Food and Mood: An Investigation Into the Association Between Diet Quality and Depressive Symptoms

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ABSTRACT

FOOD AND MOOD: AN INVESTIGATION INTO THE ASSOCIATION BETWEEN DIET AND DEPRESSIVE SYMPTOMS

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Background: Research suggests an association between diet and depressive symptoms. This association has not been well examined among emerging adults, a population at increased risk of developing both depression and unhealthy lifestyle habits.

Objectives: To examine the cross-sectional association between depressive symptoms and each of diet quality, fruit/vegetable intake, omega-3 fatty acids, protein, “other foods”, and glycemic index/glycemic load in emerging adult females.

Methods: 141 subjects were recruited at the University of Guelph. Measures of diet, depressive symptoms, and other health behaviours were collected. Descriptive statistics and age-adjusted linear regressions were performed to test for associations between depressive symptoms and diet.

Results: Depressive symptoms were inversely associated with diet quality (r = 0.21, p = 0.043). There were no associations between depressive symptoms and other dietary variables (p > .05).

Conclusion: Results indicate that elevated depressive symptoms are cross-sectionally associated with the consumption of diets of poor nutritional quality.
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LIST OF ABBREVIATIONS

ALA – Alpha-linolenic acid
BBB – Blood brain barrier
BMI – Body mass index
CES-D – Center for Epidemiologic Studies Depression Scale
CVD – Cardiovascular disease
DHA – Docosahexaenoic acid
DRIs – Dietary Reference Intakes
DSM-5 – Diagnostic and Statistical Manual of Mental Disorders, 5th edition
EPA – Eicosapentanoic acid
EWCFG – Eating Well with Canada’s Food Guide
FA – Fatty acid
FFQ – Food frequency questionnaire
GI – Glycemic index
GL – Glycemic load
HEI-C – Healthy Eating Index-Canadian Adaptation
LNAA – Large neutral amino acids
MDD – Major depressive disorder
PCA – Principal component analysis
PUFA – Polyunsaturated fatty acid
SERT – Serotonin transporter
1.0 - INTRODUCTION

Depression is one of the most common and personally and economically distressing medical conditions in the developed world (World Health Organization, 2008). Characterized by chronic low mood, loss of interest in daily activities and a plethora of other debilitating symptoms (e.g., lethargy, feelings of worthlessness), depression has a negative impact on people’s lives (American Psychiatric Association, 2014). Those who experience depression may have difficulties acquiring and maintaining employment, and may struggle to preserve relationships with partners, family, and friends (Benazon & Coyne, 2000; Maurin & Boyd, 1990; Simon, 2003). Depressive disorders can ultimately lead to suicide, the second leading cause of death amongst Canadian young adults (Public Health Agency of Canada, 2011). Incidence of depression peaks between the ages of 25 and 32, with more than half of depressive disorders being diagnosed before this time (Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005).

Emerging adulthood, defined as the period between ages 18 and 25, is an especially unpredictable time in life, in which many in this age group are starting university, beginning new jobs, and moving out of their parents’ homes (Arnett, 2000). It is these stressors unique to this life period that may contribute to the increased incidence of depressive disorders observed for many in this age group (Kuwabara, Van Voorhees, Gollan, & Alexander, 2007).

These life changes may also negatively impact the dietary and other lifestyle habits of emerging adults. Indeed, many emerging adults have been found to consume diets of poor nutritional quality, and rely on processed and convenience foods rather than cooking for themselves (Larson, Perry, Story, & Neumark-Sztainer, 2006). Since researchers have identified emerging adulthood as a time during which enduring lifestyle practices such as diet and physical activity habits are established, this period may provide an opportunity for effective intervention.
with the potential to mitigate the development of these adverse behaviors (Nelson, Story, Larson, Neumark-Sztainer, & Lytle, 2008).

Preliminary research suggests dietary intake may be associated with depressive symptoms. For instance, there is evidence indicating possible associations between intake of protein-, carbohydrate-, and omega-3 fatty acid-rich foods, intake of various micronutrients, and overall diet quality, with depressive symptoms (Akbaraly, Sabia, Shipley, Batty, & Kivimaki, 2009; Christenson & Somers, 1995; Mikolajczyk, Ansari, & Maxwell, 2009; Murakami, Miyake, Sasaki, Tanaka, & Arakawa, 2012; Park, You, & Chang, 2009; Yary & Aazami, 2012). However, only a modest number of studies is available in the literature, most of which use methods that are not the reference standards in their field. This includes the use of food frequency questionnaires (FFQs) rather than three-day food records to quantify dietary intake, which may limit the validity and reliability of any findings. Researchers have also not consistently identified whether dietary and depressive symptom data were collected in the same time frame. Since dietary and appetitive changes in depression have been demonstrated to vary as a function of both the severity and presence of a depressive episode, it is imperative that data for these two variables be collected simultaneously (Paykel, 1977).

Only three studies in the present body of literature have been completed in the emerging adult population (Liu et al., 2007; Mikolajczyk et al., 2009; Park et al., 2009), which have uncovered inverse associations between depressive symptoms and fruit/vegetable (Liu et al., 2007; Mikolajczyk et al., 2009) and meat intake (Mikolajczyk et al., 2009), and a positive association between depressive symptoms and intake of snack and fast foods (Liu et al., 2007). This leaves a considerable gap in this body of literature, as most research has been conducted in adult populations, yet peak incidence of depressive disorder peaks during emerging adulthood.
(Kessler et al., 2005). Therefore, the objective of this research was to further explore the relation between different aspects of the diet and depression among an emerging adult population, improving on current research by using three-day food records to collect dietary data, and ensuring that dietary and depressive symptoms data are collected concurrently.

2.0 – LITERATURE REVIEW

2.1 - Depression

Depression is one of the most debilitating medical conditions worldwide, and is predicted to be the number one most economically burdensome chronic medical condition by 2020 (World Health Organization, 2008). To date, treatment of depressive disorders has focused on medication and psychotherapy, which although effective, cannot treat all cases (Pigott, Leventhal, Alter, & Boren, 2010). It is therefore becoming increasingly clear that a more multifaceted approach to treatment of depressive disorders may need to be taken, including encompassing aspects of physical well-being (Lopresti, Hood, & Drummond, 2013). Depression is a disorder that tends to present early in adulthood, and it is thus logical that prevention strategies should focus on managing risk factors in this group (Kessler et al., 2005; Popa & Ladea, 2012).

Definition and Prevalence

Major depressive disorder (MDD) is characterized by long periods of low mood coupled with a variety of other symptoms, including low self-esteem and loss of pleasure in normal activities (American Psychiatric Association, 2014). The course of MDD is variable in that some patients recover spontaneously after several months, while others are unable to achieve remission even with treatment. Risk of relapse is higher in younger patients, and those who have suffered from severe, recurrent depressive episodes (American Psychiatric Association, 2014).
Youth and young adults are particularly impacted by major depressive disorders, as the average age of onset is estimated to be 25 to 32 years of age (Kessler et al., 2005). An estimated 8.2% of 15 to 24 year olds in Canada have been diagnosed with a mood disorder in the past year (Public Health Agency of Canada, 2011). There is evidence that incidence of depression and mood disorder may be even higher in university students. A systematic review of studies examining the prevalence of depressive disorders in American university students found a mean rate of 30.6% (Ibrahim, Kelly, Adams, & Glazebrook, 2013).

Researchers have indicated that depression rates have significantly increased over the past few decades (Kessler et al., 2003; Klerman & Weisman, 1989). While this change may represent a true increase in prevalence, methodological changes in diagnosis of depressive disorders, as well as a reduction in the stigma associated with a diagnosis of depressive disorder may also help explain this trend (Kessler et al., 2003).

*Diagnosis*

In Canada and the United States, the classification system used to diagnose MDD is the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) (American Psychiatric Association, 2014). The DSM-5 diagnostic criteria for MDD include nine distinct items: depressed mood, loss of interest in daily activities (i.e., anhedonia), substantial weight loss or gain (without intending to lose or gain weight), insomnia or hypersomnia, psychomotor disturbance or impairment, lethargy, feelings of worthlessness, difficulty concentrating, and suicidal ideation or behaviour (American Psychiatric Association, 2014). Persons must experience at least five of the aforementioned symptoms over a continuous two-week timespan; at least one of these symptoms must be depressed mood or anhedonia.
Individuals with depression commonly report bouts of tearfulness, irritability, repetitive over-thinking, anxiety, fearfulness, hypochondriasis, and physical discomfort (American Psychiatric Association, 2014). There is also a relation between depressive disorders and risk of death, which is not completely explained by suicide rates, and may be a function of the reduction in quality of life observed in patients with depression (Seymour & Benning, 2009).

There are several measures used to index people’s overall depressive symptomatology. One such scale is The Center for Epidemiologic Studies Depression Scale (CES-D), a 20-item questionnaire created to measure depressive symptomatology in members of the general public (Radloff, 1977). The CES-D focuses on current mood, and its items were selected based on the diagnostic criteria necessary for depression (i.e., low mood and worthlessness). Respondents are asked how often they felt a certain way, for example “I felt everything I did was an effort”, in the past week. Scores can range from 0 to 60, and any score of 16 or higher is considered be indicative of elevated depressive symptoms. The CES-D is commonly used due to its ease of use for both clinicians and study participants, and its demonstrated validity and reliability (Radloff, 1977).

Pathophysiology and Causes

The pathogenesis of depressive disorders is not currently fully understood. However, it is thought that depression results from a combination of genetic, environmental, and lifestyle influences, which lead to abnormal neurotransmitter metabolism and neuronal function (Friedman & Anderson, 2009). It is widely accepted that there are a number of established and potential causes of depression, many of which go beyond the scope of this research. For the purposes of this literature review, the causes and pathophysiology of depression will be
presented with a focus on biological and neurochemical mechanisms, and environmental factors such as stress and monumental life changes.

Neurotransmitters are chemical messengers synthesized by the brain. Their purpose is to relay messages by traveling across the synapse from one neuron to another (Best, Nijhout, & Reed, 2010). Serotonin, dopamine, and norepinephrine are the neurotransmitters thought to play the most significant role in mood regulation. Serotonin is particularly relevant to the pathogenesis of mood disorders, as it is particularly sensitive to fluctuations in its precursors. Serotonin is formed from the essential amino acid tryptophan, while dopamine and norepinephrine are formed from the non-essential amino acid tyrosine. Both tryptophan and tyrosine compete with other large neutral amino acids (LNAAs) for access to the transport proteins needed to cross the blood-brain barrier (Wurtman & Wurtman, 1979).

In normal serotonin synthesis, tryptophan crosses the blood brain barrier and is taken up by serotonergic neuron terminals, where it is converted into 5-hydroxy-L-tryptophan (Best et al., 2010). This reaction is catalyzed by the enzyme tryptophan hydroxylase and the cofactor tetrahydrobiopterin. 5-hydroxy-L-tryptophan is further converted to 5-hydroxytryptophan, commonly known as serotonin, by the enzyme aromatic amino acid decarboxylase. From here, the serotonin is transported to vesicles, and from there, the synaptic cleft. Serotonin release is dependent on the serotonin concentration, which is determined by auto-receptors in the terminal. The serotonin is either then catabolized and pumped back into the cytosol via the action of serotonin transporters (SERTs), or removed altogether. After release, serotonin is broken down by the enzymes monamine oxidase and aldehyde dehydrogenase to 5-hydroxyindolacetic acid (5-HIAA) (Wurtman & Wurtman, 1979).
Researchers have suggested that abnormalities related to neurotransmitter function and synthesis among patients with depression. For example, individuals with depression may have depleted levels of tryptophan in the plasma and cerebrospinal fluid compared to individuals who do not have depression (Benkelfat, Ellenbogen, Dean, Palmour, & Young, 1994; Van der Does, 2001). There is also evidence that polymorphism of the SERT gene in depression, which may indicate an abnormality in the structure of SERTs among some patients with depression (Caspi et al., 2003). Alterations in neurotransmitter synthesis are one such pathway by which diet is hypothesized to be associated with depressive symptoms (Wurtman & Wurtman, 1979).

Stress related to major life changes has also been demonstrated to potentially play a role in the development of depressive disorders. Both negative and positive stressful life events such as the death of a loved one or purchasing a new home are associated with an increased risk of developing depressive symptoms (Kendler, Karkowski, & Prescott, 1999; Wagner, Compas, & Howell, 1988). Among young adults, major changes associated with the transition into university life such as moving away from home and adjusting to the demands of university, as well as using avoidant coping mechanisms may contribute to increased depressive symptoms (Dyson & Renk, 2006). Other commonly reported stressful life events shown to have a positive relation with depressive symptoms include, but are not limited to, financial difficulties, the ending of a romantic relationship, academic pressures, and worries about the future (Reyes-Rodríguez, Rivera-Medina, Cámara-Fuentes, Suárez-Torres, & Bernal, 2013; Sokratous, Merkouris, Middleton, & Karanikola, 2013).

There have also been suggestions of a relation between lifestyle factors, especially smoking and physical inactivity, and depressive disorders in the literature (Pasco et al., 2008; Sacker & Cable, 2006). The link between diet and depressive symptoms has also been explored,
with intake of protein, carbohydrates, omega-3 fatty acids and various micronutrients, as well overall diet quality, being significantly linked with depressive symptoms in various populations (Akbaraly et al., 2009; Christenson & Somers, 1995; Mikolajczyk et al., 2009; Murakami et al., 2012; Park et al., 2009; Yary & Aazami, 2012).

2.2 - Diet

The relation between diet and depressive disorders has become a recent research focus in the depression literature (Akbaraly et al., 2009; Jacka et al., 2010; Mikolajczyk et al., 2010). This connection has recently garnered interest in the mental health community, with the Canadian Mental Health Association collaborating with Dietitians of Canada on a nutrition care manual titled The Role of Nutrition in Mental Health Promotion and Prevention (Davison et al., 2012). However, research in this field is still in its infancy, and few studies using strong dietary methods have been completed.

Studies to date have included participants from a wide array of demographic groups, which may contribute to the generalizability of current results. However, recent studies have used a range of data collection methods, which may limit the generalizability, and thus potentially the validity and reliability of the present findings. Diet has been examined in the context of overall diet quality, by calculating Eating Well with Canada’s Food Guide servings (Health Canada, 2007), and by quantifying amounts of macro- and micronutrient-rich foods, which makes it difficult to contrast results across studies. For these reasons, the lack of consensus observed in the current literature is perhaps not surprising.

2.2.1 - Overall Diet Quality

The relation between overall diet quality and depressive symptoms is perhaps the most extensively studied association within the area of nutrition and depression. This may be due to
the fact that calculation of a global dietary score allows for the use of food frequency questionnaires (FFQs), a commonly used measure of dietary intake (Willett et al., 1985). The FFQ assesses how often individuals consume certain foods and food groups in a specific time period (Willett et al., 1985). FFQs may be non-quantitative or semi-quantitative, meaning that they provide some information on serving sizes and quantities consumed. This method is comparatively less expensive to administer than the reference standard three-day food record, which measures dietary intake over three 24-hour periods, and usually includes intake from two weekdays and one weekend-day (Crawford, Obarzanek, Morrison, & Sabry, 1994). The ease of use and low cost of the FFQ facilitates its use among larger samples (Willett et al., 1985).

Diet quality is most often examined in the context of dietary patterns, which are usually analyzed using principal component analysis (PCA). In most studies, foods are grouped into either “healthy,” “whole” or “traditional” dietary patterns, or “Western” or “processed” dietary patterns. A healthy dietary pattern tends to contain foods such as fruits, vegetables, whole grains, fish, and lean, non-processed meats, while a Western dietary pattern tends to contain sweets, fried foods, refined grains, and high-fat, processed meats (Akbaraly et al., 2009; Jacka et al., 2010).

Studies using the PCA method have found an inverse relation between a primarily healthy dietary pattern and depressive symptoms. In a five-year prospective study of middle-aged adults, a whole food dietary pattern was found to be significantly and inversely associated with depressive symptoms, while the processed food dietary pattern was found to be significantly and positively associated with depressive symptoms (Akbaraly et al., 2009). A significant association was also found between the maintenance of a processed food dietary pattern consumed at the beginning of the study and higher levels of depressive symptoms five years later.
in participants who had non-elevated levels of depressive symptoms at the study’s commencement (Akbaraly et al., 2009). Similar results have been found among Chinese adolescents (Weng et al., 2011), Australian middle-aged adults (Jacka et al., 2010), Japanese middle-aged adults (Nanri et al., 2010), and French middle-aged adults (LePort et al., 2012).

A possible association between adherence to a Mediterranean style diet and depressive symptoms has also been examined. A Mediterranean diet consists of fruits, vegetables, legumes, fish, and lean meats, and emphasizes cooking with olive oil rather than butter or cream (Sanchez-Villegas et al., 2013). It is commonly recommended to patients who have recently suffered a myocardial infarction. Among older adults, Skarupski, Tangney, Li, Evans, and Morris (2013) found a significant inverse relation between adherence to a Mediterranean diet and development of depressive symptoms over an average period of 7.2 years.

In one of the few randomized controlled trials examining the impact of diet on depression, no association was found between adherence to a Mediterranean diet over a three-year period and incident risk of depression (Sanchez-Villegas et al., 2013). Conversely, when the analysis was restricted to only study participants with type 2 diabetes, a 40% reduction in depression risk among those assigned to the Mediterranean diet group that also supplemented with nuts was noted. However, it is important to note that the primary goal of this study was to examine the impact of the Mediterranean diet among individuals with cardiovascular disease (CVD), and it is therefore unclear how these results would generalize to those without CVD.

Many of the foods associated with the Western dietary pattern (e.g., candies, fried foods, baked goods, processed snack foods) fall into a category Health Canada has labeled “Foods to Limit” (Health Canada, 2007). These are foods that do not belong to any of the four food groups in Canada’s Food Guide, and usually contain high amounts of calories, fat, sugar, and/or
sodium (Health Canada, 2007). Researchers have also suggested a possible relation between intake of these foods and depressive symptoms. A prospective study of adult males and females found high intake of fast-foods to be a significant predictor of increased depressive symptoms (Sanchez-Villegas, Toledo, Irala, Ruiz-Canela, Pla-Vidal, & Martínez-González, 2011), while a cross-sectional study of adult females found that those with increased levels of depressive symptoms were also significantly more likely to consume higher amounts of fast-foods (Crawford, Khedkar, Flaws, Sorkin, & Gallicchio, 2011).

The increased interest in the association between diet and depression suggests there is a growing body of literature investigating the potential mechanisms behind the constituents of each of these foods and their relation to depressive symptoms. The remainder of this literature review will discuss the available studies that attempt to elucidate the role of various macro and micro-nutrients in mood regulation and their possible relation with depressive symptoms.

2.2.2 - Protein and Amino Acids

The link between protein intake and depressive symptoms may be of interest to the mental health community due to the potential role protein plays in the formation of mood-regulating molecules (Wurtman & Wurtman, 1979). Proteins are large molecules comprising amino acid chains and are an essential part of the human diet. They have various functions within the body, including being a major constituent of muscle and other tissues, and helping form cell membranes. When metabolized into individual amino acids, proteins are precursors to hormones, nucleic acids, and various other molecules including the neurotransmitters serotonin, dopamine, and norepinephrine. While some amino acids are synthesized endogenously (and are therefore non-essential), others, including tryptophan, are essential and must be obtained from the diet (Wurtman & Wurtman, 1979).
Protein is theorized to impact mood through the action of the amino acids tryptophan and tyrosine. Tryptophan, an essential amino acid in the human diet, is the precursor to the neurotransmitter serotonin, while tyrosine is a precursor to the catecholamine neurotransmitters dopamine and norepinephrine. Ingested protein is digested into individual amino acids in the small intestine; these amino acids are then absorbed into the bloodstream through the brush border membrane (Wurtman & Wurtman, 1979).

Tryptophan and tyrosine are then transported to the brain, where they are carried across the blood brain barrier (BBB) by transport proteins. There are specific transport proteins for acidic, basic, and neutral amino acids. Since both tryptophan and tyrosine are neutral in charge, they compete with other large neutral amino acids for access to these protein carriers. Rather than the amount of a specific amino acid in circulation, it is the ratio of that amino acid to the sum of the rest of the large neutral amino acids that affects the rate at which it passes through the BBB. Furthermore, tryptophan is the only amino acid that is significantly bound by albumin while in circulation, which further prevents it from gaining access to transport proteins (Wurtman & Wurtman, 1979).

As stated previously, it is the ratio of plasma tryptophan to the other large neutral amino acids rather than the amount of plasma tryptophan itself that impacts the tryptophan concentration in the brain. Since most protein-rich foods have a low tryptophan to LNAA ratio, protein-rich meals have been shown to actually decrease the plasma tryptophan to LNAA ratio (Heninger, Delgado, Charney, Price, & Aghajanian, 1992; Russ, Ackerman, Banay-Schwartz, Shindledecker, & Gerard, 1990; Wurtman, Wurtman, Regan, McDermott, Tsay, & Breu, 2003). For example, a corn-based diet has an extremely low ratio of tryptophan to other LNAAAs, and it has been found to cause a drastic decrease in brain tryptophan concentration (Wurtman &
It has been hypothesized that brain serotonin synthesis is dependent on the amount of available tryptophan (Wurtman & Wurtman, 1979). Thus, protein-rich diets with low tryptophan may lead to lower production of serotonin; this could, in turn, result in an increase in depressive symptoms among some individuals.

Increases in dietary tyrosine do not have the same effects on catecholamine synthesis as tryptophan does on serotonin synthesis, as the hydroxylation of tyrosine occurs under feedback inhibition and the hydroxylation of tryptophan does not. This means that increases in plasma and brain tyrosine do not have a significant impact on catecholamine synthesis, which is in contrast to the acceleration in serotonin synthesis that occurs under tryptophan-rich conditions (Wurtman & Wurtman, 1979). Accordingly, serotonin synthesis is considerably more relevant to the relation between diet and depression than catecholamine synthesis.

The relation between dietary protein intake and depression is complex, due to the fact that dietary tryptophan, and thus protein, is required for serotonin synthesis, yet consuming protein reduces tryptophan’s access to the BBB and reduces the brain’s ability to synthesize serotonin (Wurtman et al., 2003). Existing studies reflect this ambiguity and do not offer conclusive results regarding the link between dietary protein intake and depressive symptoms. It is important to note that rather than quantifying actual protein intake, most studies use intake of protein-rich foods such as meats, eggs, and dairy products a proxy. Dietary data tend to be obtained from food frequency questionnaires, which are unable to accurately quantify intake of individual nutrients (Crawford et al., 1994).

Researchers have found both positive and negative associations between protein intake and depressive symptoms. A cross-sectional study of female college students found that students with high levels of depressive symptoms consumed significantly lower amounts of meat and
poultry than students with lower levels of depressive symptoms (Mikolajczyk et al., 2009). In a similar cross-sectional study, Meyer and colleagues (2013) found that males, but not females, who self-reported a diagnosis of depression consumed 25% less meat and poultry than those who were not depressed. Wolfe and colleagues (2011) found a protective effect of meat and poultry intake on depressive symptoms over a roughly ten-year period as measured by the CES-D in males. However, the same study found high meat intake increased the likelihood of developing elevated depressive symptoms over the same time period in females (Wolfe et al., 2011).

In sum, these results indicate a tentative inverse relation between overall protein intake and depressive symptoms, especially in males. All of the studies utilized large sample sizes and participants of various genders, ages, and ethnic backgrounds, which may contribute to generalizability of the results. At the same time, methodological issues such as the use of FFQs and differing depression scales may limit the validity and comparability of these studies. All of the included studies used FFQs to measure diet, with the exception of Wolfe and colleagues (2011), who used a one-day 24-hour recall.

The relation between red/processed meat intake and depressive symptoms has also been examined in the literature. Although red meat is a high source of protein, it also contains high amounts of saturated fats, which may confound the relationship between the protein present in red meats and depressive symptoms. Only one study, a randomized-controlled trial, found red meat intake to be associated with a decrease in depressive symptoms (Torres & Nowson, 2012). This relation was present only among females, and was noted over a 14-week period. However, it is possible the results of this study were accounted for by the fact that participants in the study were maintaining a DASH-style diet, which is also high in nutrient-rich foods such as legumes, nuts, and fruit and vegetables.
Meanwhile, cross-sectional studies have found positive associations between red meat intake and depressive symptoms. Oddy and colleagues (2009) found that adolescents with high intakes of red meat had significantly increased depressive symptoms. Adults who met the criteria for obesity and consumed higher amounts of high fat/processed meats were 1.18 times more likely to have elevated depressive symptoms, even when controlling for obesity (Buys & Sun, 2013). Again, it may be that not that the link between protein and depressive symptoms is accounted for by the high fat content of red meats.

The conflicting results regarding intake of protein-rich foods and depressive symptoms may be due to a variety of factors (e.g. differences in methods used, differing operational definitions of protein intake), as outlined above. Improved accuracy of results may be achieved by utilizing smaller sample sizes, which may allow for the use of more valid methods of dietary data collection, such as three-day food records (Crawford et al., 1994). Lean meats such as fish and poultry could also be separated from red and processed meats during analyses. Another question that should be addressed is whether the dietary and depressive symptom data were collected within a similar time period. Change in appetite, whether it is an increase or a decrease, is a hallmark symptom of MDD (American Psychiatric Association, 2014). These appetite changes are dependent on the severity of the depression, as well as whether a person is currently experiencing a major depressive episode (Paykel, 1977). It is therefore reasonable to deduce that diet may vary depending on these factors; hence, it may be useful to measure diet at the same time as depressive symptoms. Furthermore, there is considerable disagreement in the time frame of the dietary and depressive symptom collection methods used. FFQs tend to measure intake over the previous months, while measures of depressive symptoms such as the CES-D only examine the previous week (Liu et al., 2007; Mikolajczyk et al., 2009). Finally, it is also possible
that the positive relation between low meat intake and depressive symptoms reported in several studies may be due to individuals with depression consuming fewer calories overall, or in the case of females, replacing protein-rich foods with carbohydrate-rich foods (Oddy et al., 2009).

2.2.3 - Carbohydrates

Carbohydrates, more commonly referred to as sugars and starches, are large, energy-providing molecules that are comprised of carbon, oxygen, and hydrogen atoms. Glucose, fructose, sucrose, and lactose are all carbohydrates found in various foods the human diet, including breads, grains, and rice. Carbohydrates can be either unrefined or refined, meaning that the fiber-rich outer bran coating the outside of the grain has been removed. Overconsumption of refined carbohydrates in humans has been linked to a host of chronic illnesses, including cardiovascular disease, type 2 diabetes, and more recently, depressive disorders (Simon et al., 2003).

Carbohydrates can impact mood by increasing the concentration of brain tryptophan, which results from an increase in the plasma ratio of tryptophan to other large neutral amino acids (Wurtman & Wurtman, 1979). When carbohydrates are ingested, the hormone insulin is released, which causes skeletal muscle and fat cells to take up glucose from the bloodstream. Insulin also increases protein synthesis in muscle tissue, which requires amino acids to be taken in from the blood and a decrease in amino acid release from muscle tissues. However, tryptophan concentration remains relatively constant. This is due to the fact that insulin also causes free fatty acids in circulation to be taken in by tissues as well. Since the concentration of tryptophan bound to albumin in circulation is inversely proportional to the concentration of free fatty acids, a decrease in free fatty acids results in an increase in tryptophan bound to albumin. Tryptophan
bound to albumin cannot be absorbed into tissues with other amino acids, so this results in an increase in the plasma tryptophan to large amino acid ratio (Wurtman et al., 2003).

Even though most of the plasma tryptophan is still bound by albumin, it can be removed at the brain and cause a small, while still appreciable, increase in brain tryptophan. This results in an increase in serotonin availability in the synapses, and thus more serotonin is available for neuronal firing, which may translate into more pleasurable mood (Wurtman & Wurtman, 1995). Accompanying this increase in the rate of serotonin synthesis, and resulting increase in serotonin concentration, is an increase in tryptophan hydroxylase activity. Tryptophan hydroxylase is an enzyme that catalyzes the conversion of tryptophan to 5-hydroxy-L-tryptophan, which is the initial and rate-limiting step in the production of serotonin. The increased tryptophan concentration in the brain also increases the amount of tryptophan hydroxylase, which further increases the rate of serotonin synthesis (Wurtman & Wurtman, 1979).

Unlike catecholamine synthesis, tryptophan synthesis occurs in an open feedback system, meaning that there are limited homeostatic controls on this process. The rate of serotonin synthesis is therefore somewhat proportional to the amount of available tryptophan (Wurtman & Wurtman, 1995). The potential mood lifting ability of carbohydrates may cause some people with depression, especially females, to crave carbohydrates, although this has been disputed in the literature (Wurtman & Wurtman, 1995).

A cross-sectional, cross-national study that examined the relation between sugar consumption and depression found a significant positive correlation between consumption of refined sugars and prevalence of depression in most countries (Westover & Maragnell, 2002). While it is possible another hidden variable is responsible for this relation, it seems that high sugar consumption may be somewhat related to the elevated depression rates reported in
developed countries such as Canada (Westover & Maragnell, 2002), although the direction of this relation remains unclear.

On an individual level, a cross-sectional study that used a non-validated questionnaire to quantify carbohydrate intake found that adult participants with major depression had significantly higher intakes of carbohydrates, especially sucrose, than those without depression (Leibenluft, Fierro, Bartko, Moul, & Rosenthal, 1993). Another study found similar results in that females, but not males, who had at least a moderate level of depressive symptoms consumed significantly more carbohydrates and sugars than participants with low levels of depressive symptoms (Christensen & Somers, 1995). This association is of even greater magnitude due to the use of three-day food records in collecting dietary data. These results are mirrored by another cross-sectional study, in which researchers found that females who had received a DSM-IV diagnosis of depression consumed significantly more high-calorie, sweet foods than those who had not, although FFQs were utilized when quantifying intake (Jeffery et al., 2009). Taken together, there is evidence to suggest that females with elevated depressive symptoms may consume significantly more carbohydrates than those without depression; this relation may not be present among males, however.

The positive association between carbohydrate consumption and improved mood may have a negative impact on patients with depression. For instance, one of the major issues with consuming large amounts of carbohydrate-rich foods is that many of these foods are also calorically dense and high in fat (Wurtman & Wurtman, 1995). Overconsumption of these types of foods can lead to weight gain and obesity, which may further contribute to a person’s depression (Luppino et al., 2010). Obesity has been postulated to have a bi-directional relation with depression; weight gain is associated with increased inflammation (Shoelson, Herrero, &
Naaz, 2007), hypothalamic-pituitary-adrenal axis dysregulation (Belanoff, Kalezhan, Sund, Fleming Ficek, & Shatzberg, 2001), and insulin resistance (Huber, 2008), all of which may increase risk of depression, while depression itself causes an increase in weight, through both the actions of cortisol (Bjorntorp, 1996) and adverse lifestyle habit changes such as increased sedentariness and poor diet.

Another effect of excessive carbohydrate consumption is hyperglycemia, which, when coupled with obesity, can potentially lead to type 2 diabetes mellitus. Type 2 diabetes may be associated with increased depressive symptoms among elderly females. For example, in one study involving females who had received a diagnosis of type 2 diabetes, a higher ratio of carbohydrate to caloric intake was associated with an increased likelihood of having elevated depressive symptoms (Umegaki et al., 2009). Lack of blood glucose control has also been demonstrated to have a potential relation with depression. Jordanian patients with diabetes were significantly more likely to be depressed if they were not self-monitoring their blood glucose or following a diabetic diet (Al-Amer, Sobeh, Zayed, Al-Domi, & 2011).

While there is evidence of a positive relation between carbohydrate intake and depressive symptoms among females (Christensen & Somers, 1995; Jeffrey et al., 2009; Leibenluft et al., 1993), it is clear more research is needed to further elucidate this association. Considering most of the discussed studies utilized FFQs, or in one case, a non-validated questionnaire, to assess carbohydrate intake, the validity of future results would benefit considerably from the use of three-day food record. Simultaneous measurement of diet and depressive symptoms may be especially important when considering carbohydrate intake, due to the potential carbohydrate craving during depressive episodes reported among females (Wurtman
20 & Wurtman, 1995), and the strong relation between both prevalence and severity of depressive symptoms and appetite (Paykel, 1977).

2.2.4 – Omega-3 Fatty Acids

Polyunsaturated fatty acids (PUFAs) are long chain fatty acids that contain at least two double bonds on their carbon chain (Wurtman & Wurtman, 1990). Omega-6 fatty acids and omega-3 fatty acids are both types of PUFAs. Alpha-linolenic acid (ALA), eicosaepentanoic acid (EPA), and docosahexaenoic acid (DHA) are the three main types of PUFA. Both EPA and DHA are essential, meaning the human body cannot synthesize them endogenously (Wurtman & Wurtman, 1990). Humans are able to convert a small amount of ALA to EPA and DHA, but not in sufficient quantities to meet biological needs (Logan, 2003). Females are able to convert more ALA to DHA than males due to the estrogen-dependent nature of hepatic DHA synthesis (Wurtman & Wurtman, 1990). ALA is obtainable from plant sources such as flaxseed and hemp, while EPA and DHA are only present in marine sources such as fish and algae (Logan, 2003).

Omega-3 fatty acids are a key element of normal metabolism, and have been consistently demonstrated to play a role in brain development and maintenance (Wurtman & Wurtman, 1990). Substantial research has explored the association between omega-3 fatty acid intake and depressive symptoms, as well as the possible mechanisms explaining the association. There are also suggestions in the literature that omega-3 fatty acid supplementation may reduce depressive symptoms in conjunction with a selective serotonin reuptake inhibitor, but this extends beyond the scope of this literature review (Safa, Sadr, Talischi, & Boroujerdi, 2013).

The exact link between depressive symptoms and omega-3s has not been completely elucidated, but several hypotheses have been offered. EPA and DHA are essential to proper function of the central nervous system, so much so that the brain is about 20% PUFA in dry
weight (Logan, 2003). Both EPA and DHA are essential building blocks of the phospholipid bilayer of cell membranes in brain cells. The presence of multiple double bonds in the structure of EPA and DHA allow for greater membrane fluidity, which aids in neurotransmitter receptor function and signal transduction, and results in increased serotonin transport and may lead to increased mood (Logan, 2003).

Chronic inflammation is another possible mechanism by which an omega-3 deficiency could be linked with depression (Wurtman & Wurtman, 1990). Omega-3s such as EPA, DHA, and ALA produce anti-inflammatory cytokines, while omega-6s such as arachadonic acid (AA) produce pro-inflammatory cytokines. Depression has been found to be associated with an increased pro-inflammatory response to stress, which may be exacerbated by omega-3 deficiency (Logan, 2003). Omega-3s are postulated to inhibit this pro-inflammatory response by negating the impact of pro-inflammatory cytokines such as TNF-alpha and interleukin-1beta (IL-1B) (Logan, 2003).

However, results of current research are inconsistent, in that relationships tend to differ between sexes and across age groups. While some studies measure the amount of omega-3 in a person’s diet, others use fish intake as a proxy for PUFA intake (Appleton et al., 2007; Oddy et al., 2011). This is complicated by the fact that fish contains high amounts of protein, which may also play a role in the neurotransmitter formation and brain health.

The current literature suggests that there is a significant negative association between omega-3 intake and depressive symptoms when fish is used as a proxy. Both Appleton and colleagues (2007) and Li, Dai, Experi, Dehal, and Zhang (2011) found a significant negative association between fish intake and depressive symptoms among middle-aged males, while Colangelo, He, Whooley, Daviglus, and Liu (2009) found a significant negative relationship
between fish intake and depressive symptoms among females. Two of these studies were prospective in design (Colangelo et al., 2007; Li et al., 2011), while the other was cross-sectional (Appleton et al., 2007). Inconsistent with the remainder of the literature, a similar prospective study found that participants in the highest tertile of fish consumption were significantly more likely to have received a diagnosis of depression after a two-year follow-up period (Sanchez-Villegas, Henriquez, Figueiras, Ortuno, Lahortiga, & Martinez-Gonzalez, 2007). In sum, current research indicates a probable connection between omega-3 intake and depressive symptoms. However, it is important to note the possible confounding effect of protein on the relationship between omega-3 fatty acids present in fish and depressive symptoms.

Research examining the link between measured omega-3 fatty acid intake and depressive symptoms also shows promise of a significant association. Some studies examined omega-3 intake as a whole, while others interpreted separate results for the different sub-types of fatty acid (i.e. EPA, DHA, and ALA). Preliminary research has found evidence that higher intakes of ALA (Lucas et al., 2011), EPA (Colangelo et al., 2009; Meyer et al., 2013), and DHA (Colangelo et al., 2009) are all related to significantly decreased depressive symptoms. The studies completed by Colangelo and colleagues (2009) and Lucas and colleagues (2011) were both prospective studies with 10-year follow-up periods, which strengthens any conclusions drawn by these studies.

There are only two studies finding no link between omega-3 fatty acid intake and depressive symptoms (Giltay, Geleijns, & Kromhout, 2011; Jacka, Pasco, Henry, Kotowicz, Nicholson, & Berk, 2004). One of these was a randomized-controlled trial examining the impact of omega-3 supplementation on risk of depression in patients who had recently suffered a myocardial infarction, which may limit the generalizability of these results outside of this
specific population (Giltay et al., 2011). The other study was a six-year prospective study of healthy individuals, with follow-ups two, four, and six years into the study.

The issues in this body of literature are very similar to those previously discussed, in that future research would benefit from using more valid and reliable methods of data collection and ensure dietary and depression data are collected in the same time period. Future research should also consider the effects of omega-3 fatty acids rather than using fish intake as a proxy.

2.2.5 - Micronutrients

Antioxidants

An antioxidant is a molecule that prevents the oxidation of other molecules. There are many types of antioxidants present in the human body, but common examples include vitamins A, C, and E, which are all easily obtainable from fruits and vegetables. Antioxidant depletion causes oxidative stress, which results in chronic inflammation and can eventually lead to diseases such as cancer and cardiovascular disease (Soory, 2009). Antioxidant status has more recently been linked to depression, although the direction of this association remains somewhat unclear.

Antioxidants are postulated to be connected to depressive disorders in two ways. Firstly, it is possible that having a depressive disorder decreases intake of foods (i.e., fruits and vegetables that are high in antioxidants). This is possibly a function of some patients with depression eating less, while others may not have the energy to prepare meals containing fruit and vegetables (Beydoun, Beydoun, Boueiz, Shroff, & Zonderman, 2013). Secondly, low antioxidant intake may result in increased risk of depression in some individuals. Antioxidants possess anti-inflammatory capabilities that prevent oxidative stress in the body, thus reducing inflammation and potentially having a protective effect on risk of depression (Beydoun et al., 2013)
Fruit and vegetable intake has been consistently correlated with depressive symptoms in the current literature. Cross-sectional research has demonstrated that low fruit and vegetable intake is associated with increased depressive symptoms in adolescent males and females (Oddy et al., 2009) and in female European university students (Mikolajczyk et al., 2009), while fruit intake is significantly inversely correlated with depressive symptoms in male university students (Mikolajczyk et al., 2009), and in Chinese college students (Liu et al., 2007). A similar cross-sectional study of older adults found that high consumption of vegetarian foods was inversely correlated with depressive symptoms (Buys & Sun, 2013). Taken together, it is clear that an inverse relationship between intake of fruits and vegetables and depressive symptoms likely exists, which is probably due to the high levels of antioxidants present in plant foods.

Individually, vitamin A, beta-carotene, and vitamin C have all been shown to be inversely associated with depressive symptoms. This was found by a case-controlled study conducted among a sample of Korean university students (Park et al., 2009), while a dose-dependent relationship between carotenoid intake and depressive symptoms was noted by a cross-sectional study involving American adults (Beydoun et al., 2013). Specifically, a 38% reduction in depression risk was found.

Further research into the relation between antioxidants and depressive symptoms should focus on the direction of the relation between antioxidant intake and depressive symptoms. Since research clearly indicates an inverse link between fruit and vegetable intake and depressive symptoms, future studies should focus on the relation between depressive symptoms and individual antioxidants. This would allow the mechanisms of action to be further elucidated, and potentially identify individual antioxidants that could be effective as an adjunctive treatment in depressive disorders.
B Vitamins

The B vitamins are a group of water-soluble vitamins that play a central role in human metabolism, often acting as co-enzymes in chemical reactions essential for normal functioning. This includes the metabolism of amino acids and carbohydrates, and the synthesis of neurotransmitters. Examples of B vitamins include folate, vitamin B6, and vitamin B12 among others. Folate, vitamin B6, and vitamin B12 are all hypothesized to be connected to depression through their roles in homocysteine, s-adenosyl methionine, and methionine metabolism, which are related to neurotransmitter synthesis (Skarupski, Tangney, Li, Ouyang, Evans, & Morris, 2010).

There are two theories offered to explain how B vitamins may impact depressive symptoms. Firstly, folate, vitamin B6, and vitamin B12 all are all essential to the synthesis of the neurotransmitters serotonin, dopamine, and norepinephrine (Bottiglieri, Laundy, Crellin, Toone, Carney, & Reynolds, 2000). For example, vitamin B6 acts a cofactor in the reaction that converts the amino acid tryptophan to serotonin. Deficiencies in any of these vitamins would result in decreased neurotransmitter production, and potentially an increase in depressive symptoms (Bottiglieri et al, 2000).

Secondly, B vitamins play a crucial role in homocysteine metabolism (Bottiglieri, 2005). Homocysteine, an amino acid that is synthesized in the body and not obtained from protein, is the byproduct of several common reactions in the body. A deficiency in any of the B vitamins may result in hyperhomocysteinemia, which has neurotoxic effects. Elevated levels of plasma
homocysteine have also been implicated in the pathogenesis of depression, due to the oxidative stress exerted on neurons in the brain (Kim, Stewart, Kim, Yang, Shin, & Yoon, 2008).

Studies examining the association between B vitamin intake and depressive symptoms have yielded mixed results, with most research investigating the impact of folate. Folate intake has been found to be inversely associated with depressive symptoms in adult males (Tolmunen et al., 2004), adult females (Beydoun et al., 2010), female university students (Watanabe et al., 2012), male and female early adolescents (Murakami et al., 2010), and in older adults (Payne et al., 2009). Significant associations have also been found between elevated depressive symptoms and low vitamin B6 intake in female university students (Watanabe et al., 2012), male and female early adolescents (Murakami et al., 2010), and in older adults (Payne et al., 2009). A longitudinal study of community dwelling older adults found a protective effect of vitamins B6 and B12 against elevated depressive symptoms over a 12-year period (Skarupski et al., 2010). However, in contrast to other studies (Beydoun et al., 2010; Murakami et al., 2010; Tolmunen et al., 2004; Watanabe et al., 2012), no protective effect of folate was found.

At this time, much of the research investigating the connection between B vitamin intake and depressive symptoms has been cross-sectional in nature (Beydoun et al., 2010; Murakami et al., 2010; Payne et al., 2009; Watanabe et al., 2012), however the studies completed by Skarupski and colleagues (2010) and Tolmunen and colleagues (2004) were both prospective and utilized follow-up periods of 12 years and 10 to 15 years respectively. These findings suggest that there may be generalizability regarding the link between B vitamins and depressive symptoms across ages and life stages, as a significantly negative relationship has been found among children, adolescents, emerging adults, adults, and the elderly.

Zinc
Zinc is an essential mineral in the human diet. It makes up part of hundreds of different enzymes and is the second-most prevalent mineral in the human body after iron. Zinc is postulated to impact mood regulation through several mechanisms, including the prevention of inflammatory processes and the mitigation of oxidative stress (Szewczyk et al., 2008; Szewczyk, Kubera, & Nowak, 2011). It is also an essential component of neurons in the hippocampus and amygdala, which are key regions of the brain for mood regulation (Brown and Dyck, 2004). It is hypothesized that a decrease in neuronal health in these regions may be detrimental to mood regulation. Finally, zinc may also increase levels of brain-derived neutrophic factor, which promotes brain cell health, and positively impact the function of the HPG axis and serotonin activity (Bitanihirwe & Cunningham, 2009).

The link between depressive symptoms and dietary zinc intake has begun to be explored in the literature, and results provide promising evidence for an inverse relation between the two. Zinc intake has been found to be associated with decreased depressive symptoms among males (Yary and Aazami, 2012) and females (Maserejian, Hall, & McKinlay, 2012; Yary and Aazami, 2012). There is also a potential interaction between zinc and selective serotonin reuptake inhibitor (SSRI) usage. Amongst those who are taking SSRIs, moderate to high zinc intake has been found to decrease the likelihood of elevated depressive symptoms by half (Maserejian et al., 2012). However, the only longitudinal study in this area found no link between the two over a 20-year period (Lehto et al., 2013). Similar to most of the studies included in this literature review, it was not specified whether diet was assessed at the same time as depressive symptoms.

In sum, it seems that researchers have demonstrated that there may be a relation between diet and depressive symptoms. The present body of literature includes large studies examining many aspects of the diet among samples across life stages ages, ethnicities, and health statuses,
which suggests the potential of generalizability. While the diversity of participants examined and use of large sample sizes are obvious strengths of the current body of literature, most of these studies suffer from similar limitations. These include the use of measures (such as the FFQ) that are not the reference standard in their field (Crawford et al., 1994) and lack of clarity as to whether or not data on diet and depressive symptoms were collected at the same time. Due to the tendency of appetite to fluctuate depending on the severity of depressive symptoms, validity is greatly improved when both are measured over the same time period (Paykel, 1977). Future research should address these limitations by utilizing more methodologically sound measures to assess diet, such as the three-day food record, and should ensure dietary and depression data are collected simultaneously.

2.3 - Emerging Adulthood

Emerging adulthood is defined as the life stage between ages 18 to 25 (Arnett, 2000), and has been described as a distinct developmental stage in which individuals are at an increased risk for maintaining a variety of unhealthy lifestyle habits (Laska, Pasch, Lust, Story, & Ehlinger, 2009). Arnett (2000) describes emerging adulthood as “...a time of life when many different directions remain possible, when little about the future has been decided for certain, when the scope of independent exploration of life’s possibilities is greater for most people than it will be at any other period in the life course” (p.1). Emerging adulthood is therefore a time of great upheaval, in which independence is discovered and a new, more permanent direction in life commences.

This, coupled with the fact that roughly half of emerging adults are enrolled in a post-secondary institution, means that emerging adults may face a unique set of challenges and stressors with which those in other life stages do not have to cope with (Public Health Agency of
Canada, 2011). The lives of emerging adults are in a constant state of flux, whether it be attending university, living on their own for the first time, or starting a new job (Public Health Agency of Canada, 2011). These continuous changes that are so unique to this life stage can make establishing healthy lifestyle habits challenging. Research indicates that lifestyle habits formed during this time of immense change are likely to remain for life, which means that it is an ideal time for interventions geared towards changing lifestyle habits (Nelson et al., 2008).

As previously discussed, emerging adults are potentially at an increased risk of developing a depressive disorder (Statistics Canada, 2013). Most diagnoses of MDD and other depressive disorders come either during or before this important developmental stage, and it is therefore a crucial time for effective for prevention and intervention (Kessler et al., 2005). The high prevalence of depression and other mood disorders observed in this age group have led to concerning suicide rates. Indeed, suicide is the second leading cause of death amongst Canadian young adults aged 20 to 29, with about 800 young Canadians committing suicide in 2007 (Public Health Agency of Canada, 2011). University students have also been shown to be under increased stress and have poorer emotional well-being than the rest of the general population. This is due to a variety of factors, including the stress of moving away from home for the first time, coupled with high academic expectations (Adlaf, Gliksman, Demers, & Newman-Taylor, 2001; Stallman, 2010).

Besides having an increased likelihood of developing a depressive disorder, emerging adults are also at risk of maintaining unhealthy dietary habits, and thus for being overweight or obese. Results of the 2009 Canadian Community Health Survey indicated that 29.4% of 12 to 19 year olds and 42.7% of 20-29 year olds are either overweight or obese, and that obesity rates among young adults have increased from 3% in 1978/1979 to 15% in 2007-2009 (Public Health
Agency of Canada, 2011). Furthermore, a five-year longitudinal study of adolescents transitioning into young adulthood found a significant increase in overweight and obesity over this time period (Gordon-Larsen, Adair, Nelson, & Popkin, 2004). Given the time elapsed since this study was conducted and the increase in the prevalence in obesity seen since its publication (Gotay et al., 2013), it is likely that this trend has become even more prominent.

The increase in obesity observed in the emerging adult population seems to be at least partly a function of poor dietary habits. A study of American young adults noted that those in this age group tend to consume diets insufficient in calcium, green and yellow vegetables, fruits, and whole grains, while consuming excessive amounts of fats (United States Department of Health and Human Services, 2000). This may be due to emerging adults also consuming a significant portion of their meals from fast food restaurants and convenience foods, such as candy bars, potato chips, and pre-packaged frozen meals (Larson et al., 2006). These foods are generally low in vitamins and minerals and higher in fat and sugars. Many emerging adults may be too busy or lack the skills necessary to prepare meals themselves, and those who live in university residences are usually without access to a kitchen.

However, researchers have indicated that emerging adults who frequently prepare their own meals are significantly more likely to consume appropriate amounts of fruits, vegetables, whole grains, calcium, and fats (Larson et al., 2006). This may be of potential significance to the relation between diet and depressive symptoms in this population, as many of the symptoms of depression, including lack of energy, loss of interest in activities of daily life, and changes in appetite, make preparing meals and maintaining healthy dietary habits difficult (American Psychiatric Association, 2014).
Besides dietary factors, depressive disorders and stress have been associated with various other lifestyle factors prevalent in emerging adults and university students. For example, emerging adults are more likely than other age groups to maintain detrimental lifestyle habits including physical inactivity, insufficient or poor quality sleep, binge drinking, and smoking (Von Ah, Ebert, Ngamvitroj, Park, & Kang 2004). Many of these same risky behaviors have also been demonstrated to be associated with increased depressive symptoms in emerging adults (de Wit et al., 2010; Regestein et al., 2008; Roberts, Glod, Kim, & Houchell, 2009; Sebena et al., 2012; Taliaferro, Rienzo, Pigg, Miller, & Dodd, 2008; Vallance et al., 2011).

While this literature review has considered the link between diet quality and depressive symptoms in adults, emerging adults are under-represented in this field of literature. Only three studies (Liu et al., 2007; Mikolajczyk et al., 2009; Park et al., 2009) have examined the relation between diet and depressive symptoms in this age group. This lack of research is surprising, given the increased risk in emerging adults for development of depression, and their propensity for maintaining poor quality diets. Research with a focus on understanding the link between both diet quality and individual components of the diet with depressive symptoms in emerging adults is needed to help inform appropriate intervention strategies for this age group.

3.0 – RATIONALE, OBJECTIVES, & HYPOTHESES

Rationale

Depression is a prevalent mental disorder, and is characterized by chronic low mood and loss of interest in daily activities (American Psychiatric Association, 2014). It is the second most debilitating medical condition in the world, and its ramifications are felt at both a personal and population level (World Health Organization, 2008). Emerging adults are at significantly
increased risk of developing a depressive disorder, as the peak age of diagnosis is during this life stage (Kessler et al., 2005).

The relation between diet and depression has garnered recent interest within the research community, with preliminary evidence suggesting possible associations between intake of protein, refined carbohydrates, omega-3 fatty acids and micronutrients, and overall diet quality, with depressive symptoms (Akbaraly et al., 2009; Christenson & Somers, 1995; Mikolajczyk et al., 2009; Murakami et al., 2012; Park et al., 2009; Yary & Aazami, 2012). Many of these associations are present in both sexes while the relation between refined carbohydrates and depressive symptoms is predominantly found among females (Christensen & Somers, 1995; Crawford et al., 2011; Jeffery et al., 2009).

Research has also investigated the potential mechanisms of these links. Both carbohydrate and protein intake have been demonstrated to be associated with serotonin synthesis. Protein acts as a source of the amino acid tryptophan, which is needed for serotonin formation, while carbohydrates increase the availability of tryptophan to the brain through the action of insulin (Wurtman et al., 2003; Wurtman & Wurtman, 1979). Omega-3 fatty acids are thought to influence depressive symptoms through several mechanisms, including the reduction of inflammation (Logan, 2013), while micronutrients act as cofactors in neurotransmitter formation, and also prevent inflammation (Beydoun et al., 2013; Bottiglieri et al., 2000).

The available literature is also not without its limitations. Dietary data have almost exclusively been collected using FFQs, weakening the validity, and thus the generalizability, of findings (Crawford et al., 1994). Most of the current research has also failed to collect data on diet and depressive symptoms simultaneously. Appetitive change is a symptom of depression (American Psychiatric Association, 2014), and fluctuates depending on the severity of the
depression (Paykel, 1977). It is therefore important that diet and depressive symptoms be measured over the same time period, as diet is likely to vary depending on appetite.

Most of the current research has been completed among middle-aged adults and children. This gap is problematic for several reasons. Emerging adults are at a stage in which they are experiencing life-altering changes, including beginning university, starting a new job, and moving out of the family home (Arnett, 2000). This means emerging adults are in a state of flux, rendering the maintenance or initiation of healthy dietary habits a challenge. Indeed, research indicates that diets consumed by emerging adults are generally poor in nutritional quality, and tend to be high in processed and convenience foods (Larsen et al., 2006; United States Department of Health and Human Services, 2000). This is worrisome, as the lifestyle habits of young adults track into later adulthood (Nelson et al., 2008). The fact that emerging adults are beginning to develop lifelong dietary habits, coupled with the association between diet and depressive symptoms observed in the literature, provides an interesting opportunity for examining the relationship between diet and depressive symptoms.

**Objectives**

1. The primary objective is to examine the association between overall diet quality and depressive symptoms among emerging adult females.

2. The secondary objective is to examine the association between specific dietary components (percent energy from protein, omega-3 fatty acids, glycemic index/glycemic load, number of servings of fruit and vegetables, and percent energy from “other foods”) and depressive symptoms among emerging adult females.
Hypotheses

1. It is hypothesized that diet quality will be inversely associated with depressive symptoms among emerging adults (Akbaraly et al., 2009; Jacka et al., 2010; Kuczmarski et al., 2010; LePort et al., 2012; Nanri et al., 2010; Sanchez-Villegas et al., 2013; Weng et al., 2011).

2. It is hypothesized that each of percent energy from protein (Mikolajczyk et al., 2009; Torres & Nowson, 2012; Wolfe et al., 2011), omega-3 fatty acid (Colangelo et al., 2009; Lucas et al., 2011; Meyer et al., 2013), and fruit and vegetable intake (Beydoun et al., 2013; Liu et al., 2007; Mikolajczyk et al., 2009; Oddy et al., 2009; Park et al., 2009) will be inversely associated with depressive symptoms.

3. It is hypothesized that each of glycemic index/glycemic load (Christenen & Somers, 1995; Crawford et al., 2011; Jeffrey et al., 2009; Leibenluft et al., 1993; Sanchez-Villegas et al., 2011) and percent of energy from “other foods” (Akbaraly et al., 2009; Crawford et al., 2011; Jeffrey et al., 2009; Liu et al., 2007; Sanchez-Villegas et al., 2011) will be positively associated with depressive symptoms.

4.0 - METHODS

4.1 - Study Design

This research is a secondary data analysis from the study “Reaching Out Club (ROC): Support and Experiential Learning for Academically Struggling Students in Early Year Nutrition Courses”, the goal of which was to increase student engagement and improve academic outcomes through both activities and study sessions. A total of 175 undergraduate students participated in this study. Interested students attended an information session about the study, at which they provided written informed consent (Appendix). All participants were given the
option to have their data withheld from analysis, however none chose to do so. Approval from the University of Guelph Research Ethics Board was obtained (Appendix).

4.2 - Sample

The sample included 175 undergraduate students (19 male, 156 female) enrolled in undergraduate nutrition courses. Data were collected between September 2012 and April 2013 at the Body Composition and Metabolism Lab at the University of Guelph. Due to insufficient numbers of male students, only females were included in data analyses.

4.3 - Procedure

Participants visited the Body Composition and Metabolism Lab in pairs, where they submitted to body composition analysis using a BOD POD™ Air Displacement Body Composition System (software version 4.5.1, Cosmed, Concord, CA). Participants were also weighed using the BOD POD scale and had their height measured using a wall-mounted stadiometer. The duration of the anthropometric testing was approximately 20 minutes per participant. Participants then completed several health and behavior related questionnaires, which took about 15 minutes. Lab visits were conducted by a graduate research assistant and were approximately one hour in duration.

4.4 - Measures

4.4.1 - CES-D

The Center for Epidemiologic Studies Depression Scale (CES-D) is a 20 item questionnaire that measures depressive symptomatology (Radloff, 1977). The CES-D focuses on current mood, and its items were selected based on the criteria necessary for a diagnosis of depression. Respondents are asked how often in the past week they felt a certain way, for example, “I had trouble keeping my mind on what I was doing”, and are given the choice of four
different responses; rarely or none of the time (less than one day), some or a little of the time (one to two days), occasionally or a moderate amount of time (three to four days), and most or all of the time (five to seven days). Total scores range from 0 to 60; any score of 16 or higher is considered be indicative of elevated depressive symptoms.

The CES-D has been demonstrated to have high internal consistency, with Cronbach’s alpha being equal to 0.85 and 0.90 in the general population and a depressed population respectively (Radloff, 1977). The Cronbach’s alpha of the CES-D for this sample was 0.89 (IBM SPSS Statistics (version 21.0; SPSS Inc., Chicago, IL)). For the purposes of this research, the full CES-D scale was not used. One of the items, “I did not feel like eating; my appetite was poor” may confound the diet variable, and was thus be excluded from analyses. This reduced the global CES-D score to a maximum of 57.

4.4.2 - Diet

Diet was assessed using three-day food records, which were subsequently analyzed using the ESHA Food Processor (The Food Processor for Windows, version 10.12.0; ESHA Research, Salem, OR). Participants were instructed to write down everything they consumed (i.e., foods and beverages) on three separate days - two weekdays and one weekend day. This enabled a more accurate representation of the participants’ usual diets (Thompson, Larkin, & Brown, 1986). These food records were later analyzed by an undergraduate research assistant using ESHA, which obtains its information from various databases, including the Canadian Nutrient File and the USDA Standard Reference database. Nutrient intakes assessed by ESHA include carbohydrates, protein, and omega-3 fatty acids among others.

The Canadian version of the Healthy Eating Index (HEI-C) was used to assess overall diet quality. Data obtained from ESHA were inputted into a spreadsheet created by Brauer and
Royall, which assigns an HEI-C score based on two 24-hour recalls and a questionnaire developed by Garriguet (2009) (Appendix). The spreadsheet required the number of servings consumed from each food group (Eating Well with Canada’s Food Guide, Health Canada, (2007)), total intake of unsaturated fatty acids, sodium intake, and total intake of calories. Scores range from 0 to 100, and are based on the Canada’s Food Guide’s recommendations for females 18 to 50, females 51 and older, males 18 to 50, and males 51 and older. A score of 0 to 50 is considered indicative of “poor diet quality”, between 50 and 80 is “needs improvement”, and 80 to 100 is considered “good diet quality”.

4.4.3 – Glycemic Index and Glycemic Load

To measure carbohydrate intake, we elected to calculate dietary glycemic index (GI) and glycemic load (GL) for each participant. Glycemic index, and in particular glycemic load, takes into account how specific carbohydrates impact blood glucose, thus providing a measure of dietary carbohydrate quality (Jenkins et al., 1981). The methodology for assigning GI and GL values to each individual food identified in the food records was adapted from those developed by Louie, Flood, Turner, Everingham, & Gwynn (2011) and Forbes and colleagues (2009). GI values were obtained from the “International table of glycemic index and glycemic load” (Atkinson, Foster-Powell, & Brand-Miller, 2008) and from www.glycemicindex.com, a website of values maintained by the University of Sydney. Only values obtained from studies using “normal” participants were used, thus values obtained from subjects with type 1 or type 2 diabetes were excluded. Evidence suggests that both type 1 and type 2 diabetes alter insulin response (Perley & Kipnis, 1967).

To assign values to each particular food, an adaptation of the algorithm described by Louie and colleagues (2011) was applied. If a food existed in one of the databases that was an
exact match to the food in question, that GI value was assigned. If no exact match existed, the GI value of a closely related food item (as determined by clinical judgment) was assigned instead. Foods containing less than five grams of available carbohydrate per 100 gram serving were assigned a GI value of zero. No values for condiments and sauces (i.e., ketchup) are available in the current databases, and thus the value for tomato soup was applied instead. To calculate dietary GI and GL, all foods, the serving sizes consumed, their GI value, and the amount of available carbohydrate in a serving of that food were entered into a spreadsheet (Appendix). GL of each food was calculated using the formula:

\[ \text{GL}_{\text{Food}} = \text{GI} \times \left( \frac{\text{available carbohydrate}}{\text{standard serving size}} \right) \times \text{serving size consumed} \]

Average daily GL was calculated using the formula:

\[ \text{Daily GL} = \frac{\sum \text{GL}_{\text{Food}}}{3} \]

Daily GI was calculated using the formula:

\[ \text{Daily GI} = \frac{\text{daily GL}}{\text{average daily available carbohydrate}} \times 100 \]

Energy intake was accounted for using the Willett method, which adjusted the GI and GL values using residuals obtained from a regression analysis of energy intake and GI/GL values (Willett & Stampfer, 1986). Both adjusted and non-adjusted values were included in analyses.

4.4.4 - Body Composition

Body composition was analyzed using the BOD POD, which utilizes air displacement plethysmography to calculate fat-free mass and fat mass. A total of four measurements (height, weight, body volume, and thoracic lung volume) are required in order to calculate body composition. As per the manufacturer’s recommendations, the BOD POD was re-calibrated before each use. Participants were instructed to wear only a bathing suit and bathing cap for the test, and no jewelry was required. Before entering the BOD POD chamber, height was assessed.
to the closest 0.1cm using a wall-mounted stadiometer (Medical Scales and Measuring Devices; Seca Corp., Ontario, CA, USA) and weight was measured to the nearest 0.1kg using the BOD POD scale.

After measurements of height and weight, participants were instructed to enter the BOD POD chamber, which measures participants’ body volume. For the most accurate results, this process requires participants to sit still, remain quiet, and breathe as they would normally. Finally, thoracic lung volume was measured while the participants remained seated in the chamber, while plugging their nose and breathing into tubing connected to the back of the BOD POD. Thoracic lung volume was used to calculate adjusted body volume, which in turn was used to obtain body density. Body density was then converted to fat-free mass and fat mass. All participants received a printed report of their results following the assessment.

4.4.5 - Physical Activity

Physical activity level was assessed using the International Physical Activity Questionnaire – Short Form (IPAQ-SF) (Craig et al., 2003). This measure was developed with the aim of measuring self-reported physical activity over the previous seven-day period, and includes activities such as moderate and vigorous physical activity, walking, and sedentary time. It was validated with data obtained from an activity-monitoring device (p=0.30, 95% CI 0.23-0.36), as well as the traditional long form questionnaire (Craig et al., 2003). Test-retest reliability has also been demonstrated, with the pooled p being equal to 0.76 (95% CI 0.73-0.77) (Craig et al., 2003).

4.5 - Data Analysis

Statistical analysis was completed using IBM SPSS Statistics (version 21.0; SPSS Inc., Chicago, IL). A p-value of ≤0.05 was considered statistically significant. Data obtained from the
CES-D were assessed for normality, and the distribution was determined to be not normal. Square root transformation therefore took place to normalize the data, and thus allow for the use of parametric statistical methods.

Data obtained from both the CES-D and the food records were rigorously reviewed for errors. Three participants did not complete all the CES-D items, and their data were thus removed from analyses. Data from food records were hand coded into EWCFG servings, which were averaged and inputted into an HEI-C calculations spreadsheet. HEI-C scores were calculated using a spreadsheet developed by Brauer and Royall (2013), and scores were treated as continuous variables.

Under- and over-reporters of dietary intake were calculated using the Goldberg method (Goldberg et al., 1991), as described by Garriguet (2008) in the 2004 Canadian Community Health Survey. Basal metabolic rate (BMR) was calculated for all participants using the Harris-Benedict equations, and a ratio of actual energy intake to BMR to actual energy intake was then computed. Garriguet (2008) calculated cut-offs of 70% and 142%, meaning that participants whose ratios were below 70% were considered under-reporters, and those whose ratios were above 142% were considered over-reporters. These cut-offs were used to exclude those who under- and over-reported intake in this sample. A total of 92 participants were identified as potential under-reporters. Clinical judgment was used to examine the identified food records; records were examined for care of completion, and participants’ weights and BMIs were also examined. Eighty records were deemed to be plausible and were added back into the sample.

Pearson correlation tests were computed to determine if energy intake, physical activity level, and percent body fat were associated with the dietary variables and depressive symptoms, although all were non-significant. Though some current research supports the inclusion of these
variables, we did not include them in our statistical models as they were not related to diet or depression in this sample (Kuczmarski et al., 2010; Liu et al., 2007; Mikolajczyk et al., 2009).

Independent t-tests were conducted to examine differences in dietary variables between participants with high vs. low levels of depressive symptoms. Participants with high levels of depressive symptoms were considered those whose CES-D scores were more than one standard deviation above the mean CES-D score, while those considered to have low levels of depressive symptoms were those whose CES-D score was one standard deviation below the mean score. This method of creating the two groups was chosen in order to ensure there was a sufficient discrepancy in average CES-D score, which could allow for the potential discovery of true differences in dietary variables.

Linear regressions were performed to assess associations between depressive symptoms and each of diet quality, servings of fruits and vegetables, percent energy from protein, omega-3 fatty acid intake, glycemic index/glycemic load, and percent energy from “other foods”. CES-D score was the dependent variable, while the dietary variables acted as the independent predictors. To adjust for age, age was entered as a control variable in all models. The final sample size of 141 subjects resulted in a lack of power for most analyses; only the model using diet quality as a predictor retained power above 0.7, as calculated by SAS (version 9.2).

5.0 - RESULTS

5.1 – Participants

A total of 175 subjects participated in the study, 156 females and 19 males. However, due to their comparatively small number, males were not included in subsequent analyses. Two participants did not complete 3-day food records, and another two did not adequately complete
the CES-D questionnaire. A further 11 subjects under-reported caloric intake, and were thus excluded from analyses, leading to a final sample of 141.

Table 5.1: Descriptive characteristics of study participants
n=141

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>19.1 ± 1.5</td>
<td>18 - 28</td>
</tr>
<tr>
<td><strong>BMI[^1]</strong></td>
<td>22.3 ± 3.4</td>
<td>16.6 - 38.1</td>
</tr>
<tr>
<td><strong>Body Fat (%)[^2]</strong></td>
<td>26.2 ± 6.9</td>
<td>9.4 - 47.9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ethnicity</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Caucasian</em></td>
<td>122</td>
<td>86.50%</td>
</tr>
<tr>
<td><em>Other</em></td>
<td>19</td>
<td>13.50%</td>
</tr>
<tr>
<td><strong>IPAQ Score[^3]</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Low Active</em></td>
<td>18</td>
<td>12.80%</td>
</tr>
<tr>
<td><em>Active</em></td>
<td>51</td>
<td>36.20%</td>
</tr>
<tr>
<td><em>High Active</em></td>
<td>72</td>
<td>51.10%</td>
</tr>
</tbody>
</table>

Note: [^1]Body Mass Index (Normal=18.5-24.9, Overweight>24.9)
[^2]Healthy percent body fat levels are estimated to be between 20-32% for females (American College of Sports Medicine).
[^3]The International Physical Activity Questionnaire (IPAQ) measures physical activity and is calculated by assessing vigorous and moderate activity levels, as well as walking over the past week (Craig et al., 2003).

5.2 – Research Objectives

The primary objective of this research was to examine the relation between overall diet quality and depressive symptoms in an emerging adult population. It was hypothesized that there would be an inverse relationship between depressive symptoms and diet quality, meaning that as diet quality increased, it was expected that CES-D scores would decrease.

The secondary objective of this research was to examine the relation between specific dietary components (% energy from protein; omega-3 fatty acids; number of servings of fruit and vegetables; % energy from “other foods”; glycemic index and glycemic load) and depressive
symptoms in an emerging adult population. It was hypothesized that each of % energy from protein, omega-3 fatty acids, and fruit and vegetable intake would be inversely associated with depressive symptoms, and each of dietary glycemic index, dietary glycemic load, and % of energy from “other foods” would be positively associated with depressive symptoms.

5.3 – Center for Epidemiologic Studies Depression Scale Scores

Results of the both the unadjusted and modified CES-D scale are reported in Table 5.2. The mean total score was 12.1±8.3, which is less than the cut-off of 16 said to indicative of elevated depressive symptoms. Item number two (I did not feel like eating; my appetite was poor) was thought to interact with the dietary variables examined in this study, and was thus removed from the global CES-D score prior to analyses. Therefore, the modified CES-D score, which had a mean of 11.6±8.1, was used in all subsequent statistical analyses.
Table 5.2: Center for Epidemiologic Studies Depression (CES-D) scale statements and mean scores

<table>
<thead>
<tr>
<th>Statement</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I was bothered by things that don't usually bother me.</td>
<td>0.57 ± 0.74</td>
</tr>
<tr>
<td>2. I did not feel like eating; my appetite was poor.</td>
<td>0.57 ± 0.72</td>
</tr>
<tr>
<td>3. I felt that I could not shake off the blues.</td>
<td>0.48 ± 0.78</td>
</tr>
<tr>
<td>4. I felt I was just as good as other people.</td>
<td>0.72 ± 0.89</td>
</tr>
<tr>
<td>5. I had trouble keeping my mind on what I was doing.</td>
<td>1.23 ± 0.90</td>
</tr>
<tr>
<td>6. I felt depressed.</td>
<td>0.38 ± 0.70</td>
</tr>
<tr>
<td>7. I felt that everything I did was an effort.</td>
<td>0.94 ± 0.89</td>
</tr>
<tr>
<td>8. I felt hopeful about the future.</td>
<td>0.9 ± 0.83</td>
</tr>
<tr>
<td>9. I thought my life had been a failure.</td>
<td>0.17 ± 0.46</td>
</tr>
<tr>
<td>10. I felt fearful.</td>
<td>0.43 ± 0.66</td>
</tr>
<tr>
<td>11. My sleep was restless.</td>
<td>1.17 ± 0.9</td>
</tr>
<tr>
<td>12. I was happy</td>
<td>0.53 ± 0.64</td>
</tr>
<tr>
<td>13. I talked less than usual.</td>
<td>0.53 ± 0.71</td>
</tr>
<tr>
<td>14. I felt lonely.</td>
<td>0.54 ± 0.83</td>
</tr>
<tr>
<td>15. People were unfriendly.</td>
<td>0.30 ± 0.56</td>
</tr>
<tr>
<td>16. I enjoyed life.</td>
<td>0.52 ± 0.71</td>
</tr>
<tr>
<td>17. I had crying spells.</td>
<td>0.38 ± 0.68</td>
</tr>
<tr>
<td>18. I felt sad.</td>
<td>0.73 ± 0.75</td>
</tr>
<tr>
<td>19. I felt that people dislike me.</td>
<td>0.27 ± 0.55</td>
</tr>
<tr>
<td>20. I could not get &quot;going&quot;.</td>
<td>0.66 ± 0.73</td>
</tr>
<tr>
<td>Total CES-D Score</td>
<td>12.1 ± 8.3</td>
</tr>
<tr>
<td>Total Modified CES-D Score(^1)</td>
<td>11.6 ± 8.1</td>
</tr>
</tbody>
</table>

Note: The CES-D has a minimum score of zero and a maximum score of 60, and scores >16 are considered to be indicative of elevated depressive symptoms. Each individual statement has a minimum score of zero and a maximum score of three. \(^1\) The total modified CES-D score is the summed total of all the CES-D statements, not including statement two (“I did not feel like eating, my appetite was poor”) (Eaton et al., 2004).

5.4 – The Canadian Healthy Eating Index

Results of the HEI-C are reported in Table 5.3. Overall mean diet quality was calculated to be 68.2±15.2, which is classified as “needs improvement”. Study participants diets were also not consistent with Canada’s Food Guide recommendations. Subjects consumed fewer vegetables, dark green and orange vegetables, grains, whole grains, and unsaturated fats than recommended for their age and sex, and consumed more saturated fats and sodium than indicated.
by the DRIs. Overall, only 23% of diets were classified as “good”, while 77% of diets were classified as “poor” or “needs improvement”. With regards to fruits and vegetables, total fruit and vegetable intake was correlated with both whole fruit and vegetable intake \((r=0.68, p<0.001)\) and dark green and orange fruit and vegetable intake \((r=0.60, p<0.001)\). Whole fruit and vegetable intake, which does not include fruit juices, was lower than total fruit and vegetable intake, averaging 2.8±2.2 servings per day.

Table 5.3: Mean ± standard deviation of individual components of the Healthy Eating Index (HEI-C) and total score in all participants

<table>
<thead>
<tr>
<th>Component</th>
<th>Max Score</th>
<th>Avg Score</th>
<th>Avg Svgs/Day</th>
<th>EWCFG Servings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Fruits and Vegetables</td>
<td>10</td>
<td>7.1 ± 2.5</td>
<td>5.3±2.1</td>
<td>7-8</td>
</tr>
<tr>
<td>Whole Fruits and Vegetables</td>
<td>5</td>
<td>4.0 ± 1.7</td>
<td>2.8±2.2</td>
<td>---</td>
</tr>
<tr>
<td>Dark Green and Orange Vegetables</td>
<td>5</td>
<td>3.1 ± 2.0</td>
<td>1.2±1.1</td>
<td>2</td>
</tr>
<tr>
<td>Total Grains</td>
<td>5</td>
<td>3.7 ± 1.1</td>
<td>4.8±1.9</td>
<td>6-7</td>
</tr>
<tr>
<td>Whole Grains</td>
<td>5</td>
<td>2.9 ± 1.9</td>
<td>2.0±1.6</td>
<td>3-4</td>
</tr>
<tr>
<td>Milk and Alternatives</td>
<td>10</td>
<td>7.6 ± 2.9</td>
<td>1.9±1.3</td>
<td>3-4</td>
</tr>
<tr>
<td>Meat and Alternatives</td>
<td>10</td>
<td>8.4 ± 2.3</td>
<td>2.1±1.0</td>
<td>2</td>
</tr>
<tr>
<td>Unsaturated Fats(^1)</td>
<td>10</td>
<td>7.6 ± 2.5</td>
<td>1.6±0.8</td>
<td>2</td>
</tr>
<tr>
<td>Saturated Fats (%kcal)</td>
<td>10</td>
<td>6.4 ± 3.4</td>
<td>10.5±3.1</td>
<td>&lt;7%</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>10</td>
<td>7.1± 2.6</td>
<td>2499.5±882.6</td>
<td>&lt;2300</td>
</tr>
<tr>
<td>% Energy from “Other Foods”(^2)</td>
<td>20</td>
<td>10.0 ± 6.6</td>
<td>22.2±13.7</td>
<td>---</td>
</tr>
<tr>
<td>Total HEI-C Score</td>
<td>100</td>
<td>68.2 ± 15.2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Healthy Eating Index - Canadian Adaptation (Garriguet, 2009) assesses diet quality on a scale of 0 to 100, with a score of 100 indicating high diet quality. HEI-C scores \(\leq 50\) are considered "poor", scores 50-80 are "needs improvement", and scores >80 are "good".

\(^1\)One serving of unsaturated fats is equal to 15g. \(^2\) "Other foods” were defined as any food that did not fit into one of the Canada's Food Guide food groups. Abbreviations: EWCFG, Eating Well with Canada’s Food Guide; DRIs, Dietary Reference Intakes.
5.5 – Associations Between Diet and Depressive Symptoms

Preliminary correlations tests detected a significant negative correlation between CES-D score and overall diet quality (r=−0.215, p=0.01). Small correlations were also present between CES-D score and each of omega-3 intake (r=0.157, p=0.062) and percent energy from “other foods” (r=0.144, p=0.09).

Results from a forced entry linear regression are presented in Tables 5.4 and 5.5. Overall diet quality was significantly inversely associated with CES-D score (β=−0.016, CI=(−0.029−(0.003)), p=0.017). The effect estimate remained relatively unchanged after adjustment for age (β=−0.017, CI=(−0.03−(0.004)), p=0.043). No significant association was found between depressive symptoms and fruit and vegetable intake, omega-3 fatty acid intake, percent energy from protein, percent energy from other foods, and glycemic index/glycemic load.

Figure 5.1: Scatterplot of the relationship between CES-D score and overall diet quality

(CESDMSQRT = squared-rooted modified CES-D score)
Table 5.4: Results of a linear regression examining the unadjusted association between modified CES-D score and dietary intake

<table>
<thead>
<tr>
<th>Model</th>
<th>Outcome</th>
<th>Predictor Variables</th>
<th>$\beta$</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Modified CES-D HEI-C</td>
<td></td>
<td>-0.016</td>
<td>-0.029(-0.003)</td>
<td>0.017*</td>
</tr>
<tr>
<td>B</td>
<td>Modified CES-D Fruit and vegetable intake</td>
<td></td>
<td>-0.076</td>
<td>-0.169-0.016</td>
<td>0.103</td>
</tr>
<tr>
<td>C</td>
<td>Modified CES-D Omega-3 intake</td>
<td></td>
<td>0.172</td>
<td>-0.046-0.39</td>
<td>0.121</td>
</tr>
<tr>
<td>D</td>
<td>Modified CES-D % energy from “other foods”</td>
<td></td>
<td>0.012</td>
<td>-0.002-0.027</td>
<td>0.09</td>
</tr>
<tr>
<td>E</td>
<td>Modified CES-D % energy from protein</td>
<td></td>
<td>-0.029</td>
<td>-0.087-0.028</td>
<td>0.313</td>
</tr>
<tr>
<td>F</td>
<td>Modified CES-D GI (raw)</td>
<td></td>
<td>0.024</td>
<td>-0.023-0.07</td>
<td>0.319</td>
</tr>
<tr>
<td></td>
<td>Modified CES-D GI (energy-adjusted)$^1$</td>
<td></td>
<td>-0.024</td>
<td>-0.071-0.023</td>
<td>0.32</td>
</tr>
<tr>
<td>G</td>
<td>Modified CES-D GL (raw)</td>
<td></td>
<td>-0.221</td>
<td>-1.682-1.239</td>
<td>0.765</td>
</tr>
<tr>
<td></td>
<td>Modified CES-D GL (energy-adjusted)$^1$</td>
<td></td>
<td>-0.353</td>
<td>-2.371-1.666</td>
<td>0.73</td>
</tr>
</tbody>
</table>

Note: 1 GI and GI values were adjusted for energy intake using the Willett method. Abbreviations: HEI-C, Canadian Healthy Eating Index; GI, Glycemic Index; GL, Glycemic Load. Analyses were also ran with the full sample including males. No differences in results were observed. *p<0.05

Table 5.5: Results of a linear regression examining the adjusted association between modified CES-D score and dietary intake

<table>
<thead>
<tr>
<th>Model</th>
<th>Outcome</th>
<th>Predictor</th>
<th>$\beta$</th>
<th>CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Modified CES-D HEI-C</td>
<td></td>
<td>-0.017</td>
<td>-0.03(-0.004)</td>
<td>0.043*</td>
</tr>
<tr>
<td>B</td>
<td>Modified CES-D Fruit and vegetable intake</td>
<td></td>
<td>-0.077</td>
<td>-0.169-0.016</td>
<td>0.252</td>
</tr>
<tr>
<td>C</td>
<td>Modified CES-D Omega-3 intake</td>
<td></td>
<td>0.173</td>
<td>-0.046-0.392</td>
<td>0.284</td>
</tr>
<tr>
<td>D</td>
<td>Modified CES-D % energy from “other foods”</td>
<td></td>
<td>0.013</td>
<td>-0.002-0.027</td>
<td>0.223</td>
</tr>
<tr>
<td>E</td>
<td>Modified CES-D % energy from protein</td>
<td></td>
<td>-0.031</td>
<td>-0.089-0.027</td>
<td>0.554</td>
</tr>
<tr>
<td>F</td>
<td>Modified CES-D GI (raw)</td>
<td></td>
<td>0.024</td>
<td>-0.023-0.07</td>
<td>0.583</td>
</tr>
<tr>
<td></td>
<td>Modified CES-D GI (energy-adjusted)$^1$</td>
<td></td>
<td>-0.024</td>
<td>-0.071-0.023</td>
<td>0.582</td>
</tr>
<tr>
<td>G</td>
<td>Modified CES-D GL (raw)</td>
<td></td>
<td>-0.272</td>
<td>-1.762-1.218</td>
<td>0.893</td>
</tr>
<tr>
<td></td>
<td>Modified CES-D GL (energy-adjusted)$^1$</td>
<td></td>
<td>-0.403</td>
<td>-2.446-1.641</td>
<td>0.883</td>
</tr>
</tbody>
</table>

Note: All models adjusted for age. $^1$ GI and GI values were adjusted for energy intake using the Willett method. Abbreviations: HEI-C, Canadian Healthy Eating Index; GI, Glycemic Index; GL, Glycemic Load. Analyses were also ran with the full sample including males. No differences in results were observed. *p<0.05
Differences in dietary variables between subjects with high levels and low levels of depressive symptoms were assessed using independent t-tests, and are reported in Table 5.6. Those whose CES-D scores were one standard deviation above the means were compared with those whose CES-D scores were one standard deviation below the mean. No significant differences were found between the two groups in any of the dietary variables, although it is likely that the lack of statistical power prevented the detection of any differences. However, there was a statistically significant difference in the intakes of whole fruits and vegetables (not including fruit juice), with those with lower levels of depressive symptoms consuming an average of 4.5±1.4 servings and those with higher levels of depressive symptoms consuming 3.2±2.4 servings (t=2.08, p=0.046). There may be several clinically significant differences between the diets of those with elevated depressive symptoms and those with very low levels of depressive symptoms. Those with low CES-D scores consumed 0.9 more servings of fruits and vegetables than those with high CES-D scores, and had a mean HEI-C score that was 7.5 points higher.
Table 5.6: Results of an independent t-test examining differences in dietary variables between participants one standard deviation above and one standard deviation below mean modified CES-D score

<table>
<thead>
<tr>
<th></th>
<th>One Std Dev Below Mean CES-D Score</th>
<th>One Std Dev Above Mean CES-D Score</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Average Kcal/day</td>
<td>1847.0 ± 409.5</td>
<td>1784.4 ± 476.6</td>
<td>0.677</td>
</tr>
<tr>
<td>Servings of Fruits and Vegetables</td>
<td>5.5 ± 2.6</td>
<td>4.6 ± 2.2</td>
<td>0.275</td>
</tr>
<tr>
<td>% Energy From “Other Foods”</td>
<td>18.9 ± 12.1</td>
<td>22.2 ± 13.0</td>
<td>0.437</td>
</tr>
<tr>
<td>% Energy From Protein</td>
<td>17.4 ± 3.8</td>
<td>17.1 ± 3.8</td>
<td>0.78</td>
</tr>
<tr>
<td>Omega-3 Intake</td>
<td>0.8 ± 0.5</td>
<td>1.3 ± 2.2</td>
<td>0.394</td>
</tr>
<tr>
<td>HEI-C</td>
<td>71.9 ± 11.4</td>
<td>64.4 ± 17.9</td>
<td>0.145</td>
</tr>
<tr>
<td>GI (raw)</td>
<td>52.2 ± 3.7</td>
<td>52.9 ± 4.7</td>
<td>0.655</td>
</tr>
<tr>
<td>GI (energy-adjusted)</td>
<td>51.9 ± 3.7</td>
<td>51.2 ± 4.7</td>
<td>0.62</td>
</tr>
<tr>
<td>GL (raw)</td>
<td>104.7 (74.1-134.9)</td>
<td>93.3 (63.1-138.0)</td>
<td>0.34^4</td>
</tr>
<tr>
<td>GL (energy-adjusted)</td>
<td>102.3 (83.2-120.2)</td>
<td>93.3 (69.2-125.9)</td>
<td>0.399^4</td>
</tr>
</tbody>
</table>

Note: 1Participants in the top 50th percentile of CES-D scores have greater levels of depressive symptoms than those in the lower 50th percentile. 2GI and GI values were adjusted for energy intake using the Willett method. 3Distribution was log-adjusted to obtained normality. 4P-value was obtained from non-transformed data. Abbreviations: HEI-C, Canadian Healthy Eating Index; GI, Glycemic Index; GL, Glycemic Load.

Results from a logistic regression are reported in Tables 5.7 and 5.8. Participants were divided into two groups: those with CES-D scores that were one standard deviation above the mean (high depressive symptoms), and those with CES-D scores that were one standard deviation below the mean (low depressive symptoms). None of the resulting models reached statistical significance.
Table 5.7: Results of an unadjusted logistic regression examining differences in dietary variables between modified CES-D score one standard deviation above and one standard deviation below the mean

<table>
<thead>
<tr>
<th>Model</th>
<th>Outcome</th>
<th>Predictor</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Modified CES-D</td>
<td>HEI-C</td>
<td>0.966</td>
<td>0.922-1.012</td>
<td>0.141</td>
</tr>
<tr>
<td>B</td>
<td>Modified CES-D</td>
<td>Fruit and vegetable intake</td>
<td>0.85</td>
<td>0.637-1.134</td>
<td>0.27</td>
</tr>
<tr>
<td>C</td>
<td>Modified CES-D</td>
<td>Omega-3 intake</td>
<td>0.271</td>
<td>0.631-2.725</td>
<td>0.467</td>
</tr>
<tr>
<td>D</td>
<td>Modified CES-D</td>
<td>% energy from “other foods”</td>
<td>1.022</td>
<td>0.968-1.079</td>
<td>0.426</td>
</tr>
<tr>
<td>E</td>
<td>Modified CES-D</td>
<td>% energy from protein</td>
<td>0.974</td>
<td>0.817-1.162</td>
<td>0.772</td>
</tr>
<tr>
<td>F</td>
<td>Modified CES-D</td>
<td>GI (raw)</td>
<td>1.038</td>
<td>0.886-1.216</td>
<td>0.645</td>
</tr>
<tr>
<td>G</td>
<td>Modified CES-D</td>
<td>GI (energy-adjusted)</td>
<td>0.959</td>
<td>0.817-1.126</td>
<td>0.609</td>
</tr>
</tbody>
</table>

Note: 1 Modified CES-D score was dichotomized into scores one standard deviation above the mean, and scores one standard deviation below the mean. 2 GI and GI values were adjusted for energy intake using the Willett method. Abbreviations: HEI-C, Canadian Healthy Eating Index; GI, Glycemic Index; GL, Glycemic Load.

Table 5.8: Results of an adjusted logistic regression examining differences in dietary variables between modified CES-D score one standard deviation above and one standard deviation below the mean

<table>
<thead>
<tr>
<th>Model</th>
<th>Outcome</th>
<th>Predictor</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Modified CES-D</td>
<td>HEI-C</td>
<td>0.965</td>
<td>0.921-1.012</td>
<td>0.143</td>
</tr>
<tr>
<td>B</td>
<td>Modified CES-D</td>
<td>Fruit and vegetable intake</td>
<td>0.85</td>
<td>0.637-1.134</td>
<td>0.269</td>
</tr>
<tr>
<td>C</td>
<td>Modified CES-D</td>
<td>Omega-3 intake</td>
<td>1.312</td>
<td>0.634-2.713</td>
<td>0.465</td>
</tr>
<tr>
<td>D</td>
<td>Modified CES-D</td>
<td>% energy from “other foods”</td>
<td>1.022</td>
<td>0.968-1.079</td>
<td>0.426</td>
</tr>
<tr>
<td>E</td>
<td>Modified CES-D</td>
<td>% energy from protein</td>
<td>0.974</td>
<td>0.817-1.162</td>
<td>0.772</td>
</tr>
<tr>
<td>F</td>
<td>Modified CES-D</td>
<td>GI (raw)</td>
<td>0.086</td>
<td>0.886-1.216</td>
<td>0.644</td>
</tr>
<tr>
<td>G</td>
<td>Modified CES-D</td>
<td>GI (energy-adjusted)</td>
<td>0.959</td>
<td>0.817-1.126</td>
<td>0.608</td>
</tr>
</tbody>
</table>

Note: All models adjusted for age. 1 Modified CES-D score was dichotomized into scores one standard deviation above the mean, and scores one standard deviation below the mean. 2 GI and GI values were adjusted for energy intake using the Willett method. Abbreviations: HEI-C, Canadian Healthy Eating Index; GI, Glycemic Index; GL, Glycemic Load.*p<0.05
Over the past few years, it has become increasingly evident that diet is associated with mental health (Quirk et al., 2013). Overall diet quality, as well as specific dietary components, have been examined in the context of their association with both depressive symptoms (i.e., risk of depression) and clinician-diagnosed depression (Akbaraly et al., 2009; Christenson & Somers, 1995; Mikolajczyk et al., 2009; Murakami et al., 2012; Park et al., 2009; Yary & Aazami, 2012). Several hypotheses postulate how poor quality diet may augment depressive symptoms (Beydoun et al., 2013; Bottiglieri et al., 2000; Logan, 2013; Wurtman et al., 2003; Wurtman & Wurtman, 1979), although it may be that depression itself can lead to one consuming a poor quality diet. Research to date has shown that the connection between diet and depression is especially prevalent in females. The main objective of this study was to examine the relation between overall diet quality and depressive symptoms in a female emerging adult population. The secondary objective was to examine the relation between consumption of specific dietary components (% energy from protein; omega-3 fatty acids; fruit and vegetables; % energy from “other foods”; glycemic index and glycemic load) and depressive symptoms in this same population. Overall, our results revealed that diet quality was significantly inversely associated with depressive symptoms as measured by the CES-D. None of the dietary components identified in the secondary objective significantly predicted CES-D score, although fruit and vegetable intake and percent energy from “other foods” trended towards significance, with fruit and vegetable intake inversely associated with depressive symptoms, and percent energy from “other foods” positively associated with depressive symptoms.
6.1 – Depressive Symptoms and Overall Diet Quality

This was the first study to our knowledge to have examined the association between diet quality and depressive symptoms in an emerging adult population. Consistent with our hypothesis, we found that diet quality was significantly inversely associated with depressive symptoms: as higher diet quality scores corresponded with lower levels of depressive symptoms ($\beta=-0.017$, $p=0.043$). While results of an independent t-test comparing diet quality scores amongst those with high and low levels of depressive symptoms were non-significant, participants with low levels of depressive symptoms had a mean HEI-C score of 71.9±11.4, while participants with high levels of depressive symptoms had a mean score of 64.4±17.9. Thus, though non-significant, these results were as anticipated.

Several longitudinal studies have been completed in an attempt to elucidate the temporal order of this association. For instance, in middle-aged adults, Akbaraly and colleagues (2009) found that CES-D score was significantly predicted by dietary pattern as measured five years prior, and they thus concluded that poor diet was a risk factor for elevated depressive symptoms. LePort and colleagues (2012) reported similar results over a 10-year period, finding that consuming a healthy dietary pattern at baseline was significantly inversely associated with CES-D score amongst adult females. Similarly, a prospective study of Australian adolescents found that healthy diet was significantly and inversely associated with depression score at both baseline and follow-up two years later, although depression score at baseline did not predict diet quality at the completion of the study (Jacka et al., 2011).

Taken together, these results indicate that poor quality may lead to increased depressive symptoms, rather than increased depressive symptoms leading to poor diet quality. Dietary patterns classified as being of poor quality are typically low in fruits, vegetables, lean meats and
fish, and whole grains, and tend to be high in snack, fast, and convenience foods not included in Canada’s Food Guide (Garriguet, 2009). Poor quality diets thus consist of larger amounts of simple carbohydrates and saturated fats, and smaller amounts of omega-3 fatty acids, fibre, and essential micronutrients. Diets low in omega-3 fatty acids have been linked to activation of inflammatory pathways in the body and decreased brain cell health, which may play a role in the pathophysiology of depression (Logan, 2003; Wurtman & Wurtman, 1990), while dietary antioxidants such as vitamins A, C, and E have been postulated to decrease inflammation and prevent oxidative stress, which again, may be associated with the development of depressive disorder (Beydoun et al., 2013). It is through these mechanisms that poor diet quality may lead to the onset of depressive symptoms.

In the lone randomized controlled trial examining the impact of overall diet quality on depression, a Mediterranean diet supplemented with nuts significantly reduced risk of depression, although this relationship was only present in subjects with type 2 diabetes and not otherwise healthy subjects (Sanchez-Villegas et al., 2013). A traditional Mediterranean-style diet did not have a significant impact on depression risk (Sanchez-Villegas et al., 2013). While the investigators of this study concluded that consuming a Mediterranean diet could help alleviate symptoms of depression, these results were only found in individuals with type 2 diabetes, not otherwise healthy subjects. Given that these results have been found by only one study, more randomized controlled trials are therefore needed to further determine whether a healthy diet can act as an adjunctive treatment for depression.

Appetitive change is a common symptom of depression, which frequently presents as a reduction in appetite (American Psychiatric Association, 2014; Paykel, 1977). A reduction in appetite may be further augmented by a lack of interest in food and absence of the energy
required to buy groceries and prepare meals. There is also limited evidence to suggest that a subset of people with depression may crave high-carbohydrate and high-sugar foods, the consumption of which would result in a reduction in overall diet quality (Wurtman & Wurtman, 1995). This hypothesized craving may be due to the potential effect of simple carbohydrates on serotonin formation in the brain. Carbohydrates are hypothesized to increase the availability of the amino acid tryptophan (a serotonin precursor) to the brain by reducing its competition for receptors required to cross the blood-brain barrier, which could result in a reduction of depressive symptoms (Wurtman & Wurtman, 1979).

Results of this study were consistent with other research conducted in university student populations (Liu et al., 2007; Mikolajczyk et al., 2009). While there are no previous studies examining overall diet quality and depressive symptoms in an emerging adult population, two have assessed diet in the context of individual food groups. Mikolajczyk and colleagues (2009) reported cross-sectional inverse associations between intake of fruits, vegetables, meat, and fish and depressive symptoms in female university students. Similarly, Liu and colleagues (2007) found a cross-sectional association between the lowest intakes of ready-to-eat and fast foods and lower levels of depressive symptoms, and an inverse association between higher fruit intake and depressive symptoms in female university students. High intakes of fruits, vegetables, meat, and fish would be associated with higher diet quality, while ready-to-eat and fast foods would be associated with lower diet quality.

As previously stated, this is one of the first studies to examine diet quality using a score or index, and it is therefore somewhat difficult to contrast the results of this study with those in the current literature. This study used three-day food records as a means of dietary data collection, which are more accurate than the food frequency questionnaires commonly used in
the literature (Crawford et al., 1994). Finally, study participants were emerging adult female university students, who are at a different stage of life than participants in other studies, who tend to be middle-aged adults.

It is important to note that participants in this study had a higher average diet quality score (68.2) than subjects of the same age and gender in the Canadian validation study (56.9) (Garriguet, 2009). This may be due to the fact that participants in this study were highly active university students (87.3% of participants were considered highly active or active) and were enrolled in a nutrition course. Enrolment in a nutrition course may indicate an interest in diet, which may translate to more healthful food choices. Enrolment in university is also often indicative of higher socioeconomic status, which along with education itself has been demonstrated to be strongly associated with better diet quality (Raffensperger et al., 2010). This highlights the fact that even though participants in this study were consuming a more healthful diet than the average emerging adult, differences in diet quality still existed between those with higher and lower levels of depressive symptoms. It is therefore probable that differences in HEI-C scores between those with higher and lower levels of depressive symptoms may be even greater amongst emerging adult females who are not university students, and potentially even amongst university students who are not enrolled in a nutrition course.

### 6.2 – Depressive Symptoms and Fruit and Vegetable Intake

Consistent with our hypothesis, results of a linear regression indicated a negative association between fruit and vegetable intake and depressive symptoms, although this was non-significant. A t-test comparing differences in fruit and vegetable intake between participants with CES-D score one standard deviation above vs. one standard deviation below the mean score yielded similar results. Those whose CES-D scores were one standard deviation above the mean,
that is, with higher depressive symptomology, consumed an average of 4.6±2.2 servings per day, while those whose scores were one standard deviation below the mean, that is, with lower depressive symptomology, consumed 5.5±2.6 servings. While not statistically significant, this 20% difference may be clinically significant, or useful in a prevention or treatment setting. It is possible that higher consumption of fruits and vegetables, and thus many essential micronutrients, may decrease risk of depression. Micronutrients present in fruits and vegetables such as vitamins A, C, and E are thought to prevent many of the biological mechanisms that may cause depression (Beydoun et al., 2013; Park et al., 2009). For example, some micronutrients act as antioxidants, which reduce inflammation in the body. Chronic inflammation causes oxidative stress, which is damaging to neuronal health and may inhibit neurotransmitter formation, and may lead to depression (Beydoun et al., 2013).

In this study, average fruit and vegetable intake was 5.3±2.1 servings per day, which did not meet the EWCFG guidelines of seven to eight servings daily (Eating Well with Canada’s Food Guide, 2007). Low fruit and vegetable intake is common in young adults and university students, who may lack the time or resources to prepare fruits and vegetables on their own (Kwan, Faulkner, Arbour-Nicitopoulos, & Cairney, 2013). Common barriers to consuming fruits and vegetables cited by young adult university students include lack of preparation skills, dislike of taste, the belief that fresh produces spoils quickly, eating meals with friends who do not eat fruits and vegetables, and cost (Graham, Pelletier, Neumark-Sztainer, Lust, & Laska, 2013).

Whole fruit and vegetable intake, which does not include fruit juices, was even lower than total fruit and vegetable intake, averaging only 2.8±2.2 servings per day. Total fruit and vegetable intake was highly correlated with whole fruit and vegetable intake (r=0.68, p<0.001) and moderately correlated with dark green and orange fruit and vegetable intake (r=0.60,
p<0.001), implying that high total fruit and vegetable intake does not necessarily equate to high intake of nutrient-rich whole fruits and vegetables. There was also a statistically significant difference in the intakes of whole fruits and vegetables, with those with lower levels of depressive symptoms consuming an average of 4.5±1.4 servings and those with higher levels of depressive symptoms consuming 3.2±2.4 servings (t=2.08, p=0.046). Since most fruits and vegetables consumed by participants in this study came from juice, participants may not have been obtaining the full nutritional benefits of whole fruits and vegetables. Whole fruits and vegetables are high in fibre and numerous micronutrients, while juice lacks these benefits and is a concentrated source of sugar. For this reason, juice does not have the same beneficial effect on health as whole fruits and vegetables. This may also at least partially explain why Oddy and colleagues (2009) and Liu and colleagues (2007) found associations only between whole fruit/leafy greens intake and depressive symptoms, and not global fruit and vegetable intake. Both of these studies were cross-sectional in design, but Oddy and colleagues (2009) examined adolescents, while Liu and colleagues (2007) examined university students.

The findings of this study were consistent with those found in the current literature. Low fruit and vegetable intake has been found to be significantly associated with increased depressive symptoms in a cross-sectional study of female European university students (Mikolajczyk et al., 2009). A 2013 cross-sectional study conducted by Meyer and colleagues found similar, although non-significant results, in that females with depression had 12% lower fruit and vegetables intakes than their healthy counterparts. This association was not present in males. Low fruit intake has also been associated with increased depressive symptoms in a cross-sectional study of Chinese university students (Liu et al., 2007), while another cross-sectional study concluded that low intake of fresh fruit and leafy greens were associated with increased depression symptoms in
adolescents (Oddy et al., 2009). Taken together, it is likely that there is an inverse association between fruit and vegetable intake and depressive symptoms. Given the cross-sectional nature of studies completed to this date, the temporal order of this association cannot be determined.

6.3 – Depressive Symptoms and Omega-3 Fatty Acid Intake

Contrary to our original hypothesis, the results of a linear regression revealed no significant inverse association between omega-3 fatty acid intake and depressive symptoms. In fact, results were in the opposite direction of what we expected: there was a trend towards a positive association between omega-3 FA intake and depression risk. Similarly, an independent t-test also found no significant differences between the omega-3 FA intakes of participants with high vs. low depressive symptomology, although again, these results indicated a trend that suggested higher levels of depressive symptoms were positively associated with omega-3 FA intake.

There are several reasons that could explain why our results were not consistent with our hypothesis. First, omega-3 FA intake was in general so low that differences in intake between those with higher and lower depressive symptomology may be negligible. Participants in this study had an average omega-3 FA intake of 0.82 grams per day, which is considerably lower than the recommendations of 1.1 grams of alpha-linolenic acid and 0.3 to 0.45 grams combined of eicosapentaenoic acid and docosahexaenoic acid set by Dietitians of Canada (Dietitians of Canada, 2012). The low omega-3 intake observed in this study is consistent with studies conducted in the general population, with data collected form NHANES suggesting that Americans consume only 0.023 grams of EPA and 0.063 grams of DHA from food per day (Papanikolaou, Brooks, Reider, & Fulgoni, 2014). Since intake is so low across all participants, it
may be impossible to detect any associations between inadequate omega-3 FA intake and its potential consequences.

Secondly, this study measured risk of depression using the CES-D, and not actual clinician-diagnosed depression. Therefore, it is possible that some of the participants who had elevated depressive symptoms are not actually depressed. Attempting to associate omega-3 FA intake with depressive symptoms and not clinician-diagnosed depression may undervalue the potential protective effect of omega-3 FAs.

Lastly, it is possible that other studies found an association between depression and omega-3 FA intake because high omega-3 FA intake is strongly associated with other factors such as socio-economic status (Appleton et al., 2007). In this case, omega-3 FA intake would act as a proxy for these other lifestyle factors in their association with depression. It is possible that other behaviors may account for some of the association between omega-3 FAs and depression, thus reducing the strength of this relationship. For instance, in a cross-sectional study of middle-aged males, the association between fish consumption and depressed mood was attenuated after controlling for socioeconomic status and other lifestyle factors (although it still remained significant) (Appleton et al., 2007). Li and colleagues (2011) found that both males and females who lived below the poverty line were less likely to consume fish. A cross-sectional inverse association between fish consumption and depressive symptoms was found in males, but after controlling for socioeconomic status, this association was not found in females (Li et al., 2011).

It is difficult to contrast the results of this study with others available in the literature, due to the wide variety of methods of measuring omega-3 FA intake. Many studies examine fish intake as a proxy for omega-3 FAs, while others, including ours, measure grams of omega-3 FAs consumed per day. Some studies have taken the next step of examining the different types of
omega-3 FAs; (EPA, DHA, and ALA), all of which have different functions in the body, and thus different potential mechanisms of preventing depression. While the majority of current studies have found a potential relation between omega-3 FAs and depression/depressive symptoms, results of several other studies are congruent with ours. For instance, Sanchez-Villegas and colleagues (2007) conducted a prospective study in which participants in the highest tertile of fish consumption were significantly more likely to have received a diagnosis of depression in the two years after the study was initiated.

Two other studies also found no association between omega-3 FA intake and depressive symptoms. Giltay and colleagues (2011) conducted a randomized-controlled trial examining the impact of omega-3 supplementation on risk of depression in patients who had recently suffered a myocardial infarction, and found no decrease in depression risk after the treatment period. Jacka and colleagues (2004) implemented a six-year prospective study of depression risk in healthy individuals, and found no association between risk depression and omega-3 FA intake at any of the follow-up periods over the six-year length of the study. Thus, it is clear that due to the contradicting results seen in this body of literature, more research should be undertaken to further elucidate the potential relation between the various omega-3 fatty acids and depressive symptoms. For example, this field of research would benefit from studies examining the impact of individual omega-3 fatty acids (EPA, DHA, and ALA), as all three have differing functions in the body and on the brain.

6.4 – Depressive Symptoms and Percent Energy from Protein

Contrary to our hypothesis, percent energy from protein was not significantly inversely associated with depressive symptoms. An independent t-test also found no differences in percent energy from protein between those at higher risk and those at lower risk of depression.
Participants in this study consumed on average 17.2±3.5 percent of their daily energy from protein, consistent with the recommended daily allowance of 10-35% (Health Canada, 2006). Mean daily servings of meat and alternatives was 2.1±1.0, which is adequate for the participants’ age and sex according to EWCFG (Eating Well with Canada’s Food Guide, 2007). This would indicate that study participants were likely consuming sufficient amounts of protein.

There are several possible explanations for why our results were not consistent with our hypothesis. In the study, we measured percent energy from protein rather than using meat and poultry intake as a proxy. All studies that have found a significant association between protein intake and depression/depressive symptoms have used meat and poultry intake as a proxy for protein (Meyer at al., 2013; Mikolajczyk et al., 2009; Wolfe et al., 2011). In our study, we accounted for total protein intake, which includes not only the intake of complete protein-rich meat and poultry, but also protein obtained from incomplete plant sources such as beans, legumes, and rice. These plant-based proteins do not contain all the necessary essential amino acids, and are particularly low in tryptophan, the amino acid precursor to serotonin (Wurtman & Wurtman, 1979). Since serotonin, and thus tryptophan, are required for mood regulation, a diet low in tryptophan may be associated with decreased serotonin production and decreased mood (Wurtman & Wurtman, 1979). In fact, a Chinese study showed a significant direct association between high legume consumption and depression risk in premenopausal females (Li, Dai, Tedders, Arroya, & Zhang, 2010).

An inverse association between protein (or meat and poultry products) and depressive symptoms has primarily been noted in males and not females (Meyer et al., 2013; Wolfe et al., 2011), although one study did show an inverse association between meat/poultry intake and depressive symptoms in only females (Mikolajczyk et al., 2009). This may be because males
tend to consume more meat than females, and therefore get more of their protein from complete protein sources (Statistics Canada, 2007), which would suggest a greater intake of the amino acid tryptophan.

Though the study most similar to ours, conducted by Mikolajczyk and colleagues (2009), found a significant inverse cross-sectional association between meat/poultry intake and depression risk in female university students, two other studies found no relation between meat/poultry intake and depression in females (Meyer et al., 2013; Wolfe et al., 2011). Studies that found a negative effect of meat intake primarily examined the effects of red meat or processed meat intake (Buys & Sun, 2013; Oddy et al., 2009). While red meat is high in tryptophan, it is also high in saturated fat, which may be positively associated with depression itself (Sanchez-Villegas et al., 2011). However, it is more likely that high red meat consumption is indicative of other unhealthy dietary or lifestyle habits, which may be associated with an increased risk for depression (Williamson, Foster, Stanner, & Buttriss, 2005).

Since this current study showed no relation between percent energy from protein and depressive symptoms, it may be relevant in the future to examine protein in the context of itself different sources. Since different sources of protein contain varying amounts of the specific amino acids, they would have differing impacts on the mechanisms responsible for mood regulation in the body.

6.5 – Depressive Symptoms and Percent Energy from “Other Foods”

This study was the first to our knowledge to examine intake of so-called “junk foods” in the context of percent energy from “other foods”. “Other foods” are foods not included in EWCFG, and tend to be high in fat and sugar, and low in vitamins and minerals (Eating Well with Canada’s Food Guide, 2007). We hypothesised we would find a significant association
between percent energy from “other foods” and depressive symptoms in a female emerging adult population. The results of our linear regression were consistent with our hypothesis, although not significantly so. Similarly, the results of an independent t-test showed a non-significant difference between those at higher risk and those at lower risk of depression. Participants at the highest risk of depression consumed 22.2±13.0 percent of their daily energy from “other foods”, while participants at the lowest risk of depression consumed only 18.9±12.1 percent. Both the linear regression and independent t-test were underpowered, meaning the sample size may have been too small to detect statistical differences.

Although participants in the study had considerably higher diet quality than average Canadians, their intake of “other foods” was similar to values obtained in the HEI-C validation study. Average diet quality in this study was 68.2, while mean diet quality in the validation study was 58.8 (Garriguet, 2009). Participants in this study received an average “other foods” score of 10.0 points out of 20, and the validation study average was 10.7 (Garriguet, 2009). This suggests that although our study participants are maintaining a higher quality diet than most Canadians, they are still consuming similarly large amounts of confectionary and fast foods.

Convenience and processed food intake is common amongst university students, who are busy and may lack the time necessary to prepare nutritious meals (Larson et al., 2006). Many students in this study were also living in residence and therefore consuming many of their meals at the hospitality services locations on campus. While these facilities do provide increased access to prepared fresh fruits and vegetables, they primarily sell processed, fast, and convenience foods. There is also a strong connection between stress, depression, and “other foods” intake in university students. Prevalence of elevated psychological stress may be increased in the university student population, with one study finding that 19% of students had very high levels
of psychological distress, compared to only 2% of the general population (Stallman, 2010). Elevated levels of psychological stress have also been shown to be strongly associated with depression in students (Sherina, Rampal, & Kaneson, 2004).

Two cross-sectional studies have examined the connection between depression, stress, and diet in a university setting, with both finding that snack and fast food intake were more strongly associated with stress than depressive symptoms (Liu et al., 2007; Mikolajczyk et al., 2009). Mikolajczyk and colleagues (2009) hypothesized that the higher intakes of “other foods” seen amongst stressed study participants coupled with the lack of an association between “other foods” and depressive symptoms may suggest that consumption of “other foods” may alleviate the symptoms of depression. Limited research has shown a potential for carbohydrate/sugar cravings amongst females with depression, although more research is needed (Wurtman and Wurtman, 1995). Wurtman posited that participants with high levels of stress, but not high depressive symptoms, simply consume high amounts of “other foods”, rather than having psychological cravings.

Previous studies support our findings of a potential association between depressive symptoms and percent energy from “other foods”. For instance, a cross-sectional study of early adolescents found a positive association between the highest quartiles of fast/convenience foods and confectionary foods and depressive symptoms (Oddy et al., 2009). Cross-sectional studies of middle-aged females have also found positive associations between depressive symptoms and consumption of sweet foods (Jeffrey et al., 2009) and fast foods (Crawford et al., 2011), while Sanchez-Villegas and colleagues (2011) found that fast-food intake at baseline was associated with depressive symptoms 6.2 years later, but commercial baked good intake was not. Liu and colleagues (2007) uncovered a cross-sectional association between fast foods and ready-to-eat
foods and depressive symptoms in male and female university students. This was the only study to quantify stress, however, it was not controlled for in the logistic regression model (Liu et al., 2007). To strengthen future research, the effects of stress on “other foods” consumption should be considered. Stress and depression are inextricably linked, and thus stress should be accounted in future research examining “other food” intake. Doing so would ensure any relationship discovered between diet and depression is truly the result of depression, and not a result of stress functioning as a hidden variable.

6.6 – Depressive Symptoms and Glycemic Index/Glycemic Load

Related to the above findings about “other foods,” we felt it important to do a separate analysis to explore the association between glycemic index and load and risk of depression. The results of a linear regression and an independent t-test examining the associations between glycemic index/glycemic load and depression risk were not congruent with our hypotheses. It was hypothesized that both glycemic index and glycemic load would be significantly positively associated with depressive symptoms; however, our results were not consistent with this theory.

Glycemic index and glycemic load were used as proxies for carbohydrate intake in this study. Glycemic index is an indicator of how a particular food (or in this case, diet) impacts blood glucose, while glycemic load is an estimation of how much a certain amount of a food (or diet) will raise blood glucose (Jenkins et al., 1981). Glycemic load is therefore a measure of both the quality and quantity of carbohydrates consumed in the diet. It could be argued that glycemic load may be the best method for measuring carbohydrate intake, as it takes into account exactly how much a serving of particular food raises blood glucose. It is through this increase in blood glucose that carbohydrates are hypothesized to increase tryptophan availability, and thus serotonin formation (Wurtman & Wurtman, 1979).
There are two reasons for which we found no apparent association between glycemic index/load and risk of depression. It is likely that glycemic index lacked sufficient variation for differences between those at higher and lower risk of depression to be apparent (GI SD=4.3). Similar to “other foods”, university students in general tend to consume high carbohydrate (and high GI) diets, and eat large amounts of foods such as snack foods, confectionary foods, and sugar-sweetened beverages (Larson et al., 2006; United States Department of Health and Human Services, 2000). The relation between stress and snack food intake may also be confounding these results. University students are a stressed population, and stress has been shown to be associated with intake of high-GI foods such as intake of snack and confectionary foods (Liu et al., 2007; Mikolajczyk et al., 2009). It seems probable that many of these “stressed” participants are also consuming large amounts of high-GI foods, thus preventing this study from finding an association between GI/GL and depressive symptoms.

Our study is unique in that it is the first study of depression risk to measure carbohydrate intake by calculating glycemic index and glycemic load. Results are therefore difficult to contrast with those in the current literature, as an entirely different method of measuring carbohydrate intake was used. Only two studies have examined measured carbohydrate intake and depressive symptoms in otherwise healthy participants. Leibenluft and colleagues (1993) and Christensen and Somers (1995) both found significant cross-sectional associations between depression and carbohydrate intake. These studies were both conducted in adults who had received a diagnosis of clinical depression, a study population that differs considerably from our female university students.
6.7 – Strengths and Limitations

To our knowledge, this study is the fourth (Liu et al., 2007; Mikolajczyk et al., 2009; Park et al., 2009) to have examined the association between various aspects of diet/diet quality and depressive symptoms in an emerging adult population. Emerging adults are at increased risk of developing depression (Kessler et al., 2005; Public Health Agency of Canada, 2011). In particular for those attending university, there is a unique set of stressors that can lead to the development of depressive disorder (Kubawara et al., 2007). Since half of all depressive disorders develop during or before this developmental period, emerging adulthood may be a key time for detection, intervention, and treatment (Kessler et al., 2005). Furthermore, emerging adulthood may be a key period for successful lifestyle intervention. Many lifelong habits are formed during this time, and research suggests many of lifestyle practices and behaviours are malleable to change (Kwan et al., 2013).

Furthermore, this study is also one of the first in the diet and depressive symptom literature to have measured diet quality using a calculated score, in this case, the Healthy Eating Index – Canadian Adaption (HEI-C). The use of an index such as the HEI-C may provide a more accurate representation of overall diet quality as it takes into account several aspects of the diet, including fruit/vegetable intake, intake of whole grains, and percentage of calories consumed from foods not included in Canada’s Food Guide (Garriguet, 2009). Given that the human diet involves simultaneous exposure to a wide variety of macro-/micronutrients and dietary components, it is challenging to relate intake of individual nutrients to a specific disease or its symptoms. Furthermore, the use of a tool such as the HEI-C allows for the detection of the combined effects of nutrients consumed in the diet. For this reason, it may be more relevant to examine diet in the context of overall quality rather than by individual components.
This study used three-day food records for dietary assessment, considered to be the reference standard in this field (Crawford et al., 1994). Most studies in the current literature used food frequency questionnaires, which are less accurate at estimating usual food intake in part because they are unable to measure servings sizes of foods. The use of three-day food records also allowed for the use of the HEI-C, which requires data obtained from food records rather than food frequency questionnaires.

Under-reporting of dietary data is impossible to avoid when conducting nutrition research, but was accounted for by this study during data analysis. Under-reporting of intake promotes bias and may conceal results, especially when intake is being examined with regards to other lifestyle behaviours (Black, 2000; Garriguet, 2008). To account for both under- and over-reporters of dietary intake, reported intake was compared to each participant’s corresponding calculated basal metabolic rate (BMR). Under- and over-reporters were then determined by the Goldberg method, which employs a cut-off based on the ratio of BMR to actual energy intake (Black, 2000).

While there were many strengths to this study, it was not without some limitations. The sample of 141 emerging adult females was relatively small, and resulted in some of our tests being under-powered. The original sample of 175 participants was reduced due to insufficient numbers of males enrolled in the study, inadequate completion of the CES-D questionnaire, and under-reporting of dietary intake. The reduced sample size and lack of male participants means that this study’s results cannot be generalized outside of a population of female, emerging adult university students.

This study utilized a cross-sectional design, meaning that measures were taken at only one point in time. While a cross-sectional design can infer associations, it cannot infer causation
or directionality. To better understand whether a cause-and-effect relationship exists between diet and depressive symptoms, prospective observational studies will need to be conducted, preferably in a large group of currently healthy individuals. This would allow the development of depression as it relates to diet to be tracked over time. Randomized controlled trials similar to the one conducted by Sanchez-Villegas and colleagues (2013) could also be performed in order to better understand whether a nutritious diet can relieve symptoms of clinician-diagnosed depression.

Participants in this study were emerging adult females who were enrolled in an undergraduate nutrition course, which indicates an interest in diet and nutrition. Increased nutrition knowledge has been demonstrated to be related to both maintenance of a healthy diet and greater weight control (Kornith, Schiess, & Westenhoefer, 2009). This was exemplified by our study sample, for which the rate of overweight and obesity was 15.6%, which is lower than the Canadian average of 39% for young adult females (Public Health Agency of Canada, 2011). Participants also had diet quality scores over ten points higher than participants of the same age and gender in the HEI-C validation study (Garriguet, 2009). This would indicate that overall, study participants in this study are consuming more nutritious diets than their peers. Despite the high average diet quality and low obesity rates observed in study participants, a significant inverse relation between diet quality and depressive symptoms was still detected. While the nature of the sample used in this study certainly limits the generalizability of the results, it can likely be inferred that the association between diet quality and depressive symptoms would be further augmented if this study were conducted in a more diverse population of emerging adult females.
Finally, this study measured depressive symptoms using the CES-D rather than measuring clinician-diagnosed depression. The CES-D is not a diagnostic tool, and therefore measures only depressive symptoms (Radloff, 1977). It is thus likely that some of the participants in this study considered to have elevated depressive symptoms may in fact not have depression at all. For this reason, results of this study can only be interpreted with regards to depressive symptoms/depression risk, and not depression itself. Future studies should examine diet in a group of healthy subjects, and track diet and clinician-diagnosed depression over a period of years. This would ensure participants actually have depression rather than using elevated depressive symptoms as a proxy, and would better elucidate the temporal order of the association between diet and depression.

6.8 – Conclusions and Future Research

Depression is a complex medical condition for which there are many causes (American Psychiatric Association, 2014). It is becoming increasingly clear that many lifestyle factors, including diet, may be related to both its development and presentation (de Wit et al., 2010; Mikolajczyk et al., 2009; Regestein et al., 2008; Roberts et al., 2009; Sebena et al., 2012; Taliaferro et al., 2008; Vallance et al., 2011). It can be argued that research on depression and its lifestyle-related causes is particularly important in the emerging adult population. Emerging adults, those aged 18-25, are at particular risk of developing depression, and are at a key stage of dietary habit formation and development (Arnott, 2000, Nelson et al., 2008). Current research has shown a potential link between various dietary components and depression/depressive symptom, both in emerging adults and the general adult population (Liu et al., 2007; Mikolajczyk et al., 2009).
The study sample consisted of 141 female undergraduate university students. We found a significant, inverse association between diet quality and depressive symptoms, which suggests that subjects who were consuming diets of poorer nutritional quality were more likely to have elevated depressive symptoms. Non-significant associations were also noted between depressive symptoms and both fruit and vegetables intake and percent energy from “other foods”. Participants with lower intakes of fruits and vegetables were more likely to have elevated depressive symptoms, while participants who consumed more calories from “junk foods” not included in Canada’s Food Guide were also more likely to have elevated depressive symptoms. No association was found between depressive symptoms and each of omega-3 fatty acid intake, percent energy from protein, and glycemic index/glycemic load. These results suggest an association between high quality diet and lower risk of depression.

To our knowledge, this study was the first to examine the association between depressive symptoms and diet quality as measured by a validated index in an emerging adult population. Future studies examining the association between diet quality and depression should be prospective in nature in order to further elucidate whether poor diet quality causes depression, or depression causes poor diet quality. Randomized controlled trials should also be conducted to confirm whether a healthy diet could alleviate symptoms of depression or potentially prevent the onset of depressive disorder. Similar to this study, the continued use of three-day food records, and accounting for dietary under-reporters, would allow for the collection of high quality dietary data. Future studies should use clinician diagnosis to identify those with depression versus a self-report of depressive symptoms. Currently, most dietary research has been conducted only with regards to depressive symptoms or depression risk. The use of participants with clinician-
diagnosed depression would allow for a greater understanding of how diet relates to actual depressive disorder.

The results of this study provide important information for mental health professionals. While a cause-and-effect relation between diet and depressive symptoms cannot be deduced, it is likely that a diet of high nutritional quality is associated with lower levels of depressive symptoms. Limited evidence suggests that a high quality diet may alleviate some of the symptoms of depression (Sanchez-Villegas et al., 2013). Dietary interventions could thus be a low-risk adjunctive treatment to current psychotherapy and pharmacological protocols.

Universities would also benefit from this information, as almost one third of university students may be personally affected by depressive disorder (Ibrahim et al., 2013). Mental health professionals could use these results to educate students on the importance of consuming a healthful diet, both as a preventative measure and for those already living with depression. University food providers could also increase the availability of ready-to-eat healthy foods on campus (i.e., pre-cut vegetables and fruit, salads, and whole grains) as lack of time and energy is a major barrier to the consumption of healthful foods, and this may be further augmented amongst those with depression (Graham et al., 2013).

These results could also be of interest to physicians. Both poor diet and depression are strongly associated with chronic diseases such as cardiovascular disease, type 2 diabetes, and cancer (Hu et al., 2001; Joynt, Whellan, & O’Connor, 2003; Key, Allen, Spencer, & Travis, 2002; Knol et al., 2006; Panagiotakos, Pitsavos, & Stefanadis, 2006; Penninx et al., 1998). Since nutritionally poor diets and depression are considered risk factors for chronic disease, their prevention, identification, and treatment could reduce a patient’s risk of developing disease in the future. Health professionals could potentially identify those at risk of depression, and
implement dietary intervention before depressive disorder sets in. This may be especially important in emerging adults, who are at an increased risk for developing depressive disorders, and are also at a stage when their lifestyle habits are most malleable to change (Nelson et al., 2008). Prevention and treatment of both depressive disorders and poor quality diet at this early life stage would ensure both mental and physical health in the future, thus avoiding the development of various preventable co-morbidities.
7.0 - REFERENCES


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meeting recommended levels for fish and omega-3 fatty acid intake: Results of an analysis using observational data from NHANES 2003–2008. Nutrition Journal, 13(31), 64.


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Willett, W., & Stampfer, M. (1986). Total energy intake: Implications for epidemiologic


8.0 – APPENDIX

8.1 – Consent Form

CONSENT TO PARTICIPATE IN A CLASS PROJECT AND RESEARCH

Step Up Study Sessions

You are asked to participate in a class project and research study conducted by Dr. Andrea Buchholz and Dr. Jess Haines of the Dept Family Relations and Applied Nutrition, University of Guelph. Funding is from the Learning Enhancement Fund, University of Guelph.

If you have any concerns about this research, please feel free to contact:

• Dr. Jess Haines, tel 519-824-4120 ext 53870, jhaines@uoguelph.ca
• Dr. Andrea Buchholz, tel 519-824-4120 ext 52347, abuchhol@uoguelph.ca
• Graduate Research Assistant: Anne Szeto, szetoa@uoguelph.ca
• Graduate Research Assistant: Caroline Fraser, cfrase07@uoguelph.ca

PURPOSE

The main purpose of this project is a learning opportunity for students to participate in optional study sessions (Step-Up Study Sessions and assignment for NUTR*2050). It is also a research project because the investigators would like to publish the data collected.

PROCEDURES

There are three components of this project. You can volunteer to participate in none, one, two or all three components.

Component 1: Optional Assignment

This assignment will involve a tour of the University of Guelph Body Composition and Metabolism Lab (room 206 J.T. Powell Building), body composition testing (percent body fat) using the BOD POD, 3-day food records and diet analysis, a general health survey, and a physical activity survey. The optional assignment is described fully on the next page. You will also be asked to complete 1, 5-minute survey, during which you will be asked various questions on your experience with the assignment.

Component 2: Use of Data from the Optional Assignment for Research Purposes

If you choose to do the optional assignment above, we request your permission to use your data to explore the association between sleep, diet quality, and body composition in young adults.
Component 3: Assessment of the Step-Up study sessions for Research Purposes
You will be asked to complete two, 5-minute questionnaires to assess your study habits and approaches to learning; we will ask you to fill out these questionnaires during the first and final Step-Up study sessions of the semester. Also during the last Step-Up study session, you will also be asked to complete 1, 5-minute survey, during which you will be asked various questions on your experience in the study sessions, what you learned, etc. If you are not able to attend the last Step-Up study session, we will email you the surveys to complete and then email back to us.

Full Description of the Optional Assignment
If you volunteer to participate in the optional assignment, you will visit the Body Composition and Metabolism Lab once, for about 45 minutes. Approximately 1 week before your visit to the lab we will email you instructions and forms to complete a 3-day food record (a list of all the foods you ate over 3 days). We will ask you to bring the completed food record with you to your lab visit. At your lab visit, you will undergo a BOD POD test that will measure your body composition, pictured below.

The BOD POD™ measures the body’s per cent fat mass using air displacement. A test takes approx. 15 minutes.

The BOD POD, which will be operated by the Graduate Research Assistant, uses the displacement of air inside an enclosed chamber to determine your body volume. From the body volume measurements, whole-body density is determined and body fat is calculated. The procedure is simple and painless. The whole test takes approximately 15 minutes, but of these 15 minutes, you will sit in the BOD POD™ for only approximately 5 minutes. You will be asked to wear a bathing suit and bathing cap for this test to minimize the trapping of air between clothing, hair and your skin. Please bring your bathing suit with you. We will provide the bathing cap. You will also have your height measured using a wall mounted stadiometer. We will provide you with a copy of your results before you leave the lab.

We will ask you to complete some questionnaires that will ask about your health behaviours, sleep habits, physical activity habits and your experience in the lab, what you learned, etc. The questionnaires will take approximately 20 to 25 minutes in total to complete.

You will have the opportunity to complete an optional assignment – worth up to 5 extra percent on your grade for midterm 1 - and for which you will interpret your laboratory data. The assignment will be marked by another professor in the Applied Human Nutrition program, who will communicate your assignment mark to a course Graduate Teaching Assistant (GTA). The GTA – and not the instructor teaching the course – will make the necessary adjustment to your midterm grade. You will be eligible to receive up to a 5 percent bonus on midterm 1. E.g., if
you scored 53% on midterm 1, the optional assignment could raise your grade to a maximum of 58% (53% + 5%). Your marked assignment will be returned to you.

POTENTIAL RISKS AND DISCOMFORTS

Component 1: Optional Assignment
There is a small risk of claustrophobia while sitting in the BOD POD™. This risk is minimal, however you will be able to stop the test at any time by pressing a button under your left knee (and which will release the door), or by simply telling the Graduate Research Assistant that you would like the test to stop.

You may feel slightly embarrassed at having to wear a bathing suit, however, this will be minimized by wearing a hospital gown immediately before the test. You will be able to change back into your clothes immediately after the test. There is a film on the window of the lab, preventing passersby in the hall from seeing into the lab. We will also make sure that there will be very few people in the lab during your test.

If you do not feel comfortable completing the BOD POD™ test, you will be given a sample printout from the BOD POD, and so you will still be able to complete the optional assignment.

You may feel slightly embarrassed when filling out the questionnaires. You are free to omit any questions that may cause you some embarrassment.

Component 2 (Use of Data from the Optional Assignment for Research Purposes) and Component 3 (Assessment of the Step-Up Study Sessions for Research Purposes)
There is a small risk that someone other than the above mentioned researchers may see your completed survey data. To minimize this risk, your data will be coded immediately and stored in password-protected computer files. Thus, your individual data will not be identifiable with your name. Any results published or presented will be done using group data and/or coded (unidentifiable) results.

POTENTIAL BENEFITS TO PARTICIPANTS AND/OR TO SOCIETY
There are no direct benefits to you for participating in this class project and research study.

If you participate in the optional assignment, you will receive a data printout from the BOD POD and which will tell you your per cent body fat, and you will receive a personalized dietary analysis. In the community, body composition testing and dietary analysis can be expensive, but we will be providing this information free of charge. Data will be used for research purposes to advance the knowledge of body composition and dietary habits of young Canadian adults.

PAYMENT FOR PARTICIPATION
You will not receive any compensation, monetary or otherwise, for participating in this project.

CONFIDENTIALITY
Every effort will be made to ensure confidentiality of any identifying materials obtained during the study. Data will be coded immediately and stored in password-protected computer files. Thus your individual data will not be identified with your name. Any results published or presented will be done using group data and/or coded (unidentifiable) individual results. Data will be stored in the lab for seven years after which time paper records will be shredded and electronic records will be
deleted from computers. We will not use any of your data for anything other than what is indicated in this document.

PARTICIPATION AND WITHDRAWAL
You can choose whether to be in this class project and research, or not. If you volunteer to be in this class project and research study, you may withdraw at any time without consequences of any kind. You may exercise the option of removing your data from the study. You may also refuse to answer any questions you don’t want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise that warrant doing so.

If you decide not to participate in this research study, you can still complete the optional assignment.

RIGHTS OF RESEARCH PARTICIPANTS
You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. This study has been reviewed and received ethics clearance through the University of Guelph Research Ethics Board. If you have questions regarding your rights as a research participant, contact:

Research Ethics Coordinator Telephone: (519) 824-4120, ext. 56606
University of Guelph E-mail: sauld@uoguelph.ca
437 University Centre Fax: (519) 821-5236
Guelph, ON N1G 2W1

CONSENT AND SIGNATURE OF RESEARCH PARTICIPANT
I have read the information provided for the Step-Up Study Sessions and research study. My questions have been answered to my satisfaction, and I agree to participate. I have been given a copy of this form.

I have read this Consent Form. The components of the class project and research study, including the 3-day food record and the BOD POD™, have been explained to my satisfaction. I understand that I am free to stop participating in testing at any time, even after signing this consent form.

I agree to participate in the following components: (please check boxes for components in which you consent to participate):

☐ Component 1: Optional Assignment. I agree to visit the Body Composition and Metabolism Lab, undergo BOD POD testing, and complete a 3-day food record and some short questionnaires.

☐ Component 2: Use of Data from the Optional Assignment for Research Purposes. I give permission to the investigators to use my data from the optional assignment in a published manuscript and/or presentation at a conference. I understand that in order to do Component 2, I must also do Component 1.
Component 3: Assessment of the Step-Up Study Sessions for Research Purposes. I agree to complete two, 5-minute surveys that will ask about my study habits and approaches to learning, and one, 5-minute survey that will ask my opinions about the study sessions. I give permission to the investigators to use my survey data and my [NUTR*2050][NUTR*1010] course marks in a published manuscript and/or presentation at a conference.

PARTICIPANT

________________________________________  __________________________
(Printed name)                        (Signature)                       (Date)

____________________________________________________________
(University address)

____________________________________________________________
(Permanent address)

____________________________________________________________
(E-mail address)

WITNESS

________________________________________  __________________________
(Printed name)                        (Signature)                       (Date)
Health Behaviour Questionnaire

**SECTION I: Demographics**

1. Age: _______ years

2. Gender:
   - □ male
   - □ female

3. How do you define yourself? *(Check all that apply)*
   - □ White, Caucasian
   - □ Black, African Canadian, African American
   - □ Middle Eastern, Arabic
   - □ South Asian (i.e., Indian, Pakistan)
   - □ East Asian (i.e., China, Japan)
   - □ Southeast Asian (i.e., Thailand, Philippines, Malaysia)
   - □ Hispanic
   - □ Native
   - □ Other (specify):______________________

**SECTION II: Health**

4. Do you have any of the following health conditions?
   - □ Cardiovascular Disease
5. Are you currently taking any prescription medications? If so, please list the medications you are currently taking:
_______________________________________________________________________
_______________________________________________________________________

SECTION III: Overall Mood

6. Below is a list of the ways you might have felt or behaved. Please tell me how often you have felt this way during the past week.

<table>
<thead>
<tr>
<th></th>
<th>Rarely or none of the time (less than 1 day)</th>
<th>Some or a little of the time (1-2 days)</th>
<th>Occasionally or a moderate amount of time (3-4 days)</th>
<th>Most or all of the time (5-7 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) I was bothered by the things that usually don’t bother me.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>b) I did not feel like eating; my appetite was poor.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>c) I felt that I could not shake off the blues even with help from my family or friends.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>d) I felt I was just as good as other people.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>e) I had trouble keeping my mind on what I was doing.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>f) I felt depressed.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>g) I felt that everything I did was an effort.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>h) I felt hopeful about the future.</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>
SECTION IV: Lifestyle

7. Do you consume alcohol-containing drinks?
   □ Yes
   □ No

If yes, on average how many do you consume per week?
*(One drink is equivalent to: 12 oz beer, 12 oz alcoholic cooler, 4 oz wine, 1 oz hard liquor):___________

8. Do you consume caffeine-containing drinks?
   □ Yes
   □ No

If yes, on average how many do you consume per week?
*(One drink is equivalent to: 8 oz coffee, 24 oz tea, 24 oz soft drink):___________

9. Do you smoke cigarettes/cigars?
   □ Yes
   □ No

If yes, how many cigarettes/cigars per day?________________________

For how long have you smoke?_______________________________
SECTION V: Physical Activity Level

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the vigorous activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

10. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?

_____ days per week

□ No vigorous physical activities  →  Skip to question 12

11. How much time did you usually spend doing vigorous physical activities on one of those days?

_____ hours per day

_____ minutes per day

□ Don’t know/Not sure

Think about all the moderate activities that you did in the last 7 days. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.
12. During the last 7 days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

_____ days per week

☐ No moderate physical activities ➔ Skip to question 14

13. How much time did you usually spend doing moderate physical activities on one of those days?

_____ hours per day

_____ minutes per day

☐ Don’t know/Not sure

Think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you have done solely for recreation, sport, exercise, or leisure.

14. During the last 7 days, on how many days did you walk for at least 10 minutes at a time?

_____ days per week

☐ No walking ➔ Skip to question 16

15. How much time did you usually spend walking on one of those days?

_____ hours per day

_____ minutes per day

☐ Don’t know/Not sure
This question is about the time you spent sitting on weekdays during the last 7 days. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

16. During the last 7 days, how much time did you spend sitting on a week day?

_____ hours per day

_____ minutes per day

☐ Don’t know/Not sure

SECTION VI: Sleep

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, what time have you usually gone to bed at night?

   BED TIME __________

2. During the past month, how long (in minutes) has it usually take you to fall asleep each night?

   NUMBER OF MINUTES __________

3. During the past month, what time have you usually gotten up in the morning?

   GETTING UP TIME __________

4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)

   HOURS OF SLEEP PER NIGHT __________
For each of the remaining questions, check the one best response. Please answer all questions.

5. During the past month, how often have you had trouble sleeping because you ...

a) Cannot sleep within 30 minutes

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

b) Wake up in the middle of the night or early morning

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

c) Have to get up to use the bathroom

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

d) Cannot breathe comfortably

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

e) Cough or snore loudly

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

f) Feel too cold

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

g) Feel too hot

<table>
<thead>
<tr>
<th></th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
</table>
h) Had bad dreams

Not during the past month ____  Less than once a week ____  Once or twice a week ____  Three or more times a week ____

i) Have pain

Not during the past month ____  Less than once a week ____  Once or twice a week ____  Three or more times a week ____

j) Other reason(s), please describe ________________________________
____________________________________________________________________

How often during the past month have you had trouble sleeping because of this?

Not during the past month ____  Less than once a week ____  Once or twice a week ____  Three or more times a week ____

6. During the past month, how would you rate your sleep quality overall?

Very good _____
Fairly good _____
Fairly bad _____
Very bad _____

7. During the past month, how often have you taken medicine to help you sleep (prescribed or “over the counter”)?

Not during the past month ____  Less than once a week ____  Once or twice a week ____  Three or more times a week ____

8. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Not during the past month ____  Less than once a week ____  Once or twice a week ____  Three or more times a week ____
9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

- No problem at all __________
- Only a very slight problem __________
- Somewhat of a problem __________
- A very big problem __________
Step Up study    ID#:____________ (for office use only)

Three-Day Food Record

Participant name: _________________________________

Dates of recorded intake: _________________________

Research Assistant: _____________________________

Contact Information:     ___519-824-4120 ext. 56715; email szetoa@uoguelph.ca

Instructions for Keeping Your Three-Day Food Record

• PLEASE BRING THIS COMPLETED FOOD RECORD WHEN YOU VISIT THE LAB
• Please keep your three-day food record for three non-consecutive days. The days should include two weekdays and one weekend day. Select days that closely resemble your usual eating habits.
• Each time you eat or drink anything (meals, snacks, etc.) during the three days, write down what and how much you ate. Indicate what you consumed, not what you had on your plate. Please also indicate the time of your meal or snack.
• Include any supplements you are taking. Record the brand name (e.g., Centrum Women under 50) or type (e.g., 400 IU Vitamin D) and amount taken (e.g., 1 tablet every day).
• To measure how much was eaten, use a set of measuring cups and spoons to help estimate amounts. Also see the examples below to estimate portion sizes.
• Note if food choices are homemade or purchased. Please include brand names whenever possible.

Amounts and Conversions
1/4 cup = 50 ml or 4 Tablespoons
1/3 cup = 75 ml or 5 1/2 Tablespoons
1/2 cup = 125 ml or 8 Tablespoons
2/3 cup = 150 ml or 10 1/2 Tablespoons
3/4 cup = 175 ml or 12 Tablespoons
1 cup = 250 ml or 16 Tablespoons
1 oz = 1 slice of processed cheese or lunchmeat

How to Estimate Your Portion Size

<table>
<thead>
<tr>
<th>Meat</th>
<th>Three (3) ounces of meat are about the size and thickness of a deck of playing cards or an audiotape cassette.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fruit</td>
<td>A medium apple or peach is about the size of a tennis ball.</td>
</tr>
<tr>
<td>Grains</td>
<td>One cup of rice or pasta is about the size of your fist.</td>
</tr>
<tr>
<td>Cheese</td>
<td>One ounce of cheese is about the size of four dice.</td>
</tr>
</tbody>
</table>

### Three-Day Food Record Checklist

<table>
<thead>
<tr>
<th>Beverages</th>
<th>What kind of milk? Homo, 2%, 1%, skim, other. Was it fruit juice or fruit beverage or drink?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breads</td>
<td>Did you spread on butter or margarine?</td>
</tr>
<tr>
<td>Cereal</td>
<td>Did you add milk?</td>
</tr>
<tr>
<td></td>
<td>Did you add sugar or fruit?</td>
</tr>
<tr>
<td>Dairy</td>
<td>What brand or kind of yogurt?</td>
</tr>
<tr>
<td></td>
<td>What brand or kind of cheese?</td>
</tr>
<tr>
<td>Vegetables</td>
<td>Was it raw or cooked?</td>
</tr>
<tr>
<td></td>
<td>Was it fresh, frozen or canned?</td>
</tr>
<tr>
<td></td>
<td>Did you add any butter, margarine or sauce?</td>
</tr>
<tr>
<td>Fruit</td>
<td>Was it a small, medium or large fruit?</td>
</tr>
<tr>
<td></td>
<td>Was it fresh, frozen or canned?</td>
</tr>
<tr>
<td>Grains</td>
<td>Did you add any butter, margarine, peanut butter, jam or honey?</td>
</tr>
<tr>
<td></td>
<td>Was it a half or whole sandwich?</td>
</tr>
<tr>
<td></td>
<td>Was it a small or large muffin or bagel?</td>
</tr>
<tr>
<td>Fish</td>
<td>Was your canned fish packed in water or oil?</td>
</tr>
<tr>
<td></td>
<td>How did you cook your fish?</td>
</tr>
<tr>
<td>Meats</td>
<td>How did you cook your meat?</td>
</tr>
<tr>
<td></td>
<td>What kind of cut was it e.g. chicken leg or chicken breast?</td>
</tr>
<tr>
<td>Soups</td>
<td>Was your soup prepared with milk, water or cream?</td>
</tr>
<tr>
<td>Restaurants</td>
<td>What restaurant was it?</td>
</tr>
<tr>
<td>Packaged food</td>
<td>What brand was it?</td>
</tr>
</tbody>
</table>
## Sample Food Record

**Day 1: Tuesday, Oct 15, 2012**

<table>
<thead>
<tr>
<th>Time of Meal or Snack</th>
<th>Type of Food or Beverage Offered</th>
<th>Amount Eaten</th>
<th>Method of Preparation or Brand</th>
<th>Comments (e.g. amount of food served, too tired to eat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>Cereal</td>
<td>½ cup</td>
<td>Honey Nut Cheerios</td>
<td></td>
</tr>
<tr>
<td>Time: 8:30 am</td>
<td>Milk 2%</td>
<td>½ cup</td>
<td></td>
<td>On cereal</td>
</tr>
<tr>
<td></td>
<td>Banana</td>
<td>½ med</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time: 10:00 am</td>
<td>Animal Crackers</td>
<td>10</td>
<td>Christie</td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td>-----------------</td>
<td>----</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Apple juice</td>
<td>4 oz</td>
<td>Allen’s pure apple juice-canned</td>
<td></td>
</tr>
<tr>
<td>Time: 12:30pm</td>
<td>Grilled cheese sandwich</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Whole wheat bread</td>
<td>1 slice</td>
<td>Dempsters</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cheese slice</td>
<td>1 slice</td>
<td>Kraft slices</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Butter on bread</td>
<td>1 Tbsp</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yogurt – strawberry</td>
<td>75 ml</td>
<td>Mini-go</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------</td>
<td>-------</td>
<td>---------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk</td>
<td>½ cup</td>
<td>2%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**PM Snack**  
*Time: 4:00 pm*

| Granola bar | 1 bar | 35 g | Quaker Chewy, Trail Mix – tropical fruit | Ate half of it |

**Dinner**  
*Time: 6:00 pm*

<table>
<thead>
<tr>
<th>Chicken fingers</th>
<th>1 ½</th>
<th>President’s Choice</th>
</tr>
</thead>
<tbody>
<tr>
<td>French fries</td>
<td>10</td>
<td>McCain regular</td>
</tr>
<tr>
<td>Honey</td>
<td>2 Tbsp</td>
<td>For dipping</td>
</tr>
<tr>
<td>-----------</td>
<td>--------</td>
<td>-------------</td>
</tr>
<tr>
<td>Ketchup</td>
<td>2 Tbsp</td>
<td>Heinz</td>
</tr>
<tr>
<td>Carrots</td>
<td>½ medium</td>
<td>Raw, cut in sticks</td>
</tr>
<tr>
<td>Milk</td>
<td>½ cup</td>
<td>2%</td>
</tr>
</tbody>
</table>

**Evening Snack**  
Time: 8:00 pm

| Ice cream | 1 cup | Chocolate Nestle |

**Does this represent a typical day?: [X] Yes [ ] No**
<table>
<thead>
<tr>
<th>Time of Meal or Snack</th>
<th>Type of Food or Beverage Offered</th>
<th>Amount Eaten</th>
<th>Method of Preparation or Brand</th>
<th>Comments (e.g. amount of food served, too tired to eat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time: ______</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AM Snack</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time: ______</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lunch</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time: ______</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PM Snack</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Day 2

<table>
<thead>
<tr>
<th>Time of Meal or Snack</th>
<th>Type of Food or Beverage Offered</th>
<th>Amount Eaten</th>
<th>Method of Preparation or Brand</th>
<th>Comments (e.g. amount of food served, too tired to eat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time: ______</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Does this represent a typical day?: [ ] Yes [ ] No
<table>
<thead>
<tr>
<th>Time</th>
<th>AM Snack</th>
<th>Time: ______</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lunch</td>
<td>Time: _____</td>
<td></td>
</tr>
<tr>
<td>PM Snack</td>
<td>Time: ______</td>
<td></td>
</tr>
<tr>
<td>Dinner</td>
<td>Time: _____</td>
<td></td>
</tr>
<tr>
<td>Time of Meal or Snack</td>
<td>Type of Food or Beverage Offered</td>
<td>Amount Eaten</td>
</tr>
<tr>
<td>-----------------------</td>
<td>----------------------------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Breakfast</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time: ______</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Evening Snack         |                                  |              |                               |                                                      |
| Time: ______          |                                  |              |                               |                                                      |

Does this represent a typical day? [ ] Yes [ ] No
<table>
<thead>
<tr>
<th>AM Snack</th>
<th>Time: ______</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lunch</td>
<td>Time: ______</td>
</tr>
<tr>
<td>PM Snack</td>
<td>Time: ______</td>
</tr>
<tr>
<td>Dinner</td>
<td>Time: ______</td>
</tr>
<tr>
<td>Evening Snack</td>
<td></td>
</tr>
<tr>
<td>Time:</td>
<td></td>
</tr>
</tbody>
</table>

Does this represent a typical day?: [ ] Yes [ ] No
8.4 – Healthy Eating Index Scoring Codes

Points to Calculate Canadian HEI based on # of Food Guide Servings (Adequacy) and % of Energy or Mg Sodium (Moderation)

<table>
<thead>
<tr>
<th>Adequacy</th>
<th>Point range</th>
<th>0</th>
<th>0.50</th>
<th>1</th>
<th>1.5</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Veg and Fruit</td>
<td>0 to 10</td>
<td>0</td>
<td>1.4</td>
<td>2.8</td>
<td>4.2</td>
<td>5.6</td>
<td>7</td>
<td>8.4</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole Fruit</td>
<td>0 to 5</td>
<td>0</td>
<td>1.67</td>
<td>3.34</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drk Green + Org Veg</td>
<td>0 to 5</td>
<td>0</td>
<td>1.67</td>
<td>3.34</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total grains</td>
<td>0 to 5</td>
<td>0</td>
<td>0.83</td>
<td>1.66</td>
<td>2.5</td>
<td>3.32</td>
<td>4.15</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole grains</td>
<td>0 to 5</td>
<td>0</td>
<td>1.67</td>
<td>3.34</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Milk &amp; Alt</td>
<td>0 to 10</td>
<td>0</td>
<td>2.5</td>
<td>5</td>
<td>7.5</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meat &amp; Alt</td>
<td>0 to 10</td>
<td>0</td>
<td>2.5</td>
<td>5</td>
<td>7.5</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unsaturated fats (15ml=1svg)</td>
<td>0 to 10</td>
<td>0</td>
<td>2.5</td>
<td>5</td>
<td>7.5</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Moderation</th>
<th>≥15 %</th>
<th>14%</th>
<th>13%</th>
<th>12%</th>
<th>11%</th>
<th>10%</th>
<th>8 or 9%</th>
<th>7%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated fats (%)</td>
<td>0 to 10</td>
<td>0</td>
<td>1.6</td>
<td>3.2</td>
<td>4.8</td>
<td>6.4</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>460 430 400 370</td>
<td>3400 3100 2800 2300 1500</td>
<td>2300 1500</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other Food (%)</td>
<td>0 to 20</td>
<td>0</td>
<td>2.86</td>
<td>5.71</td>
<td>8.57</td>
<td>11.42</td>
<td>14.29</td>
<td>17.14</td>
</tr>
</tbody>
</table>