ± 6	17 (14; 20)	1	4
Females	19-30	Study	9	33 ± 25	23 (17; 50)	11	22
		BCNS	176	16 ± 9	13 (11; 18)	12	3
	31-50	Study	39	31 ± 50	21 (14; 28)	5	5
		BCNS	266	17 ± 13	14 (10; 19)	16	3
	51-70	Study	21	32 ± 69	16 (11; 23)	0	10
		BCNS	282	14 ± 5	12 (11; 16)	1	0

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirement (males = 6 mg/day; females 19 to 50 years = 8 mg/day and females over 50 years = 5 mg/day)
[3]UL = Tolerable Upper Intake Level

Figure C.0.17: Dot plots of iron intake from food and food plus supplement sources by age and gender (expressed as milligrams/day)

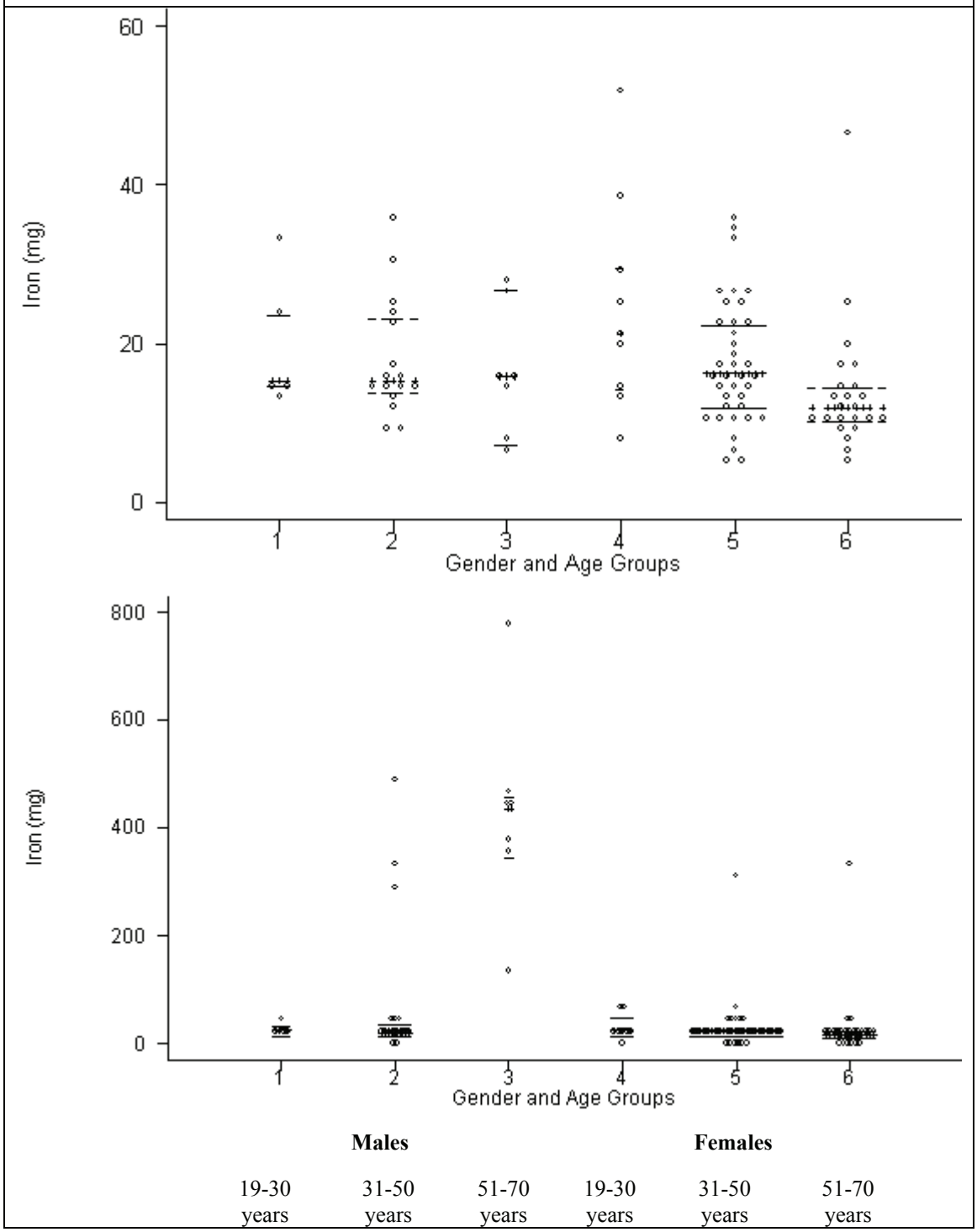


Table C.0.17: Magnesium intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	EAR[2]	% < EAR	
Food Sources								
Total Study Sample			97	309 ± 146	282 (198; 383)	--	46	
Males	19-30	Study	4	443 ± 253	374 (294; 593)	330	25	
		BCNS	142	410 ± 202	396 (323; 475)	330	32	
	31-50	Study	17	292 ± 111	279 (213; 368)	330	71	
		BCNS	205	404 ± 186	383 (343; 430)	350	33	
	51-70	Study	7	291 ± 113	290 (158; 399)	350	71	
		BCNS	249	374 ± 189	371 (303; 426)	350	44	
Females	19-30	Study	9	403 ± 151	405 (247; 543)	255	29	
		BCNS	176	304 ± 172	286 (246; 323)	255	37	
	31-50	Study	39	300 ± 141	272 (198; 405)	265	41	
		BCNS	266	296 ± 163	287 (234; 324)	265	43	
	51-70	Study	21	308 ± 167	294 (148; 383)	265	43	
		BCNS	282	308 ± 185	283 (237; 343)	265	40	
Food Plus Supplements								**% > UL[3]**
Total Study Sample			97	395 ± 252	370 (201; 512)	--	37	6
Males	19-30	Study	4	593 ± 226	599 (399; 788)	330	0	0
		BCNS	142	414 ± 107	410 (323; 482)	330	31	1
	31-50	Study	17	370 ± 207	349 (201; 464)	350	50	6
		BCNS	205	402 ± 86	384 (336; 449)	350	31	<1
	51-70	Study	7	418 ± 174	433 (346; 458)	350	43	14
		BCNS	249	391 ± 126	382 (307; 467)	350	37	1
Females	19-30	Study	9	423 ± 245	387 (206; 559)	255	23	0
		BCNS	176	305 ± 6.0	295 (242l 348)	255	31	1
	31-50	Study	39	380 ± 189	354 (251; 512)	265	31	10
		BCNS	266	336 ± 80	301 (234; 374)	265	36	3
	51-70	Study	21	416 ± 362	311 (163; 528)	265	48	0
		BCNS	282	386 ± 218	326 (264; 447)	265	27	8

[1]SD = Standard Deviation [2]EAR = Estimated Average Requirement
[3]UL = Tolerable Upper Intake Level (Based on intakes from supplements only) (350 mg)

Figure C.0.18: Dot plots of magnesium intake from food and food plus supplement sources by age and gender (expressed as milligrams/day)

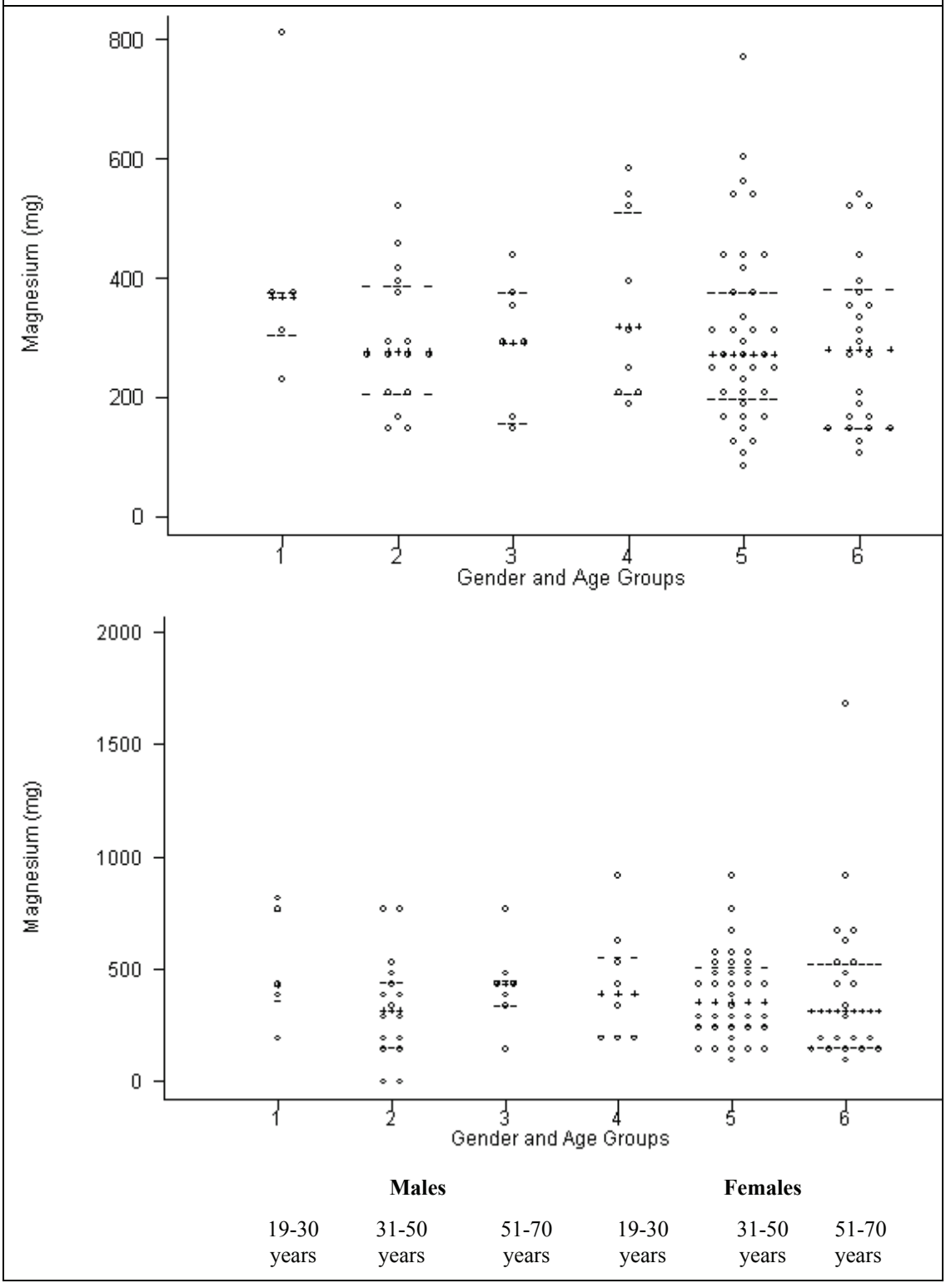
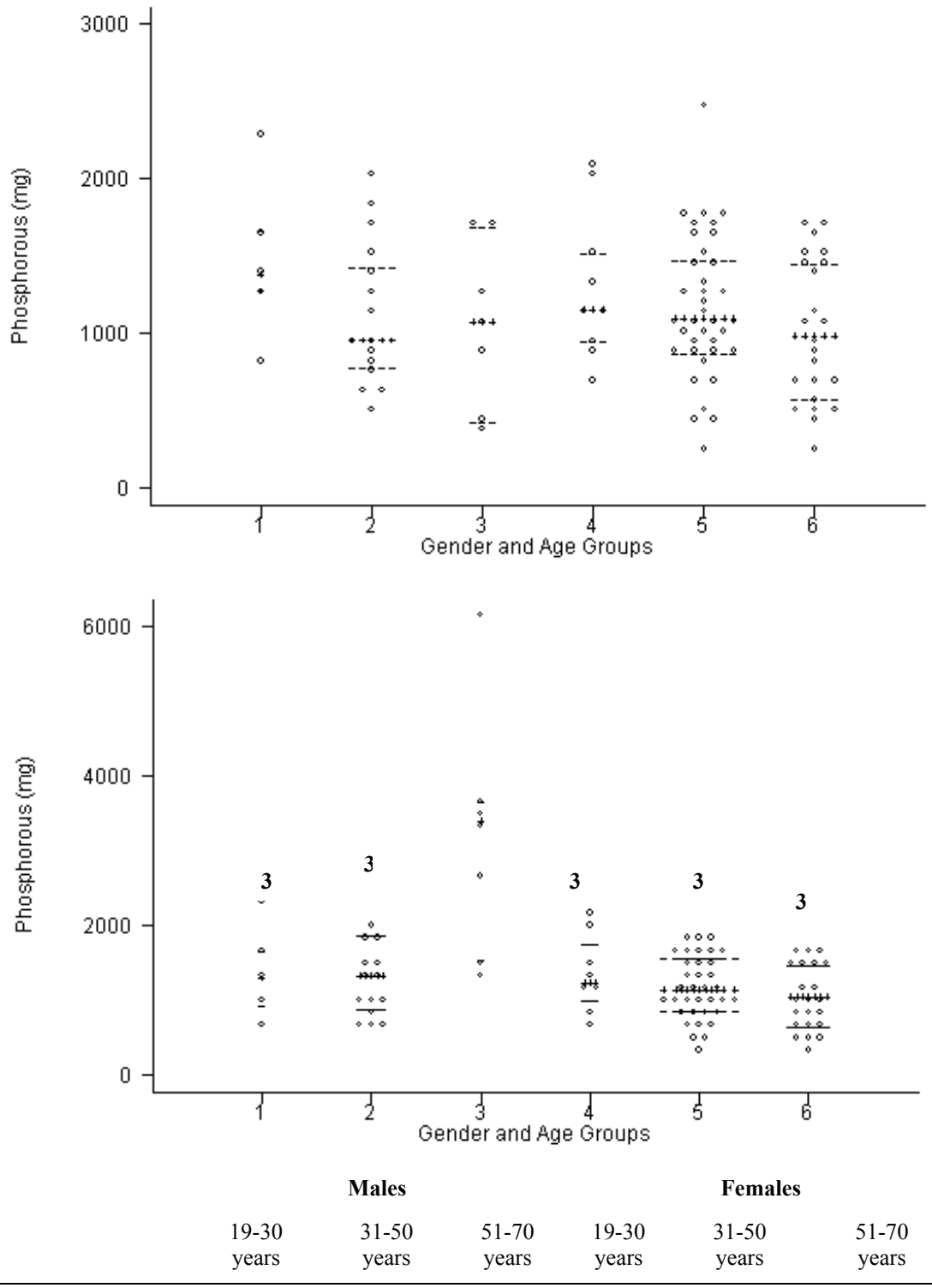


Table C.0.18: Phosphorous intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% < EAR[2]	% > UL[3]
Food Sources							
Total Study Sample			97	1145 ± 475	1083 (836; 1492)	12	0
Males	19-30	Study	4	1516 ± 618	1478 (1055; 1977)	0	0
		BCNS	142	1850 ± 941	1723 (1371; 2127)	0	<1
	31-50	Study	17	1131 ± 453	962 (822; 1386)	6	0
		BCNS	205	1625 ± 716	1589 (1327; 1786)	0	0
	51-70	Study	7	1005 ± 483	1068 (436; 1272)	29	0
		BCNS	249	1492 ± 805	1427 (1183; 1696)	2	0
Females	19-30	Study	9	1410 ± 523	1310 (1083; 2033)	0	0
		BCNS	176	1230 ± 730	1120 (997; 1277)	0	0
	31-50	Study	39	1142 ± 488	1097 (859; 1477)	13	0
		BCNS	266	1151 ± 815	1069 (917; 1240)	2	0
	51-70	Study	21	1070 ± 461	1077 (690; 1460)	19	0
		BCNS	282	1134 ± 605	1065 (927; 1315)	3	0
Food Plus Supplements							
Total Study Sample			97	1365 ± 828	1189 (863; 1649)	9	0
Males	19-30	Study	5	1547 ± 573	1478 (1117; 1977)	0	0
		BCNS	142	1831 ± 512	1725 (1406; 2149)	0	<1
	31-50	Study	16	1179 ± 490	962 (739; 1492)	0	0
		BCNS	205	1595 ± 301	1596 (1352; 1789)	0	0
	51-70	Study	7	1097 ± 484	1068 (896; 1384)	29	0
		BCNS	249	1500 ± 489	1483 (1212; 1715)	2	0
Females	19-30	Study	9	1356 ± 511	1230 (1023; 1776)	0	0
		BCNS	176	1156 ± 212	1130 (1004; 1294)	0	0
	31-50	Study	39	1197 ± 460	1122 (869; 1588)	8	0
		BCNS	266	1122 ± 277	1098 (919; 1260)	2	0
	51-70	Study	21	1035 ± 456	1026 (654; 1090)	19	0
		BCNS	282	1128 ± 353	1102 (931; 1321)	3	0

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirements (580 mg)
[3]UL = Tolerable Upper Intake Level (4000 mg)

Figure C.0.19: Dot plots of phosphorous intake from food and food plus supplements by age and gender (expressed as milligrams per day)



*Significant differences were found between males 51 to 70 years and all other gender/age groups at $p < 0.001$

Table C.0.19: Zinc intakes from food and food plus supplement sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	% < EAR[2]	% > UL[3]
Food Sources							
Total Study Sample			97	10.0 ± 6.2	8.9 (6.1; 12.6)	38	2
Males	19-30	Study	4	13.0 ± 5.0	13.2 (8.8; 17.3)	25	0
		BCNS	142	15.6 ± 9.5	14.0 (11.0; 17.0)	7	0
	31-50	Study	17	10.5 ± 4.4	9.5 (8.0; 12.6)	47	0
		BCNS	205	15.1 ± 12.9	13.0 (11.0; 16.0)	11	<1
	51-70	Study	7	8.7 ± 4.6	8.9 (3.7; 13.6)	57	0
		BCNS	249	14.8 ± 31.6	12.0 (9.4; 15.0)	26	3
Females	19-30	Study	9	16.0 ± 15.4	12.3 (4.8; 16.6)	29	11
		BCNS	176	9.6 ± 5.3	9.0 (8.0; 10.0)	11	0
	31-50	Study	39	10.2 ± 8.1	8.7 (5.4; 11.1)	33	3
		BCNS	266	9.8 ± 6.5	8.8 (7.6; 11.0)	11	0
	51-70	Study	21	9.1 ± 4.8	8.5 (5.8; 12.0)	43	0
		BCNS	282	11.1 ± 25.2	8.7 (7.3; 10.0)	20	1
Food Plus Supplements							
Total Study Sample			97	15.3 ± 16.6	10.6 (6.7; 18.5)	32	6
Males	19-30	Study	5	15.5 ± 9.1	13.2 (8.8; 22.3)	25	0
		BCNS	142	19.1 ± 0.8	15.5 (11.5; 22.9)	7	4
	31-50	Study	16	17.7 ± 20.0	9.9 (8.0; 18.6)	36	6
		BCNS	205	15.7 ± 0.4	13.5 (11.3; 18.0)	11	2
	51-70	Study	7	8.8 ± 4.1	8.9 (6.9; 11.3)	57	0
		BCNS	249	17.1 ± 0.8	13.7 (10.3; 18.0)	18	6
Females	19-30	Study	9	18.3 ± 14.7	14.4 (7.3; 24.4)	22	11
		BCNS	176	13.3 ± 0.9	9.5 (8.2; 12.4)	8	3
	31-50	Study	39	14.2 ± 10.1	11.2 (6.9; 19.5)	23	3
		BCNS	266	13.0 ± 0.5	9.8 (7.8; 14.7)	10	1
	51-70	Study	21	17.9 ± 25.5	7.9 (5.8; 18.5)	43	14
		BCNS	282	14.4 ± 0.8	10.1 (8.0; 17.0)	13	3

[1]SD = Standard Deviation
[2]EAR = Estimated Average Requirements (9.4 mg/d for males; 6.8 mg/d for females)
[3]UL = Tolerable Upper Intake Level (40 mg/d)

Figure C.0.20: Dot plots of zinc intakes from food and food plus supplements by age and gender (expressed as milligrams per day)

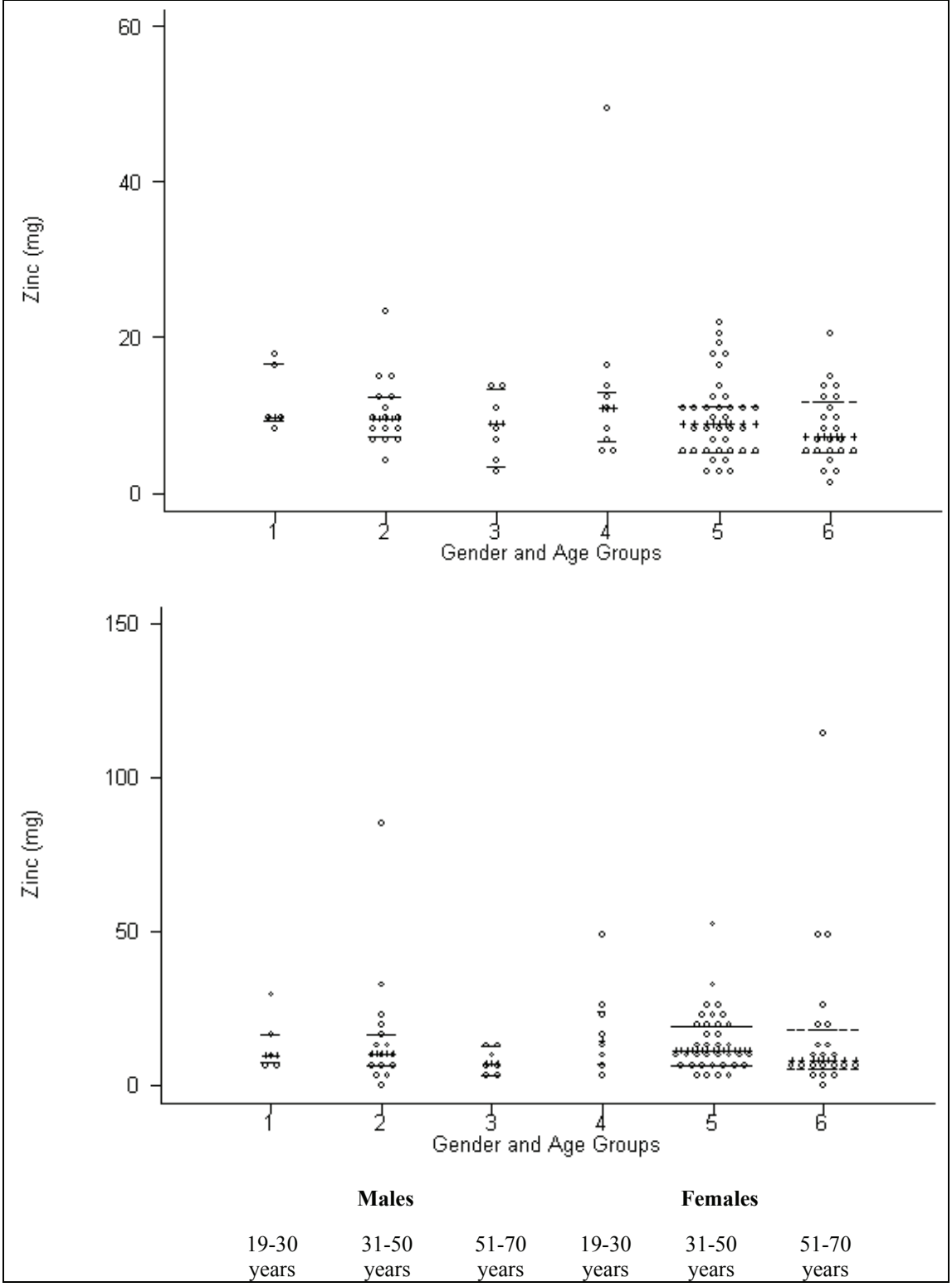
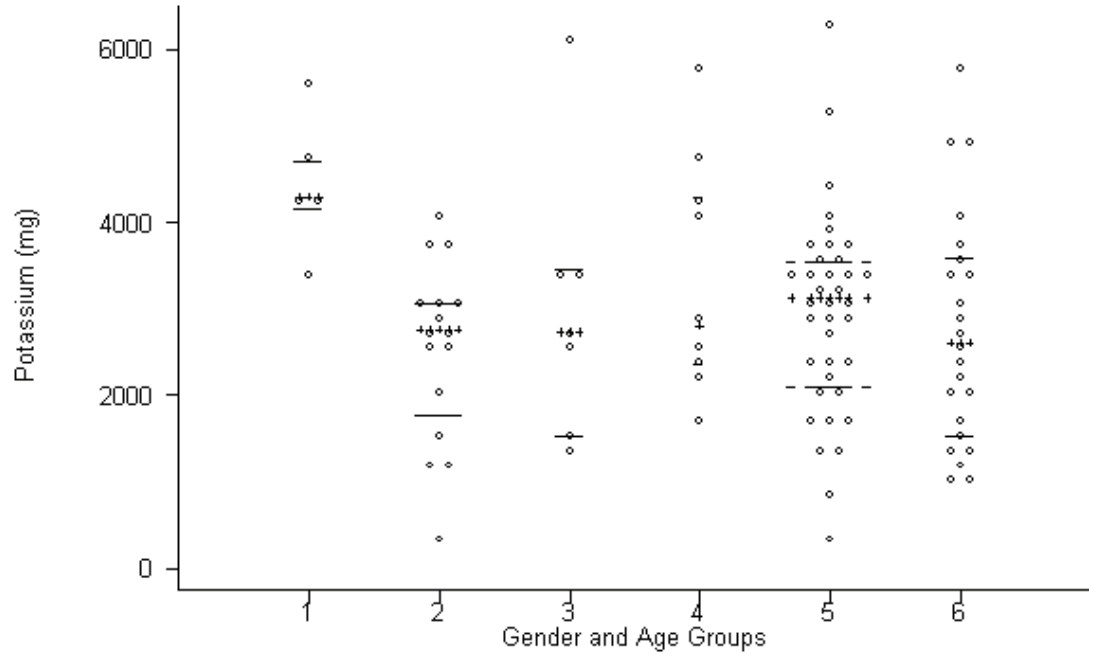


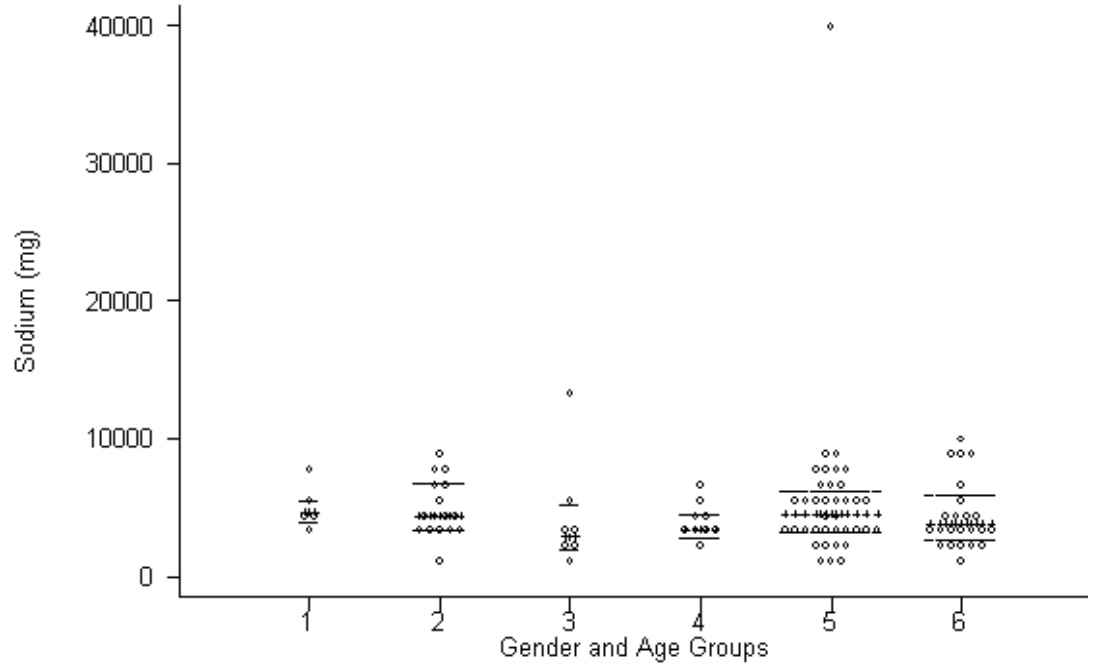
Table C.0.20: Potassium and sodium intakes from food sources by gender and age compared to the British Columbia Nutrition Survey (BCNS) (expressed as milligrams per day)

Sex	Age (years)	Sample	N	Mean ± SD[1]	50th percentile (25th; 75th)	AI[2]
Potassium						
Males	19-30	Study	4	4690 ± 626	4507 (4235; 5146)	4700
		BCNS	142	3800 ± 2192	3536 (2846; 4231)	4700
	31-50	Study	17	2740 ± 1186	2776 (2000; 3334)	4700
		BCNS	205	3594 ± 1560	3456 (2944; 4057)	4700
	51-70	Study	7	3087 ± 1581	2852 (1554; 3675)	4700
		BCNS	249	3509 ± 1894	3443 (2653; 4159)	4700
Females	19-30	Study	9	3748 ± 1390	4097 (2540; 4758)	4700
		BCNS	176	2695 ± 1552	2502 (2242; 2824)	4700
	31-50	Study	39	2682 ± 2391	2962 (2061; 3753)	4700
		BCNS	266	2719 ± 1549	2568 (2145; 2954)	4700
	51-70	Study	21	2770 ± 1225	2739 (2030; 3456)	4700
		BCNS	282	2940 ± 1478	2786 (2354; 3354)	4700
Sodium						
Males	19-30	Study	4	5256 ± 1885	5143 (3907; 6605)	1500
		BCNS	142	4096 ± 2050	3855 (3062; 4628)	1500
	31-50	Study	17	4674 ± 1950	4308 (3589; 5275)	1500
		BCNS	205	3674 ± 2090	3534 (2830; 4174)	1500
	51-70	Study	7	5035 ± 4090	3170 (2157; 7227)	1300
		BCNS	249	3501 ± 2951	3034 (2468; 3866)	1300
Females	19-30	Study	9	3479 ± 1220	3018 (2607; 4576)	1500
		BCNS	176	2695 ± 1552	2605 (2129; 2898)	1500
	31-50	Study	39	5462 ± 5906	4510 (3220; 5996)	1500
		BCNS	266	2593 ± 1386	2442 (1908; 2960)	1500
	51-70	Study	21	4282 ± 2588	3785 (2755; 4518)	1300
		BCNS	282	2271 ± 1662	2092 (1722; 2507)	1300

[1]SD = Standard Deviation
[2]AI = Adequate Intakes

Figure C.0.21: Dot plots of potassium and sodium intake from food by age and gender (expressed as milligrams per day)





Males **Females**

19-30 years	31-50 years	51-70 years	19-30 years	31-50 years	51-70 years