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Lochen, Heidi A. *Vitamin D Intake in Relationship to Mood and to Vitamin D-Related Knowledge in Undergraduate Students*

**Abstract**

High prevalence of vitamin D insufficiency is reported in the young adult population and individuals residing in northern latitudes due to negligible vitamin D synthesis from ultraviolet light exposure in winter months, therefore, obtaining adequate vitamin D is increasingly important. However, evidence suggests that university students are not consuming the recommended dietary allowance (RDA) for vitamin D. In recent years, vitamin D insufficiency has been found to have a correlational link and therapeutic effect on depressive symptoms. This research explored the relationship between vitamin D intake and mood, and vitamin D intake and vitamin D-related knowledge.

A survey was used to collect demographics, food frequency questionnaire (targeting vitamin D), supplement history, ultraviolet-B exposure, vitamin D-related knowledge questions, and a clinical depression scale score. The results show that only 1.3% of students were consuming the RDA for vitamin D. Vitamin D-related knowledge was found to have a small, yet significant, inverse relationship on vitamin D intake. Additionally, 21.9% of participants reported moderate to severe level depression. The current study did not find a relationship between total vitamin D intake and depression in the winter months, however, participant intakes were skewed and therefore the researcher recommends further investigation into this possible relationship.
Acknowledgments

With the completion of this research paper, I would like to express my sincere gratitude to Dr. Alexandra Hall for her patience, guidance, support, and expertise during this entire process. Dr. Hall, I have nothing but the utmost respect for you and appreciate all you have done for me throughout the past two years. To the amazing thesis committee who volunteered their time, energy, and knowledge to me, thank you so very much. Dr. Karen Ostenso, Dr. Kerry Peterson, and Dr. Julie Bates-Maves, you have assisted me so much in this process and I am so grateful to have had the opportunity to work with each one of you. I would also like to thank the Office of Research and Sponsored Programs and Dr. Julie Beston for their assistance. Lastly, to my family, friends, and my rescue dog Smokey, thank you for your love.
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Chapter I: Introduction

Vitamin D was discovered by Sir Edward Mellanby, who was studying the role of diet in the prevention of rickets in 1919-1920 (Norman, & Henry, 2012). Mellanby concluded that cod liver oil was an effective solution to prevent the development of rickets in canines without any exposure to UV lighting, and therefore this component was deemed a vitamin (Norman, 2012). McCollum, Simmonds, Becker, and Shipley in 1922 examined the antirachitic factor that was found in cod liver oil and named it vitamin D (Rajakumar, 2003). McCollum et al. (1922) also discovered vitamin D’s role in the absorption of calcium. Additionally, after being deemed a vitamin, in 1936 researchers categorized the molecule Vitamin D$_3$ as being chemically a steroid (Norman, 2012).

Dietary vitamin D can be found naturally in fatty fish, fish liver oils, and eggs (from hens fed vitamin D). After discovery of the role of vitamin D in the prevention of rickets, fortification of milk began in the 1920’s and is a large contributor of vitamin D into the diet. Other foods commonly fortified with vitamin D include cereals, some yogurts and cheeses, orange juice, and non-dairy milk. Common sources of vitamin D, according to the U.S. Department of Agriculture, Agricultural Research Service (2011), can be found in Table 1. Vitamin D supplements can commonly be found and are available without a prescription. They can provide a variety of doses, however, most commonly provide 10-25 μg when found in a multivitamin, 10-25 μg in many calcium supplements, or 10-1,250 μg in a single ingredient supplement.
Table 1

Common Dietary Sources of Vitamin D (U.S. Department of Agriculture, Agricultural Research Service, 2011)

<table>
<thead>
<tr>
<th>Dietary Source</th>
<th>Serving</th>
<th>Vitamin D (μg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pink Salmon, canned</td>
<td>3 ounces</td>
<td>11.6</td>
</tr>
<tr>
<td>Mackerel, canned</td>
<td>3 ounces</td>
<td>5.3</td>
</tr>
<tr>
<td>Sardines, canned</td>
<td>3 ounces</td>
<td>4.1</td>
</tr>
<tr>
<td>Milk, low-fat, fortified</td>
<td>8 ounces</td>
<td>2.5</td>
</tr>
<tr>
<td>Orange Juice, fortified</td>
<td>8 ounces</td>
<td>2.5</td>
</tr>
<tr>
<td>Breakfast Cereals, fortified</td>
<td>1 cup</td>
<td>1.0-1.3</td>
</tr>
<tr>
<td>Egg, whole or yolk</td>
<td>1 large</td>
<td>0.9</td>
</tr>
</tbody>
</table>

Vitamin D is found in dietary sources and supplements in two major forms: vitamin D$_2$ (ergocalciferol) and vitamin D$_3$ (cholecalciferol). The vitamin D$_2$ analog is photosynthesized in mushrooms, yeasts, and plants, and D$_3$ is synthesized cutaneously by animals in response to ultraviolet-B radiation (UVB). Sources of vitamin D for humans include UVB, food sources, and supplementation. UVB radiation stimulates the production of the Vitamin D in the skin, however, at latitudes of 42° N and higher, is only readily available during the spring, summer, and fall months (April-October) (Engelsen, Brustad, Aksnes, & Lund, 2005). Vitamin D$_3$ is then converted to 25-hydroxyvitamin D (25(OH)D) in the liver and is further hydroxylated in the kidneys to 1,25-dihydroxyvitamin D, otherwise known as calcitriol. Calcitriol is the most active form of vitamin D in the body.

The RDA for vitamin D was established in 2010 based on the amount of vitamin D that is required for bone health. There is some debate as to whether this target is adequate to raise serum concentrations of vitamin D to optimal status. (Holick et al., 2011). The Dietary Reference
Intakes (DRI’s), according to the Institute of Medicine, Food and Nutrition Board (2010), for vitamin D are displayed in table 2.

Table 2

*Vitamin D Dietary Reference Intakes (DRIs) (Institute of Medicine, Food and Nutrition Board, 2010)*

<table>
<thead>
<tr>
<th>Life Stage</th>
<th>AI</th>
<th>EAR</th>
<th>RDA</th>
</tr>
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<tbody>
<tr>
<td>Infants (0-12 months)</td>
<td>10 μg</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Children (1-8 years)</td>
<td>-</td>
<td>10 μg</td>
<td>15 μg</td>
</tr>
<tr>
<td>Adults (&gt;8 years)</td>
<td>-</td>
<td>10 μg</td>
<td>15 μg</td>
</tr>
<tr>
<td>Pregnancy and Lactation</td>
<td>-</td>
<td>10 μg</td>
<td>15 μg</td>
</tr>
</tbody>
</table>

*Note. AI = Adequate Intake; EAR = Estimated Average Requirement; RDA = Recommended Dietary Allowance*

Vitamin D toxicity has never been reported from sun exposure, due to photolytic degradation of vitamin D into other compounds (Webb, Kline, & Holick, 1988; Volmer, Mendes, & Stokes, 2015). Additionally, food sources, even those that are fortified, do not contain large doses. Therefore, vitamin D toxicity is only seen due to excessive ingestion of supplements. The main consequence of vitamin D toxicity, usually seen with long-term doses greater than 1,250 μg is hypercalcemia, which can lead to kidney stones and calcification of organs (Jones, 2008). Although research shows that doses of vitamin D below 205 μg are unlikely to cause toxicity, the Institute of Medicine, Food and Nutrition Board (2010) established the tolerable upper levels for all groups which can be found in table 3.
Table 3

*Tolerable Upper Intake Level for Vitamin D (Institute of Medicine, Food and Nutrition Board, 2010)*

<table>
<thead>
<tr>
<th>Age Group</th>
<th>UL (μg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants 0-6 months</td>
<td>25</td>
</tr>
<tr>
<td>Infants 6-12 months</td>
<td>37.5</td>
</tr>
<tr>
<td>Children 1-3 years</td>
<td>62.5</td>
</tr>
<tr>
<td>Children 4-8</td>
<td>75</td>
</tr>
<tr>
<td>Ages 9 and above</td>
<td>100</td>
</tr>
</tbody>
</table>

*Note. UL = Tolerable Upper Intake Level*

Vitamin D plays a crucial role in bone health by assisting in regulating and balancing calcium and phosphorus concentrations. Receptors for vitamin D are found not only in bone, they are also found on numerous types of cells in the body. By the actions of these vitamin D receptors (VDRs), vitamin D deficiency is also hypothesized to play a role in certain types of cancers, autoimmune disorders, type two diabetes mellitus, cardiovascular disease, neurodegenerative diseases, and mood disorders (Aydin, Varkal, Toker, Ozer, Karamustafalioglu, 2015).

Serum vitamin D is most commonly measured in the body as 25(OH)D as it has a longer blood half-life compared to other serum measures and is the best reflection of vitamin D status of an individual. Vitamin D insufficiency can occur as a result of lack of sun exposure or inadequate vitamin D in the diet without supplementation. Normal ranges for vitamin D were developed by the Institute of Medicine, Food and Nutrition Board (2010) based on avoidance of skeletal consequences of deficiency which are rickets in infants and children (Prié, Beck, Urena, & Friedlander, 2005; Zhao & Tenehouse, 2000), and osteomalacia in adults (Beck et al., 1998;
Deficiency is characterized as serum concentrations of 12 ng/ml or less, whereas, insufficiency of vitamin D occurs when serum vitamin D falls between 12 and 19 ng/ml (Institute of Medicine, Food and Nutrition Board, 2010). The prevalence of vitamin D insufficiency and deficiency together is estimated to be about 36% and 37% for males and females, respectively (Looker et al., 2011).

Non-skeletal consequences of vitamin D deficiency and insufficiency have been researched, such as the prevalence of depression (Anglin, Samaan, Walter, & McDonald, 2013; Ju et al., 2013). Vitamin D hormone regulates serotonin synthesis by activation of the tryptophan hydroxylase 2 (TPH2) gene, which is responsible for the conversion of tryptophan into serotonin in the brain (Patrick & Ames, 2014). Vitamin D receptors and 1α-hydroxylase, the enzyme involved in the formation of active vitamin D, are widely distributed in the human brain (Eyles, Smith, Kinobe, Hewison, & McGrath, 2005). The amygdala, thalamus, and hypothalamus are brain regions thought to have roles in the development of depression, and strong VDR and 1α-hydroxylase immunoreactivity has been found in the nuclei of the hypothalamus (Eyles et al., 2005).

Depression is one of the most severe public health issues in the United States; the Centers for Disease Control and Prevention indicates that nearly 1 in 10 United States citizens, ages 18 and older has a depressive disorder (González, Tarraf, Whitfield, & Vega, 2010). This is troublesome for many reasons, including that the pathophysiology of depression is not completely understood. The theories of the pathophysiology of depression include genetic vulnerability, dysfunction of specific brain regions, neurotoxic processes, and deficiency of monoamines, such as serotonin (Hasler, 2010). Even with the emerging research, a well-founded conclusion to the final pathophysiology is not firmly developed. Depression is a complex, and
multifactorial condition stemming from environmental and genetic backgrounds. Clinical symptoms of depressive disorders can include persistent feelings of sadness, hopelessness, or worthlessness, difficulty with concentration, appetite changes, irritability or pains and aches of all kinds (U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Mental Health, 2015). Symptoms can even include thoughts of death or suicide attempts. Specific risk factors for depression include major life changes, trauma or stress, certain physical illnesses, and medications (U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Mental Health, 2015). These risk factors, however, account for a small role in the pathophysiology and development of depression.

Depression is one of the leading causes of disability among young adults in the United States (Ganji, Milone, Cody, & McCarty, 2010). The American College Health Association (2016) found that 15.4% of students feel that depression symptoms had a significant impact on their academic performance within the past 12 months. According to the University of Wisconsin-Stout’s own counseling center, depression and anxiety related symptoms comprise about 68% of their admission complaints (Achter, 2015.) Additionally, there has been a 22.2% one-year increase in the number of individuals seeking counseling and a 4.5% increase in sessions completed, with 7.1% of enrolled students (9,371) utilizing the University counseling center during the 2014-2015 academic year.

Current treatment options for individuals with depression include medications and psychotherapy. Psychotherapy assists in teaching individuals’ skills in thinking, behaving, and coping. Medication is a common intervention for depression; however, an individual may have to try a few medications before finding a type and dosage that works, as individual responses to antidepressants can vary (Preskorn, 2014). Non-prescription pharmaceutical options are of
interest due to the significant number of both people who experience mild depressive symptoms and individuals diagnosed with depression who are interested in adjunctive or alternative therapies (Barnes, Bloom, & Nahin, 2008).

Vitamin D may play a role in depression via its role in serotonin synthesis and release. Several epidemiological studies have found a correlation between low vitamin D and the prevalence of depressive symptoms (Anglin et al., 2013; Ju et al., 2013). Furthermore, vitamin D has been shown in two large meta-analyses of randomized controlled trials to have an intervention effect on reducing depressive symptoms and prevalence (Spedding, 2014; Shaffer et al., 2014).

Statement of the Problem

Vitamin D is essential for not only bone health, but for many other physiological aspects of health. Several researchers have concluded through meta-analyses that low serum concentrations of vitamin D are strongly associated with higher levels of depressive symptoms (Anglin et al., 2013, Ju et al., 2013). However, minimal data report the relationship between total vitamin D intake and mood.

Vitamin D insufficiency and deficiency prevalence in the general population is estimated to be at 36% and 37% for males and females aged 19-30, respectively (Looker et al., 2011). Vitamin D intake, from both dietary sources and supplements, and UVB radiation contribute to serum vitamin D status. Vitamin D insufficiency is most prevalent during the winter months due to practically negligible UVB rays available for vitamin D synthesis during November to March in latitudes above 45° N (Webb et al., 1988). The campus of the University of Wisconsin-Stout is located in Menomonie, WI (44.8° N); therefore, adequate dietary consumption of vitamin D is extremely important during the winter months. However, researchers are finding that young
adults and more specifically college students, on average are not consuming over the EAR nor the RDA for vitamin D (Cress, 2014; Ouellette et al., 2012; Forney et al., 2014; Lacey, Stolfo & Rieger, 2004). These researchers all recommend efforts to increase vitamin D-related knowledge to improve intake. However, no correlational data showing that those with higher vitamin D-related knowledge are consuming more vitamin D, was found in a literature search.

**Purpose of the Study**

The purpose of this study was to determine the relationship between the daily vitamin D, from dietary sources and supplementation, vitamin D-related knowledge, and depressive symptoms of undergraduate students at the University of Wisconsin-Stout. These relationships were assessed using an adapted validated vitamin D intake questionnaire, an adapted vitamin D-related knowledge questionnaire, and the Patient Health Questionnaire, form 9. After approval by the University of Wisconsin-Stout’s Institutional Review Board for the Protection of Human Subjects, the data were collected by an online survey sent by email to students.

**Research Objectives**

This research project had the following objectives:

1. To determine the estimated daily intake of vitamin D consumed by undergraduates at the University of Wisconsin-Stout
2. To assess the prevalence and severity of depressive symptoms among students using the PHQ-9 mood score
3. To establish students’ level of vitamin D-related knowledge
4. To examine the predictive relationship of predictors of vitamin D status (intake, supplement use, UV exposure, sex, race/ethnicity, and BMI) to PHQ-9 score
5. To analyze the predictive relationship of vitamin D-related knowledge on modifiable predictors of vitamin D status (daily intake of vitamin D, supplement use, and UV exposure)

Definition of Terms

This section’s purpose is to define several terms to assist the reader in comprehension, and for clarity of the study.

Patient Health Questionnaire. The Patient Health Questionnaire is a clinical instrument used to make criteria-based diagnosis of depression and other mental illnesses. The form used in this research is form 9, which is validated with 9 items.

Vitamin D deficiency. The Institute of Medicine, Food and Nutrition Board (2010) defines vitamin D deficiency as serum 25-Hydroxyvitamin D concentrations of less than 12 ng/ml. This serum concentration is associated with the development of rickets in infants and children and osteomalacia in adults.

Vitamin D insufficiency. The Institute of Medicine, Food and Nutrition Board (2010) defines vitamin D insufficiency as serum 25-Hydroxyvitamin D concentrations from 12 to 19 ng/ml. This serum concentration is associated with inadequate bone and overall health status.

Vitamin D sufficiency. The Institute of Medicine, Food and Nutrition Board (2010) defines vitamin D sufficiency as serum 25-Hydroxyvitamin D concentrations greater than 20 ng/ml. This serum concentration is associated with adequate overall and bone health.

Vitamin D toxicity. The Institute of Medicine, Food and Nutrition Board (2010) defines vitamin D toxicity as serum 25-Hydroxyvitamin D concentrations of greater than 50 ng/ml. This serum concentration is associated with adverse health effects.
Assumptions of the Study

Several assumptions were made prior to the distribution of this survey: first, that participants of this study were able to read, comprehend, and complete the survey, second that individuals were answering the questions as truthfully and thoughtfully as possible, and third that dietary intake is the main contributor to total body vitamin D in winter months.

Limitations of the Study

There were several limitations in this study. First, the intake questionnaire used to assess dietary vitamin D had been validated in postmenopausal women, and was conducted during in-person interviews (Hacker-Thompson, Schloetter, & Sellmeyer, 2012). Due to time and staff constraints this study was unable to conduct diet recalls in-person, which may affect the results in this study due to either over- or under-reporting intake. Second, there is the assumption that dietary intake is the main contributor to total body vitamin D in the winter months, and therefore, not testing serum vitamin D is a limitation to this study.

Methodology

The objective of this study was to determine the daily intake of vitamin D and its relationship to vitamin D-related knowledge and mood scores in undergraduate students at the University of Wisconsin-Stout by administration of a survey.

Professors of a variety of programs on campus received an invitation to assist graduate research, and forwarded the email invitation to students. Students received this email from their professors, which contained information about the research, a link to the survey, and notification of a follow up survey that would allow them to win one of five, $20 Amazon Gift Cards. Upon entrance to the survey, participants were presented with an implied consent form, which provided information on risks and benefits to the student, time commitment, confidentiality, right
to withdraw, and information on the study’s approval from the University of Wisconsin-Stout Institutional Review Board for the Protection of Human Subjects.

The survey consisted of four sections: demographics, serum vitamin D predictors, vitamin D-related knowledge, and mood survey. The demographics included enrollment status, age, biological sex, race/ethnicity, and height and weight (used to calculate BMI). Questions regarding serum vitamin D predictors included: UVB exposure history, supplement use, and dietary intake, which was measured by a modified version of the Brief Vitamin D Questionnaire (Hacker-Thompson et al., 2012). Vitamin D-related knowledge utilized a series of questions adapted from Boland, Irwin, and Johnson (2014). Lastly, participants completed the Patient Health Questionnaire, form 9 (PHQ-9) which is a nine-item questionnaire used in clinical settings to diagnose depression.

The Statistical Program for Social Sciences (SPSS) version 23.0 (2014) was used to analyze data. To assess the relationship between depressive symptoms and vitamin D intake, a linear multiple regression was used with PHQ-9 scores as independent variables and vitamin D intake, race/ethnicity, BMI, and biological sex as predictor variables. To further assess the relationship between PHQ-9 score and total dietary intake, an independent t-test was used to compare the mean composite PHQ-9 scores among the group of participants consuming over the estimated average requirements (EAR, 10 µg) for vitamin D and below. To analyze a possible threshold effect, an independent t-test was also run to compare mean PHQ-9 scores between the group of participants who consumed greater versus less than the RDA (15 µg) for vitamin D. Linear regressions were used to determine the relationship between vitamin D intake and mood, and vitamin D intake and vitamin D-related knowledge. Two independent t-tests were run to examine the mean knowledge score between the group of individuals who consumed the EAR
and between those who consumed over the RDA. Frequency and descriptive statistics were run for all pertinent variables.
Chapter II: Literature Review

Vitamin D is an important fat-soluble vitamin obtained from dietary sources, ultraviolet B rays (UVB), and/or supplements. Vitamin D has been thought to only be essential for bone health, however, in recent years vitamin D has emerged as having a possible association with cancer, cardiovascular disease, neurodegenerative conditions, and mood disorders.

Vitamin D deficiency has a correlational link to depression (Anglin et al., 2013, Ju et al., 2013) among other conditions. Furthermore, a therapeutic effect of vitamin D supplementation on these depressive symptoms has been observed as well (Shaffer et al., 2014; Spedding, 2014).

Cress (2014), examined university students during winter in Tennessee and reported that 69.5% of students have suboptimal levels (< 30 ng/ml) including 32% having less than 20 ng/ml which is considered insufficient. Vitamin D insufficiency is most prevalent during the winter months due to practically negligible UVB rays available for vitamin D synthesis during November to March in latitudes above 45° N (Webb et al., 1988). With Menomonie, WI located at 44.8 °N, it places an increased importance on consuming adequate vitamin D, however recent evidence shows that university students are consuming under the EAR (10 μg) for vitamin D (Cress, 2014; Ouellette et al., 2012; Forney et al., 2014; Lacey et al., 2004).

Intervention studies examining vitamin D knowledge and intake have shown increased intake in response to personalized feedback, however, but the efficacy of educational interventions is less clear (Goodman, 2015). This chapter will review recent research and literature on vitamin D, depression, and lastly the relationship between the two.

Vitamin D

Vitamin D is a fat-soluble, steroid hormone normally produced in the skin upon exposure to certain wavelengths of ultraviolet (UV) light. Functions of vitamin D in the body include;
modulation of cell growth, promoting calcium absorption, neuromuscular and immune function, and reduction of inflammation. Serum vitamin D concentrations represent the amount of vitamin D obtained by either ultraviolet-B rays or by consuming vitamin D. Vitamin D₃ is the form of vitamin D that is synthesized in the skin from sterol 7-dehydrocholesterol (DHC) by a process involving ultraviolet-B (UVB) light (Chen, Lu, & Holick, 2010). The process of synthesizing vitamin D₃ from UVB light begins when UVB radiation (280-315 nm wavelength) passes through the epidermis and dermis levels of human skin, which contain 7-DHC. The 7-DHC absorbs the UVB photons and triggers the conversion from UVB to previtamin D₃ in a process called photoisomerization. Previtamin D₃ then undergoes thermal dependent isomerization, which results in the formation of the vitamin D₃ molecule. This process plateaus during long sun exposure, and about 10-15% of the original 7-DHC content as previtamin D₃ is photoisomerized into lumisterol and tachysterol alternatively, both biologically inactive molecules. Along with endogenous production, vitamin D can also be obtained by consuming dietary vitamin D from food sources or supplementation in the form of vitamin D₂ (ergocalciferol) or D₃ (cholecalciferol). Vitamin D₂ is less commonly consumed as it is found primarily in fungi and UVB irradiated mushrooms, while vitamin D₃ is obtained from animal sources and is more common in supplements (Byrdwell et al., 2008; Ovesen, Brot, & Jakobsen, 2003).

Following either dietary consumption or synthesis in the skin, vitamin D₃ (cholecalciferol) and vitamin D₂ (ergocalciferol) are both transported to circulation by chylomicrons, before attaching to a vitamin D-binding protein (DBP) (Christakos, Ajibade, Dhawan, Fechner, & Mady, 2010). The DBP, or less frequently albumin, transports both molecules to the liver to be converted from the biologically inactive precursors of vitamin D to 25-hydroxyvitamin D (calcidiol; calcifediol) by hydroxylation using the enzyme CYP2R1
Calcidiol is the most accurate indicator of vitamin D status as it represents the sum of 25-hydroxyvitamin D$_2$ and 25-hydroxyvitamin D$_3$. After transport to the kidneys, 25-hydroxyvitamin D-1-α-hydroxylase enzyme (CYP27B1) initiates the second hydroxylation of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D (calcitriol) (Christakos et al., 2010). This reaction also has been shown to occur in extrarenal locations such as skin, parathyroid gland, colon, prostate, breast, immune and bone cells (Bikle, 2014). Calcitriol is the metabolically active metabolite of vitamin D, and most physiological effects of vitamin D are due to the activity of calcitriol (Holick, 2003).

**Physiological effects of vitamin D.** The physiological actions of vitamin D are due mainly to the activity of the vitamin D receptor (VDR), a nuclear transcription factor (Sutton & MacDonald, 2003). This receptor functions by binding to 1,25-dihydroxyvitamin D, as well as retinoic acid X receptor (RXR) before entering the nucleus of a cell (Macdonald et al., 1993). This VDR/RXR network, in the presence of calcitriol, binds vitamin D response elements (VDRE), which are small sequences of DNA (Haussler et al., 1998). Vitamin D response elements then instigate a cascade of reactions that modulate the transcription of specific genes (Macdonald et al., 1993). There are estimated to be about 100 to 1,250 genes that are either directly or indirectly regulated by the VDR and 1,25-dihydroxyvitamin D activation (Grober, Spitz, Reichratch, Kisters, & Holick, 2013). The primary functions of vitamin D via the VDR include calcium balance, phosphorus balance, cell differentiation, immunity, and insulin secretion (Haussler et al., 1998).

**Skeletal system benefits.** Vitamin D is most commonly known for its beneficial effect on the skeletal system due to its involvement in the maintenance of serum calcium levels and its essential assistance in the utilization of calcium in the body (Holick, 2004). Optimal calcium
serum concentrations are required for not only bone mineral density but also bone growth and nervous system functioning. If calcium concentrations decrease below normal values, the parathyroid glands respond by secreting parathyroid hormone (PTH) (Parfitt et al., 1982). The increase in serum PTH then stimulates the activity of the renal enzyme 25-hydroxyvitamin D₃-1α-hydroxylase, which increases the production of 1,25-dihydroxyvitamin D (Turner, Anderson, & Morris, 2012). This increase of 1,25-dihydroxyvitamin D activates the VDR to increase serum calcium levels through three distinct mechanisms: increasing the intestinal absorption of dietary calcium (Holick, 2004), increasing the amount of calcium reabsorbed by the kidneys (Bikle, 2014), and lastly mobilizing calcium from the bone (Volmer et al., 2015). Vitamin D assists in the homeostasis of not only serum calcium, but serum phosphorus as well. The mechanism of action is similar to that of calcium balance, and utilizes the calcitropic hormones PTH as well as 1,25-dihydroxyvitamin D. 1,25-dihydroxyvitamin D increases intestinal absorption of phosphorus via the stimulation of a sodium-phosphate cotransporter located in the small intestine (DeLuca, 1979).

Vitamin D has a well-established association with skeletal health and for this reason, the Food and Drug Administration (FDA) has published the health claim that vitamin D, along with calcium, a well-balanced diet, and physical activity, may reduce the risk of osteoporosis. This is the only published health claim from the FDA for vitamin D, due to the amount of evidence linking sufficient vitamin D status to lowered risk of osteoporosis (Food Labeling, 2017). However, mounting evidence supports the idea that vitamin D supplementation, without vitamin D deficiency or specific risk factors for vitamin D deficiency, does not prove to be beneficial in reducing risk of fractures or increasing bone mineral density (Avenell, Gillespie, Gillespie, & O’Connell, 2005; Abrahamsen et al., 2010; Reid, Bolland, & Grey, 2014). Individuals with
specific risk factors, such as dark skin pigmentation or low sunlight exposure, however, are shown to benefit from supplementation (Reid, Bolland, & Grey, 2014).

**Non-skeletal system physiological effects.** Non-skeletal system physiological effects of vitamin D due to expression of the VDR include regulation of cell differentiation, immune system modulation, and endocrine functions (Haussler et al., 1998). Cell differentiation results in the specialization of cells to perform a specific function. This process also tends to decrease cell proliferation, or the rapid division of cells, which is a characteristic of cancer cells. 1,25-dihydroxyvitamin D stimulates the differentiation of cells, and therefore limits the proliferation of cells through the VDR (Nibbelink, Tishkoff, Hershey, Rahman, & Simpson, 2007).

The role of vitamin D in the immune system is moderated by the VDR expression on immune cells including regulatory T- cells, dendritic cells, and macrophages (Wagner & Greer, 2008). During special circumstances, such as during an antimicrobial response, immune cells express the enzyme 25-hydroxyvitamin D\(_3\)-1\(\alpha\)-hydroxylase which then produces 1,25-dihydroxyvitamin D to regulate the local immune response (Edfeldt et al., 2010; Smolders, Thewissen, & Damoiseaux, 2011). Vitamin D receptors are also expressed by insulin-secreting cells of the pancreas, which suggest that 1,25-dihydroxyvitamin D may have an effect on insulin secretion during times of increased demand (Bourlon, Billaudel, & Faure-Dussert, 1999; Zeitz et al., 2003). An additional endocrine function includes the down-regulation of renin production in the kidneys via the renin-angiotensin system (Li et al., 2003).

**Etiological factors of vitamin D deficiency.** The vitamin D status of humans is determined by the amount of sun exposure they obtain and the amount of vitamin D consumed in the diet from food or supplements. Deficiency of vitamin D can occur due to environmental factors, behavioral factors, dietary choices, vitamin D knowledge, and biological factors.
Environmental factors. Environmental factors, such as seasonal changes, latitude, pollution, or presence of clouds influence the UVB radiation available for vitamin D synthesis (Webb et al., 1988). Godar, Pope, Grant, and Holick (2011) examined children under the age of 19 and concluded that only during the summer were individuals able to obtain the minimum recommended daily dose of vitamin D of 600 IU. Additionally, several studies have concluded that season was a predictive factor of low vitamin D status (Al Anouti et al., 2011; Brustad, Alsaker, Engelsen, Aksnes, & Lund, 2003; Eggemoen, Knutsen, Dalen, & Jenum, 2013; Gordon, DePeter, Feldman, Grace, & Emans, 2004; Holvik, Meyer, Haug, & Brunvand, 2005; Rejnmark et al., 2004). The first documented correlation between season and decreased vitamin D was discovered by Kassowitz in 1897. Several changes occur during the winter months, including increased clothing cover, less time outdoors, and the zenith angle of sunlight increases in the fall to winter months. This causes a steep decrease in the ability of solar UVB radiation to reach the earth’s surface (Holick, 1995). Moreover, Chen et al. (2010) concluded that vitamin D synthesis from UVB was negligible from the months of December through February in Boston, Massachusetts (42° N). Los Angeles, California, however, experienced vitamin D synthesis all year as the latitude is 34° N and the zenith angle of sunlight is lower compared to higher latitudes. Menomonie, Wisconsin is located at 41° N, putting its residents at high risk of vitamin D deficiency during the winter months. In areas with increased air pollution there is an increase in ozone concentration. The ozone absorbs the UVB wavelengths and therefore decreasing the UVB photons available for synthesis to precholecalciferol (Holick, 1995)

Behavioral factors. Behavioral factors such as clothing style, and other sun protectant use, also can increase one's risk of vitamin D deficiency. In a study of Middle Eastern women who wore traditional headscarves or covered all skin for religious and/or cultural reasons, 96%
were found to have serum vitamin D concentrations of less than 20 ng/ml, or a classification of vitamin D insufficiency by the Institute of Medicine, Food and Nutrition Board (2010). Furthermore, 60% were found to have vitamin D deficiency (<12 ng/ml) (Nichols et al., 2012). Clothing style inhibits UVB radiation in the skin in similar methods that sun safety habits do, by inhibiting vitamin D synthesis by blocking the skin's access to UVB (Matsuoka et al., 1992). Specifically, sunscreen with a sun protection factor (SPF) of 10 or higher reduces UVB radiation by ≥ 90%, inhibiting the skin's ability to synthesize vitamin D (Balk, 2011).

**Dietary factors.** Dietary predictors of vitamin D deficiency include limited consumption of fortified foods (Brock et al., 2007; Reed et al., 2007), low fatty fish consumption (Granlund, Ramnemark, Andersson, Lindkvist, & Norberg, 2016; van der Meer et al., 2008), and no use of vitamin D supplement or cod liver oil (Åkeson, Lind, Hernell, Silfverdal, & Öhlund, 2016; Halliday et al., 2011; Holvik et al., 2005; Lamberg-Allardt, Outila, Kärkkäinen, Rita, & Valsta, 2001; Shiraishi Haruna, Matsuzaki, & Murayama, 2014; van der Meer et al., 2008). These predictor variables may be used as indicators of possible lowered serum vitamin D concentrations and lowered vitamin D intake. Furthermore, lowered vitamin D intake is predictive of vitamin D deficiency in the general population across the globe (Andersson, Björk, Kristiansson, & Johansson, 2013; Cress, 2014; Brustad et al., 2003; Eggemoen et al., 2013; Gozdzik et al., 2008; Lamberg-Allardt et al., 2001; Rejnmark et al., 2004; Shiraishi et al., 2014).

**Biological factors.** Along with environmental and cultural factors, biological factors have been shown to be risk factors for a deficiency of vitamin D. These biological factors include skin pigmentation, genetics, age, kidney health, fat malabsorption conditions, obesity, and magnesium deficiency (National Institutes of Health, 2011). Having a darker skin pigmentation can put an individual at a higher risk of vitamin D deficiency, (Chen et al., 2007) and is a strong predictive
variable for vitamin D deficiency (Björk et al., 2003; Eggemoen et al., 2016; Gozdzik et al., 2008; Gordon et al., 2004; Rejnmark et al., 2004). Melanin absorbs solar radiation from the ranges of 290 to 700 nm, which includes the wavelength involved in previtamin D$_3$ (Clemens, Henderson, Adams, & Holick, 1982). Furthermore, it has been shown that while sun exposure caused Caucasian participants’ serum vitamin D concentration to increase significantly, it had no effect on black participants (Chen et al., 2010). Genetics can affect vitamin D status, as the concentration of vitamin D-binding protein that is circulating in the body varies based on genetic predisposition (Wang et al., 2010). If an individual is born without sufficient DBP to bind circulating 25-hydroxyvitamin D, the body is not able to utilize circulating vitamin D as well as an individual who has normal concentrations of DBP (Wang et al., 2010). Older age has been shown to be a risk factor for vitamin D deficiency due to lifestyle factors (sun protection, limited sun exposure), however, a number of studies have also found that younger age was a risk factor for decreased vitamin D (Qatatsheh, Tayyem, Al-Shami, Al-Holy, & Al-Rethaia, 2015; Holvik et al., 2005; Khan, Iqbal, Naureen, Dar, & Ahmed, 2012; Lamberg-Allardt et al., 2001; Rejnmark et al., 2004). This contradiction hints that there may be less of risk factor due to age as researchers once thought, and possibly the relationship is due to behavioral factors associated with the age rather than age itself. Lowered kidney health, more specifically chronic kidney disease, causes a reduction in the synthesis of 1,25-dihydroxyvitamin D as well as an increase of 25-hydroxyvitamin D present in the urine (Doorenbos, Van Den Born, Navis, & De Borst, 2009). Conditions that disrupt fat absorption including cystic fibrosis, liver diseases, and inflammatory bowel disease, can interfere with the body’s ability to absorb dietary fat and therefore limited absorption of the fat-soluble vitamin D from dietary sources (Jeppesen, Christensen, Høy, & Mortensen, 1997).
**Consequences of deficiency.** During a state of vitamin D deficiency, calcium absorption is decreased, disrupting balance within the body (Sahota et al., 2004). To restore calcium homeostasis the parathyroid glands, produce an increased amount of PTH and the mobilize calcium from the bones (Sahota et al., 2004). There are both established and hypothesized conditions that are consequences of the vitamin D deficiency, and disruption of calcium homeostasis, in both the skeletal and non-skeletal system.

**Skeletal consequences.** In times of vitamin D deficiency, the body’s ability to absorb calcium is decreased and leads to PTH production and the mobilization of calcium from the bones (Heaney, 2003). This is a primary mechanism for the development of several bone diseases in times of vitamin D deficiency. Rickets was an epidemic in the 19th century due to the lack of intake of vitamin D and sun exposure causing growth retardation, muscle weakness, hypocalcemia, seizures and skeletal deformities (Holick, 2006). Rickets has steadily declined since the advent of fortification of food products, specifically fluid milk products and cereals (Calvo, Whiting & Barton, 2005). However, rickets is still being reported throughout the world due to dietary factors, and sun protection habits (Goldacre, Hall, & Yeates, 2014; Wagner & Greer, 2008). While rickets occurs only in children and young adolescents, vitamin D deficiency in adults may result in a condition known as osteomalacia or softening of the bone. As stated previously, vitamin D deficiency leads to the mobilization of calcium out of the bones. If the deficiency is left untreated, it can lead to a quantum decrease in bone volume and osteoid accumulation, both characteristics of osteomalacia (McKenna, Freaney, Casey, Towers, & Muldowney, 1983). Osteomalacia is a strong risk factor for the development of osteoporosis (Jones & Hansen, 2009).
Both rickets and osteomalacia can be successfully treated with vitamin D supplementation (Drezner, Lyles, Haussler, & Harrelson, 1980). Other skeletal conditions associated with vitamin D deficiency, however, have not been responsive to vitamin D treatment. Osteoporosis is a multifactorial condition, which vitamin D has been shown to be an etiological factor for. While multiple case-control studies have shown that individuals who had low serum vitamin D levels in comparison to sufficient individuals experienced more osteoporotic fractures (Lips & van Schoor, 2011; Lips et al., 2006; Torbergsen et al., 2015), intervention trials of vitamin D supplementation have been less conclusive. A cohort study that examined 72,000 postmenopausal women for 18 years found that those who consumed 3.5 µg of vitamin D per day or less had a 37% increased risk of fracture in comparison to those who consumed at least 15 µg/day (Feskanich, Willett, & Colditz, 2003). In comparison, the Randomized Evaluation of Calcium Or vitamin D (RECORD) study concluded that neither 20 µg of vitamin D supplementation alone nor an accompanying calcium supplement (1,000 mg), was preventative in risk of fractures (RECORD Trial Group, 2005). Three large meta-analyses of randomized controlled trials of vitamin D supplementation and osteoporotic risk all concluded that vitamin D supplementation or vitamin D along with calcium was effective in significantly reducing risk of osteoporotic fractures in older men and postmenopausal women (Avenelle, Mak, & O’Connell, 2014; Boonen, Lips, Bouillon, Bischoff-Ferrari, Vanderschueren, & Haentjens, 2007; Chung, Lee, Terasawa, Lau, & Trikalinos, 2011). The current dietary recommendations of 20 µg per day, along with sufficient calcium, therefore may be helpful in reducing one’s risk for osteoporosis and loss of bone mineral density (Institute of Medicine, Food and Nutrition Board, 2010).
**Non-skeletal consequences.** The only established non-skeletal consequence of vitamin D deficiency is muscle pain and weakness. This was most prominently displayed in a cross-sectional study conducted in Minnesota which concluded that 93% of individuals who were referred to a clinic for non-specific muscle pain and weakness had serum vitamin D concentrations of less than 20 ng/ml, which is clinically deficient (Plotnikoff & Quigley, 2003). This pain and weakness may also greatly contribute to falls and risk of bone fractures, specifically in the older populations. Along with muscle weakness and pain, there are documented relationships with other non-skeletal conditions including cancer, autoimmune disorders, cardiovascular disease, neurodegenerative conditions, and mood disorders (Anglin et al., 2013; Annweiler et al., 2010; Cantorna & Mahon, 2004; Giovannucci et al., 2006; Grandi, Breitling, & Brenner, 2010). However, these associations are primarily correlational in nature, and causation and intervention trials are contradictory.

*Vitamin D deficiency and cancer.* Due to the role of vitamin D in cell proliferation and differentiation, research has been conducted to explore the relationship between vitamin D status and risk of cancer. Grant (2002) conducted an ecological study and found that there is a higher risk of cancer in those who live in higher latitudes, and concluded that the geographic variation in cancer mortality rates can be credited to the variance in exposure to UVB radiation and vitamin D synthesis. Several observational, cross-sectional, prospective, retrospective and epidemiological studies have concluded that there is a significant inverse relationship between vitamin D, specifically serum 25(OH)D, and the prevalence of total cancer (Giovannucci et al., 2006), Non-Hodgkin’s Lymphoma (Drake et al., 2010), colorectal cancer (Feskanich et al., 2004; Freedman, Looker, Chang, & Graubard, 2007; Garland et al., 1989; Wu et al., 2007), pancreatic cancer (Stolzenberg-Solomon et al., 2006; Stolzenberg-Solomon et al., 2009), prostate cancer
A meta-analysis of randomized controlled trials examining vitamin D supplementation and cancer risk concluded that supplementation of vitamin D, with or without calcium, did not reduce cancer risk in community-dwelling individuals (Bolland, Grey, Gamble, & Reid, 2014). Additionally, a third review has concluded that vitamin D supplementation trials show no significant reduction in the risk of colorectal, breast, or skin cancer (Autier & Gandini, 2007). Interestingly, Keum and Giovannucci (2014) in an additional meta-analysis concluded that while vitamin D supplementation had no effect on total cancer incidence, it significantly reduced total cancer mortality risk. It can be concluded that while there may be an inverse relationship between serum vitamin D and prevalence of cancer, the cause may not be due to the vitamin D, as concluded by randomized controlled trials.

**Vitamin D and autoimmune disorders.** Similar to cancer risk, autoimmune disorder prevalence was first associated with vitamin D status after ecological and epidemiological studies concluded that risk of certain autoimmune disorders was positively correlated with higher latitude, and more specifically, lowered vitamin D synthesis (Cantorna & Mahon, 2004). This relationship to lowered serum vitamin D concentrations has been well researched in meta-analysis, specifically for type 1 diabetes mellitus (Antico, Tampoia, Tozzoli, & Bizzaro, 2012; Ponsonby, McMichael, & van der Mei, 2002; Mohr, Garland, Gorham, & Garland, 2008), multiple sclerosis (MS) (Antico, et al., 2012; Ponsonby et al., 2002), rheumatoid arthritis (RA) (Vieira et al., 2010) and inflammatory bowel disease (Antico, et al., 2012; Peyrin-Biroulet, Ouussalah, & Bigard, 2009). Recent research has concluded that a child’s risk of developing type
1 diabetes is lower if born to individuals who had high concentrations of serum vitamin D. For example, Sørensen et al. (2012) found that the odds ratio of type 1 diabetes mellitus was significantly higher in the lowest serum concentration quartile (22 ng/ml), compared to that of the highest quartile (> 35 ng/ml). Moreover, birth-cohort and case-control studies concluded that there was a significant decrease in frequency of type 1 diabetes mellitus in children born to individuals who consumed a supplement containing vitamin D (Hyppönen, Läära, Reunanen, Järvelin, & Virtanen, 2001; Stene, Ulriksen, Magnus, & Joner, 2000).

Observational studies concluded that increases in serum vitamin D concentration were associated with a reduction of relapse rate of MS (Pierrot-Deseilligny, Rivaud-Pechoux, Clerson, de Paz, & Souberbielle, 2012; Smolders, Menheere, Kessels, Damoiseaux, & Hupperts, 2008), as well as decreased incidence of RA (Merlino et al., 2004). However, these findings are merely correlational and therefore the effect seen may not be from vitamin D alone, but quite possibly lies in the fact that individuals who have higher serum vitamin D concentrations, have better overall health. Irritable bowel disease, however, has been examined in a randomized controlled trial. Vitamin D is most commonly correlated with inflammatory bowel disease due to high rates of deficiency caused by malabsorption of fat, and therefore the fat-soluble vitamins (Kuroki et al., 1993). Jørgensen et al. (2010), found that individuals who were randomized to receive 30 µg of vitamin D per day not only increased their serum levels significantly but also lowered their risk of relapse compared to the placebo group (13% vs. 29%).

**Vitamin D and cardiovascular disease.** There is a large pool of data supporting the inverse correlation between lowered serum vitamin D status and cardiovascular risk (Grandi et al., 2010; Judd, & Tangpricha, 2009). Experimental studies conducted examining vitamin D supplementation and cardiovascular risk are few and less conclusive. For example, Hsia et al.
(2007), concluded that supplementing postmenopausal women with 500 mg calcium plus 5 µg of vitamin D was not successful in either decreasing or increasing cardiovascular events. Moreover, Gepner et al. (2012) found that a high dose supplementation of vitamin D of 62.5 µg/day was not successful in significantly decreasing brachial artery flow-mediated vasodilation, carotid-femoral pulse wave velocity, or aortic augmentation index, and therefore rejected the hypothesis that vitamin D supplementation reduces cardiovascular risk. Most significantly, a meta-analysis of eight randomized controlled trials found that there was no statistically significant reduction in cardiovascular risk with moderate (25 µg) vitamin D supplementation (Wang, Manson, Song, & Sesso, 2010).

**Vitamin D and neurodegenerative diseases.** Research in animal models have suggested that vitamin D deficiency and/or the disruption of the vitamin D-VDR pathway in the cerebral regions of the cortex and hippocampus may have a role in the degradation of neurons and cognitive function (Gezen-Ak, Yilmazer, & Dursun, 2014). In observational studies, decreased serum vitamin D was correlated with cognitive impairment (Annweiler et al., 2010; Annweiler et al., 2012; Hooshmand et al., 2014). Several meta-analyses examining observational and cross-sectional studies made a similar conclusion, that hypovitaminosis D is correlated with worse performance on cognitive function tests compared to those with adequate vitamin D (Balion et al., 2012; Annweiler et al., 2013; van der Schaft et al., 2013). Intervention trials with vitamin D supplementation and its effect on cognitive function provide conflicting results. Annweiler et al. (2012), found that supplementation of either 20 µg per day or 2,500 µg per month, for 16 months was successful in significantly improving individuals’ scores on the Mini-Mental State Examination score, the Cognitive Assessment Battery score, and Frontal Assessment Battery score, as well as increasing their serum vitamin D levels. This study however, was not a
randomized controlled trial, and therefore is merely exploratory and requires further confirmation. Conversely, the Women’s Health Initiative explored the effect of calcium and vitamin D supplementation on cognitive function in elderly women and concluded that the treatment group (1000 mg calcium plus 10 µg vitamin D) compared to the placebo did not differ in cognitive impairment (Rossom et al., 2012). The lower dose of supplementation, 10 µg of vitamin D which is lower than the RDA, could be a possible reasoning for the lack of effects seen. Overall, this is a topic that requires further exploration using randomized controlled trials.

**Vitamin D and mood disorders.** Active vitamin D works through two vitamin D response elements on the *TPH1* and *TPH2* genes to transform tryptophan into serotonin in tissues, including the brain (Patrick & Ames, 2014). This is the hypothesized etiological link of how decreased serum vitamin D may lead to mood disorders. Low serotonin and serotonin metabolites have been found to be low in autopsy studies as well as cerebral spinal fluid in depressed individuals (DeVane & Sallee, 1996). Moreover, vitamin D deficiency in individuals who have predispositions to decreased serotonin production may result in an increase in the severity of mental illness (Patrick, & Ames, 2015). Insufficient serum vitamin D has been shown to be correlated with increased risk of bipolar disorder, anxiety, depression, and seasonal affective disorder (Anglin et al., 2013; Aydin et al., 2015; Hoogendijk et al., 2008; Kiraly, Kiraly, Hawe, & Makhani, 2006; Patrick & Ames, 2014). Depression specifically, has been most commonly explored with multiple meta-analyses concluding both a significant relationship between low serum vitamin D and depression (Anglin et al., 2013; Ju et al., 2013), as well as a significant improvement of in depressive symptoms with vitamin D supplementation in randomized controlled trials (Spedding, 2014; Shaffer et al., 2014). These findings are discussed below.
**Vitamin D deficiency and insufficiency prevalence.** Much of the research completed in the United States on vitamin D insufficiency and deficiency has focused on the older adult population due to the link between low serum vitamin D concentrations and fractures, bone density, risk of falling, slower reaction time, and muscle weakness. However, according to the National Health and Nutrition Examination Survey (NHANES), 36% of males and 37% of females aged 19-30 have a serum vitamin D concentration less than 30 ng/ml, while in adults over the age of 70 is 24% and 27%, for males and females respectively (Looker et al., 2011). Of note, NHANES testing done in the northern area of the United States is performed during the summer months, when serum vitamin D levels are likely to be at their highest, so these numbers may be an underestimate of true insufficiency.

A more accurate representation of vitamin D status during the winter months is given in a study conducted in Boston, Massachusetts that examined serum vitamin D concentrations in healthy young adults aged 18-29 (Tangpricha, Pearce, Chen, & Holick, 2002). Serum concentrations were measured at the end of the winter and summer months, and the young adults were compared to individuals aged 30-39, 40-49, and over 50. Tangpricha et al. (2002) found that 36% \((n = 69)\) of the young adult participants had vitamin D insufficiency \((\leq 20 \text{ ng/ml})\) at the end of winter. The younger participants also had more seasonal variation than any other age group, and had 20% lower serum 25(OH)D concentrations than the 50 and over age group (Tangpricha et al., 2002). Whiting, Langlois, Vatanparast, and Greene-Finestone (2011) conducted a study in Canada also analyzing vitamin D status during both the summer and winter months in a variety of age ranges, including ages 20-39. The authors reported that the mean serum concentration of this age range in the winter months was 22.3 ng/ml, compared to 26.4 ng/ml in the summer months (Whiting et al., 2011).
Serum vitamin D in college students. Despite many studies examining the prevalence of vitamin D deficiency and insufficiency in the general population, less is known about the prevalence in college students. Due to skin pigmentation and sun protectant clothing traditions, vitamin D insufficiency prevalence in university students in the Middle East is well documented. Faghih et al. (2014) for example, found that 44% of the female university students in Iran had vitamin D deficiency (5-10 ng/ml), compared to only 1.6% of male students. Faghih et al. (2014) also reported that 49.5% of the male students had vitamin D insufficiency (10-20 ng/ml), compared to 51.2% of female students. There was also a significant difference between the means of the male and female population ($M = 19.75$, $SD = 5.16$ ng/ml and $M = 11$, $SD = 4.15$ ng/ml, respectively). Al-Elq (2012), also found that within a Saudi Arabian sample the mean of male serum vitamin D concentrations was significantly higher than female participants ($M = 10.75$, $SD = 5.05$ ng/ml vs. $M = 6.42$, $SD = 3.32$ ng/ml, respectively). Even though significantly higher, both concentrations are insufficient for optimal health. Al Anouti et al., reported that the mean vitamin D concentrations of participants in an university at Abu Dhabi, United Arab Emirates were ($M = 10.9$, $SD = 6.3$ ng/ml) for males and ($M = 9.7$, $SD = 6$ ng/ml) for female participants (2011). The female subjects in this study also scored higher on a sun avoidance inventory, showing a relationship between decreased direct sunlight and decreased vitamin D concentrations. Other studies in the Middle East have focused on finding the number of female students with vitamin D deficiency. Sharif and Rizk (2011) reported that 50.7% of female participants in Qatar ranked as severely deficient (<10 ng/ml). Qatatsheh et al. (2015) studied female students at a University in Jordan and found that 31.2% had serum vitamin D concentrations below 10 ng/ml.
With an increased recognition of the importance of vitamin D for extra-skeletal functions has come studies reporting vitamin D concentrations in college students in North America. In the United States, for example, Cress (2014) examined university students in Tennessee during the winter and reported that 32% had insufficient serum vitamin D (< 20 ng/ml). This research also reported a surprising result of females having a significantly higher mean serum 25(OH)D concentration \( (M = 28.77, SD = 10.76) \) than the male students \( (M = 21.76, SD = 8.2) \). These data do not support those reported from studies conducted in the Middle East, however, factors such as time spent outside, or use of tanning beds, were not evaluated for and could account for differences between gender. Additionally, Forney et al. (2014) analyzed the mean serum vitamin D in students at a university in Louisiana and found that the mean serum vitamin D for all participants was 34.88, \( SD = 1.9 \) ng/ml with females having only a slightly higher level than male subjects \( (M = 36.73, SD = 3.2 \) ng/ml vs. \( M = 33.02, SD = 2.1 \) ng/ml, respectively), which was not statistically significant. Interestingly, the subjects were all healthy and active college students whose blood samples were tested during the months of July and September when stores are expected to be at their highest due to sun exposure. The subjects also lived in the Southern US, where latitude allows for more months of cutaneous production of vitamin D than in more northern regions. A more applicable study to the current subject population was completed by Gozdzik et al. (2008) in Canada (43.5° N) and examined vitamin D concentrations during the winter months in young, healthy adults (age 18-30, \( M = 21 \) years of age). The authors reported that not only was the mean serum vitamin D concentration of the participants insufficient at 15.79 ng/ml, but also that a total of 74% of subjects had concentrations of less than 20 ng/ml. Although the sample was not university students, it displays the impact that winter in a northern latitude can have on serum vitamin D concentration in healthy, young adults.
**Vitamin D intake.** Another way to examine the vitamin D status of individuals is through vitamin D intake, either by dietary sources or supplementation. Bailey et al. (2010) examined vitamin D intake using NHANES data and reported the mean vitamin D intake from diet alone in individuals aged 19-30 to be 5.1 µg (SD = 0.3) for males and 3.6 µg (SD = 0.3) for females.

**Vitamin D intake in college students.** Several studies of college students completed in the United States concluded that the range of mean intakes varies from 3.6 to 8.2 µg/day, all of which fall well below the RDA for vitamin D (Cress, 2014; Ouellette et al., 2012; Forney et al., 2014; Lacey et al., 2004). Ouelette et al. (2012) reported that male subjects had a mean intake of 7.4 (SD = 4.8) µg per day, while female individuals consumed significantly less at 4.0 µg per day (SD = 3.9). Furthermore, 96% of the female participants were consuming less than the RDA of vitamin D which was significantly higher than the male students (76%) (Ouelette et al., 2012). Interestingly, the participant group was heavily made up of students in a nutrition class and an assumption would be that they have adequate knowledge about vitamin D. An additional population that one may predict to have better nutritional status is Division I college athletes. However, Halliday et al. (2011) conducted a study on university athletes and found that mean dietary consumption of vitamin D was 6.1 (SD = 4.5) µg/day, which is also under the RDA for vitamin D requirements. The authors also noted that only 36% of participants reported regularly consuming a vitamin D-containing supplement in the winter months, putting the majority of Wyoming university athletes at risk of deficiency. A similar result was found in a more general college population in a study conducted by Hilliard, Fuhrmann, and Brunt (2015), which found that 97% of their participants did not consume the RDA for vitamin D in their usual diet in college dining halls. Along with serum vitamin D status, Forney et al. (2014) examined the
relationship between mean vitamin D intake and serum levels. The overall mean intake for all the participants was 3.99 ($SD = 0.6$) µg of vitamin D per day with males consuming significantly more than females ($M = 5.7, SD = 1.0$ vs. $M = 2.2, SD = 0.4$, respectively). Cress (2014) reported that only 8% of university students consumed over the EAR for vitamin D ($M = 8.2, SD = 10.6$), and only 19% of students consumed a vitamin D containing supplement. Lacey et al. (2004) reported that 71% of participants consumed under 5 µg of vitamin D with a significantly higher percentage of women consuming under 5 µg than male participants (78% vs 53%, respectively). In this study, the reported total mean of vitamin D consumed by participants was 3.6 ($SD = 3.0$) with no significant differences between female and male participants (Lacey et al., 2004). A visual display of dietary intake of college students research is shown in table 4.

Table 4

*Total Vitamin D Intake in College Students*

<table>
<thead>
<tr>
<th>Authors, year</th>
<th>% below RDA</th>
<th>$M$ Vitamin D Intake, µg/day ($SD$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cress, 2014</td>
<td>92%*</td>
<td>8.2 (10.6)</td>
</tr>
<tr>
<td>Ouellette et al., 2012</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>76%*</td>
<td>7.4 (4.8)***</td>
</tr>
<tr>
<td>Females</td>
<td>96%*</td>
<td>4.0 (3.9)</td>
</tr>
<tr>
<td>Forney et al., 2014</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>97%</td>
<td>4.0 (0.6)</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td>5.7 (1.0)</td>
</tr>
<tr>
<td>Hilliard, Fuhrmann, Brunt, 2015</td>
<td>97%</td>
<td></td>
</tr>
<tr>
<td>Lacey et al., 2004</td>
<td>71%**</td>
<td>3.6 (3.0)***</td>
</tr>
</tbody>
</table>

Note. * Percentage of students consuming under the EAR (400IU), ** Percentage of students consuming under 5 µg, *** Indicates significant difference compared to female
Knowledge and perceptions of vitamin D. A potential contributor to vitamin D insufficiency and deficiency is lack of vitamin D-related knowledge. Vu et al. (2010) explored knowledge and attitudes related to vitamin D, and the relationship to the sun protection habits of office-based workers in Australia. The authors concluded that knowledge around sources and benefits of vitamin D was low and that individuals who agreed with the statement that sun protection might cause vitamin D deficiency were less likely to be regular sun protectant users. This risk was explored by Magulak et al. (2012), who examined vitamin D and calcium knowledge in university students eating in on-campus dining halls (n = 132). The authors found that while 76% of students could identify three or more good dietary sources of calcium, only 22% of students could do the same for vitamin D. The authors concluded that more education was necessary to ensure that students would consume enough vitamin D and calcium to ensure bone health.

Boland et al. (2014), examined the vitamin D-related knowledge of college students and found that students were able to correctly answer only 29% of questions about vitamin D sources, factors affecting status, and health effects of vitamin D. They stated that this is a public health concern as peak bone mass (Khairi & Johnston, 1978) is developed between the ages of 18 to 25, and stressed the need for more education on vitamin D. This, however, is an assumption that with increased knowledge there will be a behavior change. Kolodinsky, Harvey-Berino, Berlin, Johnson, and Reynolds (2007) did find that college students with higher knowledge about dietary guidelines was associated with an increased likelihood of meeting such guidelines for consumption of fruit, dairy, protein, and whole grains. This result, however, was of food groups and not specific nutrients and therefore not entirely applicable. Ha, Caine-Bish, Holloman, and Lowry-Gordon (2009) reported a significant increase in dairy consumption after
completion of a 15-week basic nutrition educational intervention. This type of intervention may not be realistic for some communities and populations, due to financial and personnel requirements, and while dairy servings were increased there was no data on whether students were meeting the recommended amounts of vitamin D. Goodman (2015) studied a mobile application for participants’ phones that calculated vitamin D intake based on self-reported food records and found that while there was an increase in both vitamin D knowledge and intake, there was no statistical difference in serum vitamin D concentrations of the intervention group versus the control. Other studies have also found significant changes in other health behaviors using mobile applications for personalized feedback (Hebden, Cook, van der Ploeg, & Allman-Farinelli, 2012; Kratzke & Cox, 2012).

**Depression**

In 2014, the National Survey on Drug Use and Health reported that 1 in 5 adults aged 18 and over (18.1%) had a mental illness in the past year (Center for Behavioral Health Statistics and Quality, 2015). This number represents the number of any mental illness, or AMI, which is defined as any mental, behavioral or emotional disorder that is either diagnosable currently or within the past year. The most common neuropsychiatric condition in high-income countries is depression, per the World Health Organization. Depression is the number one disabling condition for individuals aged 0 to 59, with 15.8 million individuals with a moderate or severe disability (World Health Organization, 2008). According to the National Institute of Mental Health, there are several different types of depression; major depression, persistent depressive disorder, psychotic depression, postpartum depression, seasonal affective disorder, and bipolar disorder. Major depression is one of the most common mental illnesses with around 15.7 million
adults in the United States experiencing at least one major depressive episode (MDE) in the last year (Center for Behavioral Health Statistics and Quality, 2015).

**Changes in prevalence of depression.** Debate exists around the number of individuals affected by depression, and whether it is increasing. There are three study design types used to examine this question: time-lag, cross-sectional, and longitudinal (Twenge, 2015). A flaw in utilizing the longitudinal design to look for an increase in depression is that longitudinal studies hold the cohort constant and examine changes in age and time. This leaves the cultural differences between cohorts as a cofounder. In a cross-sectional study, the American Psychological Association (2013) reported that individuals aged 18 to 33 reported more stress than older individuals did. However, one is unable to distinguish whether the difference in depression prevalence is due to age or cohort differences. For example, younger individuals may always have a higher prevalence of depression when compared to older individuals, and it is not increasing. A time-lag study analyzed depressive symptomatology and reported that Americans in 2000-2010 reported statistically higher levels of these symptoms compared to 1980-1990 (Twenge, 2015). These results hold age constant and thus the change in depression prevalence is due to time or cohort. Other researchers in the United States, using time-lag design, found that birth cohorts in recent years’ experience higher rates of depression than older birth cohorts (Compton, Conway, Stinson, & Grant, 2006; Herbst, 2011; Lester, 2013).

There has been a push to medicalize these symptoms and to define them and to eliminate the stigma behind mental health, therefore the seemingly visible increase in depression, may only be an increase in actual diagnosed depression, or increased comfortability in reporting depressive symptoms, and not necessarily increase in depression. The Diagnostic and Statistical Manual of Mental Disorders (DSM) has become the standard for diagnosing depression; however, the
symptoms and diagnostic criteria are not adapted for gender or age (Blom, Forsman, Yang, Serlachius, & Larsson, 2014). This can lead to low diagnostic validity, since many symptoms of depressive disorders are not consistent through the experiences of age and of gender (Blom et al., 2014; Kendler, Gardner, & Lichtenstein, 2008).

**Depression among college students.** Nearly one-half of the disease burden for young adults in the United States is due to a mental health disorder (World Health Organization, 2008), most having the first onset by age 24 (Kessler et al., 2005). The prevalence of mood disorders does not appear to differ between college students and non-college-attending peers; however, college students are sometimes seen as being a privileged population, however the burden of disease falls on them evenly (Hunt & Eisenberg, 2010). Additionally, according to the 2015 Association for University and College Counseling Center Director Survey, 73.1% of directors’ report that the severity of student mental health concerns has risen on their campus (Reetz, Barr, & Krylowicz, 2014). The top two presenting concerns directors report are anxiety (47.34%) and depression (40.13%). According to the American College Health Association (2011), 10% of students were diagnosed and/or being treated for major depressive disorder in the past 12 months. In addition to students diagnosed or treated for major depressive disorder, there may also be a large number of individuals with depressive symptoms who do not seek out diagnosis or treatment. Michael, Huelsman, Gerard, Gilligan, and Gustafson (2006) found that only 27% of the students who reported high depressive symptoms on the Symptom Checklist 90- Revised scale, were undergoing some form of supervised treatment. These data suggest that there is a substantial number of students experiencing moderate to severe depressive symptoms and are not requesting and/or receiving care. Potential barriers to receiving treatment include limited time, concerns of privacy, lack of emotional openness, and financial restraints (Givens & Tjia, 2002;
There are three primary clinical tools used for the assessment of depression based on frequency of depressive symptoms: the Beck Depression Inventory (BDI), the Center for Epidemiologic Studies Depression Scale (CES-D), and the Patient Health Questionnaire, form 9 (PHQ-9). Roberts, Glod, Kim, and Houchell (2010), examined data from the National College Health Risk Behavior Survey and found that 22% (n = 92) of the sample scored over 20 on the BDI, indicating moderate level depression. Reyes-Rodríguez, Rivera-Medina, Câmara-Fuentes, Suárez-Torres, and Bernal (2013) assessed freshman students at the University of Puerto Rico and found that 9% of the students reported moderate level depression (BDI > 20). Utilizing the CES-D to assess depressive symptoms, Goebert et al. (2009) found that 12% (n = 226) of students had probable major depression (CES-D >21). However, this study examined depression prevalence in medical students and residents, and is therefore not entirely applicable to the study at hand. Similar to the current study, Eisenberg, Gollust, Golberstein, and Hefner (2007) examined undergraduate students in a midwestern university using the PHQ-9 and reported that more than 13.8% (n = 93) of participants scored six or higher. In a study conducted at Emory University, Garlow et al., 2008) reported that 30.6% (n = 217), 16.6% (n = 118), and 6.6% (n = 47) of students screened at moderate (10-14), moderately severe (15-19), and severe depression (≥ 20), respectively.

Complications of depression may affect many aspects of students’ lives, including reduced quality of life, impaired social functioning, and most concerningly, suicide (U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Mental Health, 2015). Suicide is the major complication of major depressive disorder (WHO,
2009), and is the 4th leading cause of death in individuals aged 15-44 (Rui, Kang, & Albert, 2013). Additionally, moderate depression in college students is also associated with tobacco use, poor eating, binge drinking, and engaging in fights (Allgöwer, Wardle, & Steptoe, 2001; Ellis & Trumpower, 2008; Glied & Pine, 2002; Weitzman, 2004). The National Alliance on Mental Illness found in 2012 that more than 45% of college students who stopped attending university, did so due to mental health reasons that went untreated and unseen by university counseling centers (D’Amico, Mechling, Kemppainen, Ahern, & Lee, 2016). In fact, only one-third of students are reported to talk about these feelings and/or seek help for them (Sarokhani et al., 2013). This can be a significant problem for universities, as retention and graduation rates are of utmost importance.

**Pathophysiology of depression.** While there are a few accepted risk factors for depression, including familial history, major life changes, and physical illness, there is no one established biochemical marker to diagnose depression nor one mechanism by which depression occurs. Depression is multifactorial, stemming from genetics and environmental factors. One promising hypothesis into the development of depression is found from data collected in family, twin, and adoption studies. Sullivan, Neale, and Kendler (2000) examined the genetic epidemiology of major depressive disorder with a meta-analysis and concluded that MDD was a familial disorder, and the familiality results from genetic influences and variations more than environmental factors (i.e., parenting style and socioeconomic status). Several genome-wide association studies examining single nucleotide polymorphisms (SNP) report finding no SNP that reached significant correlation to MDD diagnosis (Muglia et al., 2010; Lewis et al., 2010; Shi et al., 2011; Shyn et al., 2011; Sullivan et al., 2009; Terracciano et al., 2010; Wray et al., 2012).
Along with SNP’s, certain candidate genes specifically those dealing with serotonin and dopamine neurotransmission have been hypothesized to be associated with MDD as those with depression have dysfunctional neurotransmission of serotonin and dopamine. Specific genes and transporters examined include 5-HTT and TPH, however several replication and meta-analyses report no significant association (Ahdidan, Foldager, Rodell, Videbech, & Mors, 2013; Munafò, Durrant, Lewis, & Flint, 2009). Despite minimal evidence supporting specific candidate genes or SNP’s having association with MDD, strong links between reduced dopamine and serotonin transmission and MDD are present.

**Psychological and pharmacological treatment for depression.** Current treatment options for individuals experiencing depression include a mixture of psychotherapies and antidepressant medications. Psychological treatment strategies have emerged and have been shown to be highly effective, including cognitive-behavioral therapy, interpersonal psychotherapy, psychodynamic therapy, and problem-solving therapy (Gelenberg et al., 2010). The most commonly prescribed pharmacological intervention is SSRI’s, or selective serotonin reuptake inhibitors, especially in the cases of adolescents and young adults (Cousins & Goodyer, 2015). However effective, some of the side effects of these medications: weight gain, sleep disturbances, and sexual dysfunction, may hurt a new student's’ school performance, social confidence and perhaps overall health (Gelenberg et al., 2010). These individuals may be looking for non-pharmaceutical therapies to use alongside or instead of pharmacological interventions.

**Non-pharmaceutical treatments of depression.** Forms of non-pharmaceutical treatment that have been successfully implemented for mild depression include exercise, mind-body therapy, nutritional interventions, and dietary supplements. In a study completed by Edman, Lynch, and Yates (2014), regular exercise displayed a significant negative correlation to the
participant’s depression score; however, this significant effect was only experienced in male participants. One of the challenges to trials on exercise is the inability to blind the participants to the treatment they are receiving. Mind-body therapy has been shown to be an effective approach to not only treating current symptoms but in preventing them as well (D'Silva, Poscablo, Habousha, Kogan, & Kligler, 2012). Bo, Mao, and Lindsey (2017), in a meta-analysis found that, when compared to control groups, individuals who were active in mind-body interventions had short-term reductions in depressive symptoms. However, no long-term effects were reported.

Nutraceuticals, or non-prescription naturally occurring substances, are a class of products that are a common adjunct or treatment option for those with mild to moderate depression. Commonly studied nutraceuticals for the complementary or alternative treatment of depression are omega-3 fatty acids, vitamin B, folic acid, S-Adenosylmethionine (SAMe), and lastly vitamin D. Abnormal omega 3-fatty acid composition in the cell membrane can affect signal transduction and regulation (Su, Huang, Chiu, & Shen, 2003). Nemets, Nemets, Apter, Bracha, and Belmaker (2006) found that an Omega-3 fatty acid supplement, compared to a placebo, had a significant effect on improving the Children’s Depression Rating Scale, Children’s Depression Inventory, and Clinical Global Impression score. Additionally, Su et al. (2003) conducted a randomized controlled trial examining the interaction effect of omega-3 fatty acid supplementation (9.6 grams) or placebo alongside current depression medication. Individuals consuming the omega-3 fatty acids experienced a significant decrease in depression score, measured by the Hamilton Rating Scale for Depression. Conversely, Marangell et al. (2003) reported finding no intervention effect of a two-gram omega-3 supplement compared to the placebo. However, the dose was exclusively docosahexaenoic acid (DHA) and is a relatively low dose.
Several B vitamins have been tested for their effect on depression, most commonly vitamin B$_{12}$ (cobalamin) and B$_9$ (folic acid). These specific B vitamins are commonly targeted for testing due to their activity in the synthesis of S-adenosylmethionine (SAMe), which is involved in the metabolism of dopamine, norepinephrine, and serotonin (Brown, Gerbarg, & Bottiglieri, 2002). Tiemeier et al. (2002) reported a significant correlation between both cobalamin and folic acid and depression disorders. Similarly, Skarupski et al. (2010) concluded that higher intakes of vitamin B$_6$ (pyridoxine), folate, and cobalamin were associated with lower rates of incident depression after a 12-year follow-up. However, randomized controlled trials have not shown that supplementation of vitamin B$_6$ (Bryan, Calvaresi, & Hughes, 2002; Williams et al., 2005) vitamin B$_{12}$ (Bryan et al., 2002; Hvas, Juul, Lauritzen, Nexo, & Ellegaard, 2004), or folic acid (Walker et al., 2010) is effective in minimizing depressive symptoms. However, folate is shown to be a beneficial addition to antidepressant therapy (Coppen & Bailey, 2000; Taylor, Carney, Goodwin, & Geddes, 2004).

SAMe is a synthetic form of dietary amino acids and its role in the body is as a methyl donor involved in the metabolism of dopamine, serotonin, and norepinephrine (Bottiglieri, 2002; Mischoulon & Fava, 2002). Williams, Giard, Jui, Sabina, and Katz (2005) conducted a meta-analysis and concluded that there was an effect of SAMe in the treatment of depression, however, all studies were short-term supplementation and only a few were randomized controlled trials. Therefore, more long-term research should be conducted to examine the safety, bioavailability, and mechanism of action of SAMe.

Depression involves the abnormal neurotransmission of serotonin. Vitamin D has been proposed to assist in the synthesis, release, and function of serotonin by the action of the vitamin D binding proteins and DBP receptors located in the brain (Patrick & Ames, 2015). Vitamin D
has been correlated with mood disorders in correlational studies conducted with the general population (Anglin et al., 2013). Vitamin D remains one of the more promising interventions for mild to moderate depressive symptoms and has been shown in meta-analyses to be statistically significant in improving symptoms by supplementation (Shaffer et al., 2014; Spedding, 2014).

**Vitamin D Deficiency and Depression**

Vitamin D has endocrine, paracrine and autocrine functions via the VDR’s (Hendrix, Anderson, May, & Morris, 2004). Vitamin D is utilized in many physiological systems, including the brain (Ramagopalan et al., 2010), as evidenced by the presence of the hydroxylating enzymes which act on 25-hydroxyvitamin D to form 1,25-dihydroxyvitamin D (Obradovic, Gronemeyer, Lutz, & Rein, 2006). These enzymes can be found in the hypothalamus, cerebellum, and substantia nigra, which are all thought to be involved in the pathophysiology of depression. Using VDR’s in the adrenal medulla, vitamin D modulates the hypothalamic-pituitary-adrenal axis and assists in the regulation of adrenaline, noradrenaline and dopamine production (Puchacz, Stumpf, Stachowiak, & Stachowiak, 1996). Additionally, vitamin D protects dopamine and serotonin from depletion (Cass, Smith, & Peters, 2006).

Several epidemiological and cross-sectional studies have concluded that in healthy adult populations, there is an increased risk of depression in individuals with decreased serum vitamin D (Hoang et al., 2011; Jaddou et al., 2012; Kjærgaard, Joakimsen, & Jorde, 2011; May et al., 2010; Milaneschi et al., 2014). Conversely, Zhao, Ford, Li, and Balluz (2010), examined the National Health and Nutrition Examination Survey’s participants’ depression scores and found no significant difference in prevalence of depression between the low and high serum vitamin D concentration groups. However, the mean concentration of serum vitamin D was significantly lower in the clinically depressed group (PHQ-9 ≥ 10) compared to the participants with PHQ-9
scores of less than 10. Nanri et al. (2009), reported finding no significant associations between serum vitamin D and depressive symptoms in Japanese adults. However, during winter months the prevalence of depressive symptoms decreased with an increase in serum vitamin D. This result hints at the effects of seasonality on mood and serum vitamin D concentrations. Two recent meta-analyses, inclusive of studies done on populations other than the healthy general population, have also concluded that there is a significant correlation between lowered serum vitamin D and an increase in risk of depression (Anglin et al., 2013, Ju et al., 2013).

Along with the research completed on the general population, there have been several studies that examined younger populations and have conflicting results. Ganji et al. (2010), utilized National Health Examination Survey data to examine the correlation between vitamin D status and depression in young adults (n = 7,970), aged 15 to 39. The authors found that the odds ratio for having current depression symptoms is significantly higher (OR = 1.85) in those with low serum vitamin D (≤ 20 ng/ml) compared to those with a higher concentration (≥ 30 ng/ml). Conversely, Kwasky and Groh (2012) examined female college students (n = 139) and found that no significant difference in serum vitamin D between those who were categorized as “depressed” (BDI-II ≥ 20) or “not depressed” (<20). A score of ≥ 20 on the BDI-II indicates moderate to severe depression, and the use of this high cutoff is a possible reason for not finding significant results. Similarly, Kwasky and Groh (2014) examined the depression scores of female college students (n = 77) and found no significant differences of BDI scores between those who had sufficient versus insufficient vitamin D. Conversely, Kerr et al. (2015) conducted a seasonal study with undergraduate females (n = 185) and concluded that lower vitamin D status predicted clinically significant depressive symptoms (assessed with CES-D) when controlling for season. Furthermore, Kerr et al. (2015) found that lower levels of depressive symptoms in fall
participants, when compared to winter and spring, were explained by the higher levels of vitamin D.

Along with correlational data connecting vitamin D to depression, there is experimental data showing the effectiveness of vitamin D as a treatment for depressive symptoms. Experimental research with healthy adult populations, populations with vitamin D insufficiency and deficiency, and individuals with diagnosed MDD, show potential for vitamin D supplementation to improve depressive symptoms. Sanders et al. (2011), examined the effect of an annual dose of 12,500 µg or a placebo dose for 3-5 consecutive years and found that while the serum levels in the intervention group improved by 41% in the first year, there was no improvement in any of the four mental health questionnaires. However, this study was conducted on postmenopausal women and depression was not specifically evaluated at using clinical tools. Also examining postmenopausal women, Bertone-Johnson et al. (2011) concluded that women who consumed a 10 µg of vitamin D with 1,000 mg calcium supplement did not have a significantly lower odds ratio of experiencing depressive symptoms compared to the placebo group. However, the authors did not test serum levels of participants to examine if there was an increase in serum levels with the supplementation or placebo group. Vieth, Kimball, Hu, and Walfish (2004), enlisted healthy outpatient adults (n = 51) to receive either or 15 µg or 100 µg per day of vitamin D and found that both dosages were effective in significantly increasing serum vitamin D by 31 ng/ml and 44 ng/ml respectively. Additionally, the authors found that the group which consumed 100 µg per day, had significantly higher scores in wellbeing compared to the 15 µg per day group. However, the participants were outpatient clients being seen for thyroid issues and therefore not thoroughly applicable to the general population. Jorde, Sneve, Figenschau, Svartberg, and Waterloo (2008), enlisted otherwise healthy overweight and obese
individuals to consume either 1,000 or 500 µg of vitamin D or a placebo supplement per week for one year. At baseline, individuals at baseline with serum vitamin D of < 16 ng/ml scored significantly higher on the Beck Depression Inventory (BDI) than those with serum levels higher than 16 ng/ml. Jorde et al. (2008), then examined the effect of supplement intervention and concluded that the 1,000 and 500 µg per week groups increased their serum vitamin D after one year compared to baseline. Additionally, the 1,000 µg per week group significantly reduced their BDI score, while the 500 µg per week saw significant improvements only in the 14-21 score subgroup, and the placebo saw no significant changes. However, the population group causes the results to not be generalized. A study conducted on young adults in Queensland, Australia found that supplementing with 125 µg daily for six weeks was not effective in improving their BDI scores compared to the placebo (Dean et al., 2011). However, the mean serum concentration of participants was 30.7 (SD = 8.1) at baseline; therefore, the subjects were already considered to have adequate serum concentrations according to the Institute of Medicine, Food and Nutrition Board.

A few studies have been conducted utilizing participants who were either insufficient or deficient. Kjærgaard et al. (2012), randomized healthy adults with low serum vitamin D (< 20 ng/ml) to receive either 1,000 µg per week of vitamin D or placebo for six months. The authors also implemented a case-control component and had an additional control group of individuals with high serum vitamin D (> 30 ng/ml) that received a placebo. Kjærgaard et al. (2012), concluded that while participants with low serum vitamin D were more depressed compared to those with high concentrations at baseline, there was no significant improvement in BDI scores with supplementation. However, the placebo group did exhibit a significant difference in BDI score, which may reflect some error in the study. Conversely, Högberg et al. (2012), examined
the effect of vitamin D supplementation for three months on adolescents diagnosed with depression and low serum vitamin D concentrations ($M = 16.4 \, \text{ng/ml}$) in a case series study. Participants were instructed to consume 100 µg daily for one month, followed by 50 µg daily for two months, and mood was assessed by the Mood and Feelings Questionnaire. The authors not only found a baseline correlation between low serum vitamin D and mood, but an intervention effect of supplementation on the significant reduction of depressive symptoms. An additional study in which vitamin D supplementation (37.5 µg/day) was used as an adjunctive therapy along with Fluoxetine was also successful in improving depressive symptoms, assessed by the Hamilton Depression Rating Scale, compared to control taking only Fluoxetine (Khoraminya, Tehrani-Doost, Jazayeri, Hosseini, & Djazayery, 2013). Similarly, Sepehrmanesh et al. (2016) utilized participants with existing major depressive disorder and examined the effect of 1,250 µg per week of vitamin D compared to a placebo group and found that after adjusting for baseline values, there was a significantly greater improvement in BDI scores in the supplement group compared to control. However, there was no indication of whether participants were taking antidepressant medication. In addition, two meta-analyses that focused only on studies examining the effect of supplementation on adult populations have found that vitamin D significantly improved depressive symptoms (Shaffer et al., 2014; Spedding, 2014). One limitation to many of the aforementioned studies is not taking season into account. Intervention studies conducted in the winter months in northern latitudes examining the effectiveness of vitamin D supplementation and depressive symptoms have shown beneficial effects (Lansdowne & Provost, 1998; Shipowitz, Moore, Corbett, & Bindler, 2009). Shipowitz et al. (2009) found that after consuming a daily 125 µg vitamin D supplement for eight weeks during winter,
participants with low serum vitamin D (< 40 ng/ml) not only significantly increased serum vitamin D but decreased BDI scores compared to baseline.

A miniscule number of research articles have been published examining the association of vitamin D intake, from both dietary and supplements, and depressive symptoms. Bertone-Johnson et al. (2011) examined this association in postmenopausal women, aged 50-79, in a cross-sectional and prospective study. They found at baseline a significant inverse correlation between total vitamin D intake and prevalence of depressive symptoms, assessed by the Burnam scale. Specifically, higher intake of vitamin D from food sources specifically provided a significant 20% lower risk of depression. At a three year follow up, Bertone-Johnson et al. (2011) found the same results as baseline. This research project aims to add to the existing data on low vitamin D intake, from dietary sources and supplements, and the correlation to mood.
Chapter III: Methodology

This study was a cross-sectional survey research project including 155 undergraduate students at the University of Wisconsin-Stout. The objective of this study was to determine the daily intake of vitamin D in undergraduate students during the winter months. Secondary objectives were to analyze the relationship of intake and other predictive factors of vitamin D insufficiency to mood scores, as well as correlate dietary intake to vitamin D knowledge scores. This chapter will introduce the subject selection and description, instrumentation, data collection procedures, and data analysis. Limitations of this study design will also be explored.

Subject Selection and Description

Approval of this study was obtained from the University of Wisconsin-Stout Institutional Review Board for the Protection of Human Subjects in Research (Appendix A). Undergraduate students at the University of Wisconsin-Stout were recruited for this study.

This population group was chosen for a few primary reasons. The primary investigator has a strong interest in college health, and chose to use a convenient sample of UW-Stout students to complete this research project. Additional factors in choosing this population include the reported high prevalence of depression and vitamin D insufficiency among the college age population. An estimated 30.6% of college students experience clinically diagnosable depression (Ibrahim, Kelly, Adams & Glazebrook, 2013). Additionally, 36% of males and 37% of females aged 19-30 have serum vitamin D concentrations less than 30 ng/ml, while in adults over the age of 70 it is 24% and 27%, for males and females respectively (Looker, 2011). Furthermore, mean consumption of vitamin D in is commonly under the recommended daily allowance (RDA) in the college student population (Cress, 2014; Ouellette et al., 2012; Forney et al., 2014; Halliday et al., 2011; Lacey et al., 2004). In areas in the northern hemisphere, specifically 45º N and more
north, the availability of ultraviolet rays for vitamin D synthesis is practically negligible during the months of October to March (Webb et al., 1988). Menomonie Wisconsin is located at 44.8º N, placing its residents during the winter months at high risk for being vitamin D insufficient or deficient without consuming the recommended daily amount of dietary vitamin D. This study was conducted in late February, to minimize the possibility of ultraviolet exposure as a contributor to vitamin D status.

Recruitment of Participants

A survey link was distributed to selected professors in all three University colleges to ensure participation from a wide sample of students. Professors then forwarded the link to students in their classes, a few opting to offer extra credit to students for proof of completion. Participants were asked to provide implied consent to participate in this study and were presented with a description of the study, discussion of risks and benefits, time commitment, confidentiality, right to withdraw, and information on IRB approval (Appendix A).

Inclusion and Exclusion Criteria for Eligibility

Inclusion criteria for this study included all undergraduate students at the University of Wisconsin-Stout between the ages of 18 and 24. Exclusion criteria included not being currently enrolled as an undergraduate student at the University of Wisconsin-Stout, self-reporting an age under 18 or over 24, or current pregnancy. Biological sex, race/ethnicity, and height and weight information was collected, however, no individual was excluded based on any of these factors.

Implied Consent Procedure

Participants were given access to this survey via professors and academic advisors, and not the researcher themselves. The email sent from professors included a brief description of the objectives of the study, description of the survey, and a summary of risks and benefits of
participation. It was made clear that participation was voluntary and the student would not face any academic punishment if they did not complete the survey. It was also described that they may close out of the survey at any time, however, responses could not be withdrawn after submission.

**Risks and Precautions**

The risk of participation in this research survey was minimal. Participants were informed of the possible emotional risks from taking the mood survey and becoming aware of one’s mental health status. The mood survey utilized in this research was validated in the general population, and is commonly used in clinical settings (Martin, Rief, Klaiberg, & Braehler, 2006). Participants were also given University Counseling Center resources in case taking the mood survey affected them in a negative way, or in case they wanted to talk to someone about the survey.

**Data Safety Monitoring**

Data obtained from this survey was used to assess the participant’s daily intake of vitamin D, vitamin D related knowledge, and mood status. Data collection was completed anonymously using Qualtrics, a secure and password protected tool. Access to research data was permitted to the researcher only on a password protected, personal computer. The data were not saved on any other devices, or external servers or drivers.

**Compensation of Subjects and Cost to Subjects**

Participants were offered compensation by being entered into a drawing for the chance to win one of five, $20 Amazon gift cards. Participants were directed to a separate Qualtrics form, results of which could not be correlated to the results from the survey, to enter their name and email address to be contacted in the case of winning. A name randomizer was used to select five
names from all participants of the survey. There was no cost to subjects for their participation in the study.

**Instrument Design**

The researcher created a survey that contained validated tools and instruments used or designed by other authors with permission. The survey collected data on demographic information, serum vitamin D predictors, vitamin D-related knowledge, and mood.

**Demographics and anthropometrics.** Demographic and anthropometric data collected in this study included enrollment status, age, height, weight, biological sex, and race/ethnicity.

**Serum vitamin D predictors.** Predictors of vitamin D status, including UV exposure, dietary vitamin D consumption, and vitamin D supplement use were collected. UV exposure was assessed by questions about tanning behaviors or recent travel to a warm and sunny location. To estimate daily intake of vitamin D from dietary sources, a dietary vitamin D intake questionnaire developed by Hacker-Thompson et al. (2012) was utilized. Permission for use is found in Appendix B. This questionnaire, entitled the Brief Vitamin D Questionnaire (BVDQ) was validated and found to overestimate dietary vitamin D intake by less than 100 IU/day when compared to the 3-day food record. This questionnaire was developed to assess intake in postmenopausal women, and was completed by in-person interview, however due to time constraints and to ensure proper sample size the researcher chose to complete this questionnaire via electronic survey. This questionnaire, with permission from authors, was modified to exclude foods that are not associated with a midwestern college diet, for example, condensing the fish and including more processed meals (i.e., macaroni and cheese, ravioli, lasagna). In the modification of the BVDQ, the researcher also used an intake questionnaire developed by Cress (2014), which was adapted from a validated intake questionnaire by Taylor et al. (2009). Taylor
et al. (2009), measured calcium and vitamin D intake in adolescent girls with anorexia nervosa and concluded that the questionnaire did not differ in regard to vitamin D intake values compared to a four-day food record. Cress (2014), modified the validated form to measure solely vitamin D intake through an independent survey in college students. Permission from Cress (2014) can be found in Appendix C. To assist participants during the survey, description of serving sizes were included for reference as they were included in original validation. The questionnaire used can be found in Appendix D. The use of dietary supplements was assessed by using a series of questions to determine specific brand name, doses, and frequency of use.

**Vitamin-related knowledge.** Vitamin D-related knowledge was assessed using a modified questionnaire from past research conducted by Boland et al. (2014). The questions gauged students’ knowledge on sources of vitamin D, health effects and prevalence of deficiency, roles in the body, and intake recommendations. Permission from Boland et al. (2014) can be found in Appendix E. The questions, along with scoring method, used can be found in Appendix F.

**Mood survey.** The final block of questions included questions on mood. Current diagnosis and/or treatment of depression, anxiety, bipolar or seasonal affective disorder were asked as they may be expected to confound mood survey results. This was followed by the Patient Health Questionnaire- 9 (PHQ-9), which is a 9-symptom assessment of depressive symptomatology and was chosen for two primary reasons. First, the PHQ-9 is a validated and respected tool in the clinical setting, and second, it is free to replicate and use (Kroenke, Spitzer, & Williams, 2001). The PHQ-9 is displayed in Appendix D.

**Data analysis.** The Statistical Program for Social Sciences version 23.0 (SPSS, 2014) was used to analyze the data. Frequencies and descriptive statistics were run to analyze
participants’ current dietary vitamin D intake, responses to each question of the PHQ-9, and vitamin D-related knowledge questions. Food items on the food frequency questionnaire were converted into a daily vitamin D amount using the USDA Nutrient Database. Total vitamin D per food item was multiplied by a frequency of consumption factor. Composite scores were created for dietary vitamin D, supplemental vitamin D, total vitamin D intake (intake and supplements), the PHQ-9, and vitamin D-related knowledge. Frequencies and percentages of responses and descriptive statistics were run to answer questions regarding demographics of the study participants. A new variable for body mass index (BMI) was created from the height and weight of participants.

To assess the relationship between mood scores and vitamin D intake, a linear multiple regression was used with mood scores as independent variables and vitamin D intake, race/ethnicity, BMI, and biological sex as predictor variables. To complete this, a composite score was created for the PHQ-9 questions as well as for the dietary vitamin D and supplemented amount if applicable. A filter was initiated on the data to only use responses from individuals who did not tan or travel, or who were currently diagnosed or treated for a mental illness. A multiple linear regression was used to determine the effect of vitamin D intake as a predictor of mood scores while controlling or filtering for other predictors of vitamin D status and mood.

To further assess the relationship between PHQ-9 score and total dietary intake, an independent t-test was used to compare the mean composite PHQ-9 scores among the group of participants consuming an amount of vitamin D equal to or greater than the EAR (10 µg) versus those whose intake was below the EAR. To analyze a possible threshold effect, an independent t-test was also run to compare mean PHQ-9 scores between the group of participants who consumed less than the RDA (15 µg) for vitamin D.
To assess the relationship between vitamin D-related knowledge and dietary vitamin D intake, a linear regression test was run. The independent variable was mean dietary vitamin D, which is a composite score with the amount from both foods and supplements. The predictor variable was vitamin D-related knowledge, which was a composite score variable based on all of the individual knowledge-related questions. This test was run assuming that the amount of knowledge an individual has about vitamin D, will predict the amount that they consume or supplement. An additional linear regression was run examining the predictive relationship of vitamin D-related knowledge on total vitamin D intake, including supplements.

Finally, three independent t-tests were run to further examine the differences in mean knowledge score between the group of individuals who consumed the EAR and above versus those who consumed less than the EAR, between those who consumed over the RDA compared to less than, and between those who consumed supplements versus those who did not report consuming a vitamin D supplement.

Limitations

There are a few limitations to this study. First, the study population are individuals from a medium-sized public university, located in a rural town in the midwestern region of the United States. The dietary intake, mood status, and current knowledge may not be representative of all undergraduate students in the United States. Second, the limited number of validated questionnaires designed and validated for online purposes are minimal. The present study used a validated survey that was completed in-person, however, due to time and staffing, this research project was not able to conduct this portion of the survey in person. Additionally, while data on self-reporting of mood scores, and other stigmatized feelings/behaviors, show that online reporting is more accurate than in-person interviews, diet recalls are at risk of error.
(underestimating/overestimating) and may not be fully representative of participant intakes.

Furthermore, the recall-based food frequency report for the past two months, used in this study, may be less valid than a prospective record. Additionally, due to cost and time, the researcher was not able to measure actual serum vitamin D status of participants and had to rely on predictors and markers of low vitamin D status rather than actual measured values.

Finally, individuals with symptoms of depression may have poor self-care, and may not be eating well or taking vitamins due to their symptoms, so in effect, depression could potentially be a predictor of vitamin D deficiency. Conversely, individuals may be aware that vitamin D can improve their symptoms, and so it is also possible that individuals experiencing depression are purposefully going tanning or taking D supplements, in effect causing a purposeful increase in vitamin D.
Chapter IV: Results

The primary goals of this study were to establish the relationship between University of Wisconsin-Stout undergraduate student’s mood scores and total vitamin D intake, as well as the overall vitamin D-related knowledge and vitamin D intake. Specifically, the following research objectives were addressed in this study:

1. To determine the estimated daily intake of vitamin D consumed by undergraduates at the University of Wisconsin-Stout
2. To assess the prevalence and severity of depressive symptoms among students using the PHQ-9 mood score
3. To establish students’ level of vitamin D-related knowledge
4. To examine the predictive relationship of predictors of vitamin D status (intake, supplement use, UV exposure, sex, race/ethnicity, and BMI) to PHQ-9 score
5. To analyze the predictive relationship of vitamin D-related knowledge on modifiable predictors of vitamin D status (daily intake of vitamin D, supplement use, and UV exposure)

Demographic and Anthropometric Data

A total of 169 individuals followed the link sent to them via an email from a professor. Fourteen individuals received an early exit due to reporting to be over 24 years of age. Fourteen responses were blank; therefore, the data were removed from the data set. Participants had a mean age of 20.3, and distribution is displayed in table 5. The mean calculated BMI of participants was 25.3 kg/m² \((Mdn = 23.7, \ SD = 6.4)\), with a range of 17.7 to 63.4 kg/m², respectively. Table 6 displays BMI prevalence by category, with 10 missing values due to missing weight data. There was one impossible value of “0” for the biological sex variable. This
value was deleted, and therefore the biological sex variable has 154 total participants. The frequencies of biological sex of participants are found on table 7. Table 8 summarizes the characteristics of the sample population in regard to race and ethnicities for all participants. Two of the participants who selected “other” (n = 3) wrote in “multi” to describe their race/ethnicity.

Table 5

*Age of Survey Respondents*

<table>
<thead>
<tr>
<th>Age</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>16</td>
<td>10.3</td>
</tr>
<tr>
<td>19</td>
<td>33</td>
<td>21.3</td>
</tr>
<tr>
<td>20</td>
<td>47</td>
<td>30.3</td>
</tr>
<tr>
<td>21</td>
<td>27</td>
<td>17.4</td>
</tr>
<tr>
<td>22</td>
<td>21</td>
<td>13.5</td>
</tr>
<tr>
<td>23</td>
<td>5</td>
<td>3.2</td>
</tr>
<tr>
<td>24</td>
<td>6</td>
<td>3.9</td>
</tr>
</tbody>
</table>
Table 6

*Frequency of BMI Categories in Survey Participants*

<table>
<thead>
<tr>
<th>BMI Category</th>
<th>Frequency (N = 144)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>4</td>
<td>2.8</td>
</tr>
<tr>
<td>Healthy</td>
<td>88</td>
<td>61.1</td>
</tr>
<tr>
<td>Pre-obesity</td>
<td>30</td>
<td>20.8</td>
</tr>
<tr>
<td>Obesity, class I</td>
<td>10</td>
<td>6.9</td>
</tr>
<tr>
<td>Obesity, class II</td>
<td>8</td>
<td>5.6</td>
</tr>
<tr>
<td>Obesity, Class III</td>
<td>4</td>
<td>2.8</td>
</tr>
</tbody>
</table>

*Note.* Underweight = < 18.5 m/kg²; Healthy = 18.5-24.9 m/kg²; Pre-obesity = 25.0-29.9 m/kg²; Obesity, class I = 30.0-34.9 m/kg²; Obesity, class II = 35.0-39.9 m/kg²; Obesity, class III = ≥ 40 m/kg²

Table 7

*Biological Sex of Survey Participants*

<table>
<thead>
<tr>
<th>Biological Sex</th>
<th>Frequency (N = 154)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>112</td>
<td>72.7</td>
</tr>
<tr>
<td>Male</td>
<td>42</td>
<td>27.3</td>
</tr>
</tbody>
</table>
Table 8

Race(s)/ Ethnicity(ies) of Survey Participants

<table>
<thead>
<tr>
<th>Race(s)/ Ethnicity(ies)</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>White/Caucasian/Non-Hispanic</td>
<td>139</td>
<td>89.7</td>
</tr>
<tr>
<td>Hispanic/ Latino/a</td>
<td>5</td>
<td>3.2</td>
</tr>
<tr>
<td>Black/African American</td>
<td>1</td>
<td>0.6</td>
</tr>
<tr>
<td>Asian/ Pacific Islander</td>
<td>6</td>
<td>3.9</td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>1</td>
<td>0.6</td>
</tr>
<tr>
<td>Other</td>
<td>3</td>
<td>1.9</td>
</tr>
</tbody>
</table>

Tanning and Vacation History

Participants were asked about history of using artificial tanning lamps or recent travel to a warm and sunny location in the past two months (table 9). There were 115 participants (74.1%) who reported never tanning or traveling to a warm and sunny location. Of the remaining 40 participants, 22 reported travel to a warm and sunny location, and 23 reported use of artificial sunlamp use.
Table 9

Ultraviolet-B Exposure of Participants Within Past Two Months

<table>
<thead>
<tr>
<th>Artificial Sunlamp Use</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never</td>
<td>132</td>
<td>85.2</td>
</tr>
<tr>
<td>Once or twice</td>
<td>11</td>
<td>7.1</td>
</tr>
<tr>
<td>3 to 8 times</td>
<td>10</td>
<td>6.5</td>
</tr>
<tr>
<td>More than 8 times</td>
<td>2</td>
<td>1.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Travel to Warm and Sunny Location</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>22</td>
<td>14.2</td>
</tr>
<tr>
<td>No</td>
<td>133</td>
<td>85.8</td>
</tr>
</tbody>
</table>

Supplement Use and Dietary Intake

Survey respondents were asked to self-report any supplement use and/or frequency of vitamin D-containing foods during the past two months. Sixty participants (38.7%) reported consuming a dietary supplement which may contain vitamin D. The types of supplements consumed by those 60 participants included multivitamins (21.3%), vitamin D supplements (6.5%), calcium supplement (6.5%), and “other”. Self-described “other” supplements included; a vitamin C plus iron, vitamin C, fish oil (n = 2), biotin plus protein, and a hair, skin, and nails supplement. The participants who were consuming vitamin D-containing supplements (n = 57) reported their use as daily (66.7%), four to six times per week (12.3%), two to three times per week (14%), once a week (5.3%), or never (1.8%). The mean total amount of vitamin D ingested per day from supplements was then calculated for each participant, using self-reported frequency of use and label information for each type of supplement. The mean for vitamin D consumption
from a supplement was 8.5 µg ($SD = 20.85$ µg), and ranged from 3.33 µg to 135 µg. A visual representation of amount of vitamin D ingested via supplementation is seen in figure 1.

Figure 1. Average daily Vitamin D intake from supplementation. (Accounting for frequency of use.)

The amount of vitamin D obtained from dietary sources, excluding supplements, was established by an adapted validated questionnaire. The food source which provided the highest mean daily vitamin D among participants was dairy milk ($M = 2.3$ µg, $SD = 2.81$). Table 10 ranks the top ten contributing sources of dietary vitamin D, while table 11 lists the frequency of consumption for the top ten contributing foods.
<table>
<thead>
<tr>
<th>Food Item</th>
<th>$M$ Vitamin D ($\mu$g)</th>
<th>$SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dairy Milk</td>
<td>2.28</td>
<td>2.81</td>
</tr>
<tr>
<td>American/ Mozzarella Cheese</td>
<td>0.68</td>
<td>0.93</td>
</tr>
<tr>
<td>Salmon</td>
<td>0.46</td>
<td>1.06</td>
</tr>
<tr>
<td>Yogurt, non-Greek, fortified</td>
<td>0.44</td>
<td>0.70</td>
</tr>
<tr>
<td>Breakfast Cereal, cold, fortified</td>
<td>0.36</td>
<td>0.47</td>
</tr>
<tr>
<td>Egg</td>
<td>0.31</td>
<td>0.48</td>
</tr>
<tr>
<td>Almond Milk</td>
<td>0.27</td>
<td>0.93</td>
</tr>
<tr>
<td>Orange Juice, fortified</td>
<td>0.25</td>
<td>0.71</td>
</tr>
<tr>
<td>Soy Milk</td>
<td>0.23</td>
<td>0.99</td>
</tr>
<tr>
<td>Cod Liver Oil</td>
<td>0.19</td>
<td>1.82</td>
</tr>
</tbody>
</table>
Table 11

*Frequency of Consumption for Highest Vitamin D Contributors (N = 155)*

<table>
<thead>
<tr>
<th>Food</th>
<th>Frequency, N (%)</th>
<th>Never</th>
<th>&lt; 1 x month</th>
<th>2 x month</th>
<th>1 x week</th>
<th>2-3 x week</th>
<th>4-6 x week</th>
<th>1 x day</th>
<th>2 x day</th>
<th>≥ 3 x day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dairy Milk</td>
<td>(14.8)</td>
<td>23</td>
<td>10</td>
<td>16</td>
<td>15</td>
<td>30</td>
<td>15</td>
<td>22</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>American/Mozzarella Cheese</td>
<td>(6.5)</td>
<td>10</td>
<td>12</td>
<td>26</td>
<td>37</td>
<td>45</td>
<td>10</td>
<td>4</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Salmon</td>
<td>(51)</td>
<td>79</td>
<td>43</td>
<td>21</td>
<td>8</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yogurt, non-Greek, fortified</td>
<td>(22.6)</td>
<td>35</td>
<td>26</td>
<td>22</td>
<td>31</td>
<td>24</td>
<td>8</td>
<td>7</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Breakfast Cereal, fortified</td>
<td>(18.7)</td>
<td>29</td>
<td>23</td>
<td>27</td>
<td>21</td>
<td>25</td>
<td>17</td>
<td>12</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Egg, whole or yolk</td>
<td>(13.5)</td>
<td>21</td>
<td>20</td>
<td>27</td>
<td>31</td>
<td>26</td>
<td>12</td>
<td>12</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Almond Milk</td>
<td>(72.9)</td>
<td>113</td>
<td>16</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>2</td>
<td>7</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Orange Juice, fortified</td>
<td>(40.6)</td>
<td>63</td>
<td>33</td>
<td>26</td>
<td>18</td>
<td>9</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Soy Milk</td>
<td>(80)</td>
<td>124</td>
<td>11</td>
<td>6</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Cod Liver Oil</td>
<td>(94.8)</td>
<td>147</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The mean dietary vitamin D was 3.60 µg (*SD = 3.25*), with only 3.9% (*n = 6*) reaching the threshold of the EAR for vitamin D (10 µg per day) and only 1.3% (*n = 2*) of participants reaching the RDA (15 µg per day) through dietary intake of vitamin D containing foods. The mean vitamin D intake increased to 12.11 µg (*Mdn = 4.11, SD = 22.01*) after the inclusion of intake from supplements. For participants who reported consuming a supplement (*n = 60*) the mean increased from 4.11 µg (*Mdn = 2.91*) to 26.11 (*Mdn = 17.67*). There were three students...
who reported consuming supplements with high doses of vitamin D at 125 µg (n = 2) and 135 µg (n = 1) of vitamin D per day, which caused the mean to be skewed. Therefore, the median values are more reflective of overall average intake. After adding in vitamin D from ingestion of supplements, 71.6% and 77.4% of individuals were still obtaining less than the EAR and RDA, respectively. A histogram of the total dietary vitamin D is demonstrated in figure 2, while total vitamin D intake from diet and supplements is represented in figure 3. The 49 individuals who reported consuming a supplement containing vitamin D obtained a mean of 85.6% (54 - 98.5%) of their daily vitamin D from that supplement.

Figure 2. Daily average Vitamin D consumption from dietary sources.
Figure 3. Total Vitamin D intake from dietary sources and supplementation.

Vitamin D-Related Knowledge

The knowledge questionnaire was divided into four categories; sources of vitamin D (both environmental and dietary), health benefits of vitamin D, current recommendations for intake of vitamin D, and barriers to obtaining adequate status. The mean score of participants was 59%, 73%, 42%, and 58% for identifying food sources, choosing health benefits, knowing recommendations, and selecting barriers to adequacy, respectively. There were seven correct sources of vitamin D among thirteen total options. Table 12 gives a summary of selected responses to identify sources of vitamin D. The selected responses for the health benefits of vitamin D are found on table 13. Results on the multiple choice questions, how much time a fair skinned individual would need to spend outside to produce sufficient vitamin D, the prevalence
of insufficiency, and the RDA of vitamin D are displayed in table 14, 15, and 16, respectively.

Lastly, the selected answers to barriers in achieving optimal vitamin D status are shown in table 10. Overall, out of four points (1 per section) the mean score was 57.96% ($SD = 11.5$).

Table 12

*Selected Choices for Sources of Vitamin D (Multi-Select)*

<table>
<thead>
<tr>
<th>Response</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sun*</td>
<td>131</td>
<td>84.5</td>
</tr>
<tr>
<td>Milk/Dairy*</td>
<td>107</td>
<td>69</td>
</tr>
<tr>
<td>Eggs*</td>
<td>61</td>
<td>39.4</td>
</tr>
<tr>
<td>Fatty Fish*</td>
<td>48</td>
<td>31</td>
</tr>
<tr>
<td>Cod Liver Oil*</td>
<td>46</td>
<td>29.7</td>
</tr>
<tr>
<td>Fortified Cereals*</td>
<td>43</td>
<td>27.7</td>
</tr>
<tr>
<td>Mushrooms*</td>
<td>25</td>
<td>16.1</td>
</tr>
<tr>
<td>Fruits</td>
<td>72</td>
<td>46.5</td>
</tr>
<tr>
<td>Vegetables</td>
<td>72</td>
<td>46.5</td>
</tr>
<tr>
<td>Chicken</td>
<td>24</td>
<td>15.5</td>
</tr>
<tr>
<td>Nuts</td>
<td>24</td>
<td>15.5</td>
</tr>
<tr>
<td>Air</td>
<td>5</td>
<td>3.2</td>
</tr>
<tr>
<td>Water</td>
<td>4</td>
<td>2.6</td>
</tr>
</tbody>
</table>

*Note.* * = correct selection
Table 13

*Selected Choices for Health Benefits of Vitamin D (Multi-Select)*

<table>
<thead>
<tr>
<th>Response</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone Health*</td>
<td>119</td>
<td>76.8</td>
</tr>
<tr>
<td>Heart Health</td>
<td>68</td>
<td>43.9</td>
</tr>
<tr>
<td>Cancer Prevention</td>
<td>56</td>
<td>36.1</td>
</tr>
<tr>
<td>Vision</td>
<td>44</td>
<td>28.4</td>
</tr>
<tr>
<td>Wound Healing</td>
<td>52</td>
<td>33.5</td>
</tr>
<tr>
<td>Blood Clotting</td>
<td>34</td>
<td>21.9</td>
</tr>
<tr>
<td>None of the Above</td>
<td>6</td>
<td>3.9</td>
</tr>
</tbody>
</table>

*Note.* * = correct selection

Table 14

*Selected Choice for Sun Exposure Time for Sufficient Vitamin D Production (Single-Select)*

<table>
<thead>
<tr>
<th>Response</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 10 minutes per week</td>
<td>9</td>
<td>5.8</td>
</tr>
<tr>
<td>About 10-60 minutes a week*</td>
<td>75</td>
<td>48.4</td>
</tr>
<tr>
<td>About 1-6 hours per week</td>
<td>55</td>
<td>35.5</td>
</tr>
<tr>
<td>More than 6 hours per week</td>
<td>16</td>
<td>10.3</td>
</tr>
</tbody>
</table>

*Note.* * = correct selection

Table 15

*Selected Choice for Prevalence of Vitamin D Insufficiency (Single-Select)*

<table>
<thead>
<tr>
<th>Response</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1%</td>
<td>1</td>
<td>0.6</td>
</tr>
<tr>
<td>22%</td>
<td>52</td>
<td>33.5</td>
</tr>
<tr>
<td>42%*</td>
<td>89</td>
<td>57.4</td>
</tr>
<tr>
<td>98%</td>
<td>13</td>
<td>8.4</td>
</tr>
</tbody>
</table>

*Note.* * = correct selection
Table 16

Selected Choice for the Recommended Daily Allowance for Vitamin D (Single-Select)

<table>
<thead>
<tr>
<th>Response</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>200 IU</td>
<td>8</td>
<td>5.2</td>
</tr>
<tr>
<td>400 IU</td>
<td>41</td>
<td>26.5</td>
</tr>
<tr>
<td>600 IU*</td>
<td>32</td>
<td>20.6</td>
</tr>
<tr>
<td>800 IU</td>
<td>43</td>
<td>27.7</td>
</tr>
<tr>
<td>1,000 IU</td>
<td>21</td>
<td>13.5</td>
</tr>
<tr>
<td>2,000 IU</td>
<td>10</td>
<td>6.5</td>
</tr>
</tbody>
</table>

Note. * = correct selection

Table 17

Selected Choices for Barriers to Optimal Vitamin D Status (Multi-Select)

<table>
<thead>
<tr>
<th></th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Season*</td>
<td>113</td>
<td>72.9</td>
</tr>
<tr>
<td>Time of Day*</td>
<td>111</td>
<td>71.6</td>
</tr>
<tr>
<td>Skin Pigmentation*</td>
<td>92</td>
<td>59.4</td>
</tr>
<tr>
<td>Shade/Clouds*</td>
<td>90</td>
<td>58.1</td>
</tr>
<tr>
<td>Latitude*</td>
<td>87</td>
<td>56.1</td>
</tr>
<tr>
<td>Sunscreen Use*</td>
<td>78</td>
<td>50.3</td>
</tr>
<tr>
<td>Dairy Allergy*</td>
<td>66</td>
<td>42.6</td>
</tr>
<tr>
<td>Age*</td>
<td>64</td>
<td>41.3</td>
</tr>
<tr>
<td>Lactose Intolerance*</td>
<td>64</td>
<td>41.3</td>
</tr>
<tr>
<td>Pollution*</td>
<td>54</td>
<td>34.8</td>
</tr>
<tr>
<td>Vegan/Vegetarian Diet*</td>
<td>52</td>
<td>33.5</td>
</tr>
<tr>
<td>BMI*</td>
<td>28</td>
<td>18.1</td>
</tr>
<tr>
<td>Fatty Diets</td>
<td>54</td>
<td>34.8</td>
</tr>
<tr>
<td>Smoking</td>
<td>42</td>
<td>27.1</td>
</tr>
<tr>
<td>Wind</td>
<td>13</td>
<td>8.4</td>
</tr>
</tbody>
</table>

Note. * = correct selection
Vitamin D Intake and Vitamin D-Related Knowledge

A linear regression was run to explore the hypothesized predictive relationship of vitamin D related knowledge on vitamin D intake in survey participants. The first test examined the dependent variable of total knowledge score and total intake of vitamin D, from both dietary and supplemental sources. The results of the linear regression indicated that knowledge of vitamin D explained 0.4% variance in participants total vitamin D intake, which was not significant ($R = .05$, $F(1, 153) = .39$, $p = .54$). Furthermore, knowledge of vitamin D failed to predict vitamin D intake ($\beta = -.05$, $p = .54$).

A second regression analysis was used to test if vitamin D-related knowledge significantly predicted participants’ dietary intake, not inclusive of supplemental vitamin D. The analysis indicated that knowledge of vitamin D explained 2.5% variance in individual's vitamin D intake, ($R = .15$, $F(1, 153) = 3.89$, $p = .05$). Moreover, knowledge of vitamin D significantly predicted participants’ vitamin D intake with higher vitamin D-related knowledge predicting lower vitamin D dietary intake. ($\beta = -.16$, $p = .05$).

Two independent sample t-tests were completed to further examine the relationship between knowledge and intake further. First, participants were grouped into those consuming at or above the EAR of 10 µg and those consuming less than 10 µg from dietary sources alone, and compared the mean knowledge score in each group. Regarding vitamin D-related knowledge, individuals who consumed the EAR or higher from dietary sources alone had a significantly lower score ($M = 48.4\%$, $SD = 8.54$) compared to those who consumed less than the EAR per day ($M = 58.34\%$, $SD = 11.46$), $t(153) = 2.09$, $p = .04$. 
Table 18

Comparison of Mean Knowledge Score Between Individuals Consuming Under and Over the EAR for Vitamin D from Dietary Sources

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>SEM</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10 µg per day</td>
<td>149</td>
<td>58.34</td>
<td>11.46</td>
<td>0.94</td>
<td>2.09</td>
<td>0.04*</td>
</tr>
<tr>
<td>≥ 10 µg per day</td>
<td>6</td>
<td>48.42</td>
<td>8.54</td>
<td>3.49</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. * = Statistical significant p < .05

Conversely, in regard to vitamin D knowledge again, individuals who consumed the EAR or more with the inclusion of supplementation did not have a significantly different vitamin D knowledge score (M = 57.72%, SD = 13.5) compared to those who consumed less than the EAR (M = 58.05%, SD = 10.68).

Table 19

Comparison of Mean Knowledge Score Between Individuals Consuming Under and Over the EAR for Vitamin D from Dietary Sources and Supplementation

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>SEM</th>
<th>T</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10 µg per day</td>
<td>111</td>
<td>58.05</td>
<td>10.68</td>
<td>1.01</td>
<td>0.14</td>
<td>0.89</td>
</tr>
<tr>
<td>≥ 10 µg per day</td>
<td>44</td>
<td>57.72</td>
<td>13.5</td>
<td>2.04</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Additionally, those who self-reported consuming a supplement containing vitamin D did not have a significantly different vitamin D knowledge score (M = 58.55%, SD = 12.56) in comparison to those who did not report taking a supplement (M = 57.59, SD = 10.84).
Table 20

*Comparison of Mean Knowledge Score Between Individuals Who Reported Consuming a Supplement and Not Consuming a Supplement*

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>SEM</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consumed Supplement</td>
<td>60</td>
<td>8.54</td>
<td>12.56</td>
<td>1.62</td>
<td>1.62</td>
<td>.51</td>
</tr>
<tr>
<td>No Supplement</td>
<td>95</td>
<td>7.59</td>
<td>10.84</td>
<td>1.11</td>
<td>0.61</td>
<td></td>
</tr>
</tbody>
</table>

**Depression Prevalence**

The final part of the survey was an assessment of mood status using the PHQ-9. Twenty-eight students (18.1%) reported being currently evaluated and/or treated for a mood disorder, and 20 (12.9%) reported currently taking an antidepressant or antianxiety medication. Three participants (1.9%) reported feelings of self-harm or suicide more than half the days of the week. The total score on the PHQ-9 ranged from 0 (n = 25) to 27 (n = 1), with a mean score of 6.10 ($Mdn = 5.0$, $SD = 5.61$). Moreover, participant score spread categorized by PHQ-9 clinical diagnosing criteria can be found in table 21, with table 22 showing responses of individuals not being currently evaluated or treated with a mental illness. Table 23 shows responses of participants who scored a 1 or higher on the PHQ-9, to the question of how difficult these symptoms of depression have made daily living activities. This variable had two missing values. Overall spread of PHQ-9 scores of participants is shown in figure 4.
Table 21

**PHQ-9 Scoring Categories of all Participants**

<table>
<thead>
<tr>
<th>PHQ-9 Score</th>
<th>Frequency (N = 155)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>75</td>
<td>48.4</td>
</tr>
<tr>
<td>5-9</td>
<td>46</td>
<td>29.7</td>
</tr>
<tr>
<td>10-14</td>
<td>20</td>
<td>12.9</td>
</tr>
<tr>
<td>15-19</td>
<td>9</td>
<td>5.8</td>
</tr>
<tr>
<td>20-27</td>
<td>5</td>
<td>3.2</td>
</tr>
</tbody>
</table>

*Note.* 0-4 indicates minimal or no depression, 5-9 indicates mild, 10-14 indicates moderate, 15-19 indicates moderately severe, and 19-27 indicates severe depression

Table 22

**PHQ-9 Scores, Excluding Individuals with Current Diagnosis and/or Treatment for a Mental Health Condition**

<table>
<thead>
<tr>
<th>PHQ-9 Score</th>
<th>Frequency (N = 126)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>70</td>
<td>55.6</td>
</tr>
<tr>
<td>5-9</td>
<td>38</td>
<td>30.2</td>
</tr>
<tr>
<td>10-14</td>
<td>12</td>
<td>9.5</td>
</tr>
<tr>
<td>15-19</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>20-27</td>
<td>1</td>
<td>0.8</td>
</tr>
</tbody>
</table>

*Note.* 0-4 indicates minimal or no depression, 5-9 indicates mild, 10-14 indicates moderate, 15-19 indicates moderately severe, and 19-27 indicates severe depression
**Figure 4.** PHQ-9 scores of survey participants.

**Table 23**

**Self-Reported Difficulty Dealing with Depressive Symptoms**

<table>
<thead>
<tr>
<th></th>
<th>Frequency (N = 128)</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not difficult at all</td>
<td>50</td>
<td>38.5</td>
</tr>
<tr>
<td>Somewhat difficult</td>
<td>62</td>
<td>47.7</td>
</tr>
<tr>
<td>Very difficult</td>
<td>12</td>
<td>9.2</td>
</tr>
<tr>
<td>Extremely difficult</td>
<td>4</td>
<td>3.1</td>
</tr>
</tbody>
</table>

**Vitamin D Intake and Mood**

A multiple linear regression was utilized to explore the hypothesized predictive relationship of vitamin D intake on mood scores participants. Those who reported any travel,
tanning, or current evaluation or treatment for mental illness were excluded, yielding a total of 95 participants. The multiple linear regression examined the relationships between total PHQ-9 score, BMI, biological sex, race/ethnicity, and total vitamin D intake (inclusive of supplements).

The results of the linear regression indicated that total intake of vitamin D explained 6.2% of the variance in participants mood scores (n = 95), which was not significant (R = .25, \( F(4, 83) = 1.37, p = .25 \)). Furthermore, intake of vitamin D failed to predict mood scores (\( \beta = -.06, p = .57 \)). The scatterplot display of the relationship between PHQ-9 scores and dietary vitamin D can be seen in figure 5.

![Figure 5](image)

*Figure 5.* Scatterplot display of relationship between total Vitamin D intake and total PHQ-9 score.
Due to the large range and minimal spread of data, the data were log transformed. A multiple linear regression was used to examine the relationships between total PHQ-9 score, BMI, biological sex, race/ethnicity, and log transformed total vitamin D intake (inclusive of supplements). The results of the linear regression indicated that log transformed total intake of vitamin D explained 5.8% variance in participants mood scores, which was also not significant (R = .24, F(4, 83) = 1.29, p = .28). Furthermore, intake of vitamin D failed to predict mood scores (β = -.00, p = .99).

An independent samples t-test was conducted to examine the possible relationship further. Individuals who consumed under the EAR did not have significantly different mood scores (M = 5.32, SD = 4.31) than individuals who consumed the EAR or above of vitamin D (M = 5.61, SD = 7.12).

Table 24

Comparison of Individuals Consuming Above and Below the Estimated Average Requirements on Mean PHQ-9 Score

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>SEM</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10 µg per day</td>
<td>72</td>
<td>5.32</td>
<td>4.31</td>
<td>0.51</td>
<td>-0.18</td>
<td>0.86</td>
</tr>
<tr>
<td>≥ 10 µg per day</td>
<td>23</td>
<td>5.61</td>
<td>7.12</td>
<td>1.48</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

To explore the possibility of a threshold effect, an additional independent t-test was run to determine if there was a mean PHQ-9 score difference between the group of individuals who consumed 15 µg per day (RDA) or more compared to those who consumed less than 15 µg of vitamin D. The results showed that individuals who consumed the RDA or higher of vitamin D did not score differently (M = 5.29, SD = 6.24) compared to the group that consumed less than the RDA (M = 5.41, SD = 4.91).
Table 25

Comparison of Individuals Consuming Above and Below the RDA on Mean PHQ-9 Score

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>SEM</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 15 µg per day</td>
<td>81</td>
<td>5.41</td>
<td>4.91</td>
<td>0.55</td>
<td>0.08</td>
<td>0.94</td>
</tr>
<tr>
<td>≥ 15 µg per day</td>
<td>14</td>
<td>5.29</td>
<td>6.24</td>
<td>1.67</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Chapter V: Discussion, Conclusion and Recommendations

This study examined the relationships between vitamin D intake and mood scores and vitamin D intake and vitamin D-related knowledge in undergraduate students at the University of Wisconsin-Stout. Students were recruited to partake in a survey in February 2017 to assess vitamin D intake, supplementation, tanning and vacation history, vitamin D-related knowledge, and mood status for within-subject’s analysis. The survey was distributed via email and all information was self-reported. This chapter states the limitations of the study, draws conclusions from results, compares the findings to related research, and makes recommendations for future research.

Conclusions

Participants of this study self-reported inadequate consumption of vitamin D, overall vitamin D-related knowledge, and a prevalence of clinically diagnosable depression comparable to related research. Gozdzik et al. (2008), reported that 74% of healthy young adults (18-30 years of age) in Canada had insufficient serum vitamin D levels (< 20 ng/ml). In the northern hemisphere, specifically those above 45º N, the availability of ultraviolet rays for vitamin D synthesis is practically negligible (Webb et al., 1988). Menomonie, Wisconsin is located at 44.8º N placing its residents during the winter months at high risk for being vitamin D insufficient or deficient if they do not meet these requirements from the diet. Hilliard, Fugrmann, and Brunt (2015), found that 97% of students using university dining halls were not consuming over the RDA for vitamin D. While this research did not measure serum vitamin D levels, the data collected in this survey combined with research conducted on vitamin D synthesis from UVB rays in the winter months in the northern latitudes gives an opportunity to hypothesize that a large number of students at the University of Wisconsin-Stout may suffer from vitamin D
insufficiency or deficiency due to lack of vitamin D synthesis from UVB rays and dietary vitamin D intake.

The high levels of inadequate dietary intake of vitamin D found in this study, when taken into account with previous literature, support the likelihood that a large majority of college students are not consuming enough vitamin D through any source alone. Dairy milk was the highest contributor of vitamin D to the diet, followed by American/ Mozzarella cheese, salmon, fortified yogurt, fortified breakfast cereals, and eggs. However, only 3.9% of participants were consuming over the EAR for vitamin D ($M = 3.60$ μg, $SD = 3.25$). These data support findings of previous studies concluding that college students mean vitamin D intake fall below the recommended values (Cress, 2014; Ouellette et al., 2012; Forney et al., 2014; Lacey et al., 2004).

Additionally, this study contributes data on university student tanning and supplementation habits. These two factors are the two most common methods of obtaining vitamin D in the winter months in northern latitudes. The majority of participants in this study report not tanning in the past two months (85.2%), however, others reported tanning “once or twice” (7.1%), “3-8 times” (6.5%), or “more than 8 times” (1.3). Furthermore, this study found that 38.7% of students were consuming a supplement containing vitamin D. Participants who reported consuming a supplement containing vitamin D reported taking “multivitamins” (21.3%), “vitamin D supplements” (6.5%), or “calcium supplements” (6.5%). A vitamin D supplement assisted in increasing supplement consumers’ ($n = 60$) mean intake from 4.11 μg from dietary sources, to 26.11 ($Mdn = 17.67$) μg per day. Halliday et al. (2011), found similar results in Division I athletes in that only 36% of participants reported consuming a vitamin D-containing supplement, which assisted in the athletes obtaining the recommended intakes of vitamin D.
This research contributes evidence of limited vitamin D knowledge in university students. Participants scored 59%, 73%, 42%, and 58% when asked about sources, health benefits, recommendations, and barriers to optimal status for vitamin D. Almost half of participants (46.5%) selected “fruits” and “vegetables” as sources of vitamin D. Only 39.4%, 31%, and 69% of individuals responded that eggs, fatty fish, and milk/dairy were sources of vitamin D. A majority of participants (76.8%) selected the correct response of bone health as a health benefit of vitamin D. Regarding knowledge of recommendations of vitamin D, 48.4% of participants selected the correct response for amount of sun exposure required to obtain optimal daily vitamin D (10-60 minutes per week), 57% responded correctly to the prevalence of vitamin D insufficiency in the US, while only 20.6% knew the RDA for vitamin D. Finally, when asked about barriers to achieving optimal vitamin D status, more than half of respondents correctly selected that skin pigmentation, shade/clouds, time of day, latitude, season, and sunscreen use were barriers.

The current study suggests that a higher knowledge level about vitamin D is not associated with increased intake. In fact, this study found that individuals consuming less than the EAR (without supplementation) had significantly higher knowledge scores than those consuming over the EAR. Moreover, individuals consuming a supplement did not have significantly higher knowledge scores than those not consuming a vitamin D-containing supplement. Goodman (2015) found that with the use of a mobile application, there was a significant increase in dietary vitamin D in Canadian college students. The researcher attributed the results to the participants’ ability to receive personalized nutrition information, and the ability to self-monitor diet and vitamin D intake. The ability to view current status and vitamin D intake provides instant feedback, which may be more beneficial than education alone. The current study
suggests that prior knowledge or knowledge gained in higher education does not impact intake of dietary vitamin D.

The relationship between total dietary vitamin D, inclusive of supplementation, and mood was explored in this study. This study’s results did not support a predictive relationship of vitamin D intake on mood. However, there was not a normal distribution of total vitamin D intake, and therefore a relationship may exist in populations where the intake of participants is evenly distributed and varied. This study also did not find evidence to support the hypothesis of individuals consuming under the EAR having decreased mood scores in comparison to those consuming over the EAR. This relationship was also explored using RDA values in place of EAR, however, no significant results were found. These results do not support the findings of Bertone-Johnson et al. (2011), who reported that higher intake of vitamin D from dietary sources provided a significant 20% lower risk of depression. That study, however, was conducted with 81,189 postmenopausal women and compared individuals consuming less than 2.5 µg to individuals consuming 20 µg or more of vitamin D. Bertone-Johnson et al. (2011) reported a mean vitamin D intake, including supplementation, to be 10 µg. As a comparison, the current study had a median vitamin D intake of 4.11 µg with skewed distribution of intake, hinting that Bertone-Johnson et al. (2011) having a more normal distribution to their intake data.

This study also adds to the data assessing depression prevalence on college campuses, finding that 21.9% of students scored a 10 or higher on the PHQ-9 indicating possible moderate to severe depression. When individuals who reported being treated or being diagnosed with a mental illness were filtered out, that number remained prevalent at 14.3%. This number represents students potentially living with clinically diagnosable moderate depression, and not having been evaluated nor treated. Moreover, 12.3% of students who scored more than a 0 on the
PHQ-9 (n = 128) stated that these depressive symptoms have made activities of daily living (i.e., work, school, social life) very to extremely difficult.

In conclusion, undergraduate students at the University of Wisconsin-Stout are not consuming sufficient intake of vitamin D to support normal serum vitamin D concentrations. Supplementation aided in increasing the mean intake and number of individuals who consumed above the RDA. However, those who did not report consuming a supplement were on average consuming less than the EAR. Therefore, the present research supports the benefits of supplementation as a means to achieve adequate vitamin D intake during winter months for students at the University of Wisconsin-Stout. The results of the present research do not support the assumption that increased knowledge is associated with increased intake or supplementation of vitamin D. Moreover, the present study found an inverse predictive relationship between intake and knowledge. Additionally, while no significant findings were seen between intake and mood, the skewed data towards low intake may have allowed for a relationship to be shown.

Limitations

There are several limitations to this research study. First, the study population is not representative of all undergraduate students. The University of Wisconsin-Stout is located in Menomonie, Wisconsin and is not a racially or ethnically diverse campus. The study population reported 89.7% Caucasian/White/Non-Hispanic, 3.2% Hispanic/Latina/o, 0.6% Black/African American, 3.9% Asian/Pacific Islander, 0.6% Middle Eastern, 1.9% “other”. While the demographics are representative of the University of Wisconsin-Stout’s overall campus, they are not representative of the national undergraduate population. According to the National Center for Education Statistics (2015), the 2014 fall enrollment demographics for undergraduate students was: 55.4% white, 14.0% Black/African American, 17.1% Hispanic/Latino/a, 6.2% Asian/
Pacific Islander, 0.8% American Indian/Alaska Native, 3.4% two or more identities, and 3.0% non-resident. Biological sex was also heavily skewed, as our study population was 72.3% female. This contrasts to the campus demographic of 45% female students and the National Center for Education Statistics (2015) data on undergraduates which report 56.1% are female. Additionally, students with a higher interest in health or nutrition may have been more inclined to participate in this survey.

This research study also used self-reported dietary assessment, so there is potential for under- or over-reporting intake. An additional limitation is the dietary intake questionnaire itself, as the survey was validated in postmenopausal women and using an in-person survey format (Hacker-Thompson et al., 2012). Due to time and staffing limits, this study was not able to conduct in person interviews, which could have resulted in more accurate dietary recalls. Also, the use of dietary intake as a surrogate indicator of vitamin D status rather than actual serum concentrations is a significant limitation, and prevents true assessment of true vitamin D sufficiency or deficiency. While it is assumed that at higher latitudes during the winter that dietary intake of vitamin D is the main determinant of vitamin D status, the present study did not measure vitamin D status.

**Recommendations**

The current study found that only 98.7% of student participants at the University of Wisconsin-Stout were unable to meet the RDA for vitamin D from dietary sources alone. After supplements were included, 77.4% of students were still not meeting the RDA. During winter months, vitamin D synthesis from UVB radiation is negligible in northern latitudes (Webb et al., 1988). Therefore, obtaining adequate vitamin D through dietary sources and supplementation is increasingly important. Given the difficulty of reaching adequate intake of vitamin D from dairy
milk, cheese, salmon, and fortified cereals, the role of supplements and possibly also of increasing fortification of other foods, needs to be further investigated.

This research also found that greater vitamin-D related knowledge was not associated with increased dietary intake nor supplement use. These results indicate that factual knowledge alone may not be sufficient to improve vitamin D intake. Therefore, future studies should evaluate whether educational interventions are actually effective at increasing vitamin D intake, as well as whether different approaches, such as interactive feedback via mobile applications, would be more useful.

While this research does not support the hypothesis that there is a relationship between vitamin D intake and depressive symptoms, a large majority of the participants were consuming very little vitamin D, which may not have allowed for such a relationship to be seen. Other studies in college students have also found high levels of inadequate intake (Cress, 2014; Ouellette et al., 2012; Forney et al., 2014; Hilliard et al., 2015; Lacey et al., 2004). Therefore, future correlational studies may also have difficulties in finding normally distributed vitamin D intake. Thus, in order to compensate for the low baseline vitamin D status to further investigate a relationship between vitamin D and depressive symptoms, the researcher recommends a randomized, placebo-controlled, double-blind trial of the efficacy of vitamin D supplementation on depressive symptoms of college students in northern latitudes during the winter months.

Finally, this study found that at the University of Wisconsin-Stout 21.9% of students surveyed experience moderate to severe depressive symptoms, with 47% of those individuals, or 14.3% of the total number, not being currently evaluated or treated. Furthermore, of the 15 students reporting severe depressive symptoms (PHQ-9 ≥ 15), 30% were also not being currently evaluated or treated. Given that some of the barriers to seeking help for depressive symptoms
can be difficult to overcome, such as lack of time, financial concerns, no insurance, or lack of emotional openness (Givens & Tjia, 2002; Komiya et al., 2000; Megivern et al., 2003; Mowbray et al., 2006; Tjia et al., 2005), a nutritional intervention that could alleviate depressive symptoms would be highly beneficial. As vitamin D has been shown to alleviate depressive symptoms at safe and well-tolerated doses in meta-analyses (Shaffer et al., 2014; Spedding, 2014), the researcher again recommends further investigation into the possible treatment effect of vitamin D on depressive symptoms.
References


Food Labeling, 21 e.C.F.R. §101.72 (2017)


Givens, J. L., & Tjia, J. (2002). Depressed medical students' use of mental health services and barriers to use. *Academic Medicine, 77*(9), 918-921.


Goebert, D., Thompson, D., Takeshita, J., Beach, C., Bryson, P., Ephgrave, K., ... & Tate, J. (2009). Depressive symptoms in medical students and residents: A multischool study. *Academic Medicine, 84*(2), 236-241.


Högberg, G., Gustafsson, S. A., Hällström, T., Gustafsson, T., Klawitter, B., & Petersson, M. (2012). Depressed adolescents in a case-series were low in vitamin D and depression was ameliorated by vitamin D supplementation. *Acta Paediatrica, 101*(7), 779-783.


1. *Endocrinology, 141*(6), 2159-2165.
Appendix A: University of Wisconsin-Stout IRB Approval

January 31, 2017

Heidi Lochen
MS Food & Nutritional Science
University of Wisconsin-Stout

RE: Vitamin D Intake in Relationship to Mood and to Vitamin D-related Knowledge in Undergraduate Students

Dear Heidi:

The IRB has determined your project, “Vitamin D Intake in Relationship to Mood and to Vitamin D-related Knowledge in Undergraduate Students”, is Exempt from review by the Institutional Review Board for the Protection of Human Subjects. The project is exempt under Category #2 of the Federal Exempt Guidelines and holds for 5 years. Your project is approved from January 31, 2017 through January 30, 2022. If a renewal is needed, it is to be submitted at least 10 working days prior to the approvals end date. Should you need to make modifications to your protocol or informed consent forms that do not fall within the exemption categories, you will need to reapply to the IRB for review of your modified study.

Informed Consent: All UW-Stout faculty, staff, and students conducting human subjects’ research under an approved “exempt” category are still ethically bound to follow the basic ethical principles of the Belmont Report: 1) respect for persons; 2) beneficence; and 3) justice. These three principles are best reflected in the practice of obtaining informed consent from participants.

If you are doing any research in which you are paying human subjects to participate, a specific payment procedure must be followed. Instructions and form for the payment procedure can be found at http://www.uwstout.edu/rs/paymentofhumanresearchsubjects.cfm

If you have questions, please contact the IRB office at 715-232-1126, or buchanane@uwstout.edu, and your question will be directed to the appropriate person. I wish you well in completing your study.

Sincerely,

Elizabeth Buchanan
Interim Director of Office of Research and Sponsored Programs and Human Protections Administrator, UW-Stout Institutional Review Board for the Protection of Human Subjects in Research (IRB)

CC: Dr. Alexandra Hall
Appendix B: Permission to Use Brief Vitamin D Questionnaire

Email received on November 4\textsuperscript{th}, 2016:

From eat2perform@gmail.com

To: Lochen, Heidi

Heidi,

I have attached the vitamin D questionnaire that we validated. If you do use it in your project I just request that it is referenced. The questionnaire shows the portion size for a serving and you record the number of servings consumed per week. If you have any further questions please let me know.

Andrea

Email sent on October 11\textsuperscript{th}, 2016

From: Lochen, Heidi

To: Hacker, Andrea

Hello,

I am conducting research at the University of Wisconsin-Stout examining the dietary intake of vitamin D in college students during the winter months and correlating it with mood scores. I read your research on the Validation of the BVDQ and I was very interested in the possibility of modifying it for my study as I am currently looking for ways to measure intake. Please let me know, I appreciate your time in the matter.

Thank you,

Heidi Lochen
Appendix C: Permission to Use Modified Vitamin D Intake Questionnaire

Email received on December 7th, 2016
From: CRESS@mail.etsu.edu
To: Lochen, Heidi
Dear Ms. Lochen,
You have my permission to use a similar survey. Please note, the original validated survey is referenced in my dissertation. I modified to remove the calcium foods as I was not looking at that variable for the purposes of my research. The serving amount was included for each food item on my survey and I calculated total intake of vitamin D using values obtained from Nutritionist Pro Software Program.
Good luck with your research. I would love to know your results.
Kind Regards,
Dr. Cress

Email sent on Wednesday December 7th, 2016
From: Lochen, Heidi
To: Cress, Eileen M.
Dear Dr. Cress,
I will be conducting research examining vitamin D intake of college students during the winter months at the University of Wisconsin-Stout for thesis completion. I read your dissertation, *Vitamin D Status of College Students: Implications for Health Leaders* and found it to be exceptional. I would like to request your permission to use a similar survey for the exploration of my research question in my thesis with proper citation? I am also interested to know the method of determining intake amount from the survey as well. I appreciate you time in the matter.
Thank you,
Heidi Lochen
## Appendix D: Dietary Vitamin D Intake Questionnaire

<table>
<thead>
<tr>
<th>Food</th>
<th>Serving Size</th>
<th>Amount of vitamin D per serving</th>
<th>Less than once per month</th>
<th>Twice per month</th>
<th>Once per week</th>
<th>2-3 times per week</th>
<th>4-6 times per week</th>
<th>Once per day</th>
<th>Twice per day</th>
<th>Three or more times per day</th>
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</thead>
<tbody>
<tr>
<td>Dairy Milk</td>
<td>1 cup</td>
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<tr>
<td>Soy Milk</td>
<td>1 cup</td>
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<tr>
<td>Almond Milk</td>
<td>1 cup</td>
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<tr>
<td>Rice Milk</td>
<td>1 cup</td>
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<tr>
<td>Vitamin D Fortified Orange Juice</td>
<td>1 cup</td>
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<tr>
<td>Milk or Cream</td>
<td>1 Tablespoon</td>
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<td>Fortified Breakfast Cereal</td>
<td>1 cup</td>
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<td>Ravioli</td>
<td>1 cup</td>
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<tr>
<td>Lasagna</td>
<td>1 cup</td>
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<tr>
<td>Macaroni and Cheese</td>
<td>1 cup</td>
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<td>Yogurt, non-greek, fortified (Breyers, Dannon, Stonyfield, Yoplait)</td>
<td>1/2 cup</td>
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<tr>
<td>Ice Cream</td>
<td>1/2 cup</td>
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<tr>
<td>Mushrooms, raw</td>
<td>1/2 cup</td>
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<tr>
<td>American or Mozzarella Cheese</td>
<td>1 ounce</td>
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<tr>
<td>Hard Cheese (Cheddar, Swiss, Provolone, Parmesan)</td>
<td>1 ounce</td>
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<td>Lean Cuts of Beef (strip loin, round roast, steak etc.)</td>
<td>3 ounces</td>
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<tr>
<td>Hamburger</td>
<td>3 ounces</td>
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<tr>
<td>Cheeseburger</td>
<td>3 ounces</td>
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<tr>
<td>Salmon</td>
<td>3 ounces</td>
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<td>Whitefish (cod, flounder), or</td>
<td>3 ounces</td>
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<td>Ingredient</td>
<td>Quantity</td>
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<td>Herring (Atlantic or pickled)</td>
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<td>Mackeral (Atlantic, Pacific, canned)</td>
<td>3 ounces</td>
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<td>Shrimp</td>
<td>3 ounces</td>
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<td>Egg</td>
<td>1 whole, or yolk</td>
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<td>Hot Dog (pork or beef)</td>
<td>1 each</td>
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<td>Cod Liver Oil</td>
<td>2 tablespoons</td>
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<tr>
<td>Butter or Margarine</td>
<td>1 tablespoon</td>
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<tr>
<td>Sardines</td>
<td>2 each</td>
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</table>
Appendix E: Permission to Use Modified Knowledge Questionnaire

Email received on September 19th, 2016

From: jenirwin@uwo.ca
To: Lochen, Heidi

Dear Heidi,

Yes, I don’t see any concerns with you using our tool. I can’t remember off-hand if we published it with the article. If not, you can find it as an appendix in my graduate student’s thesis: http://ir.lib.uwo.ca/cgi/viewcontent.cgi?article=2918&context=etd

Best wishes on your work. JI

Email sent on September 19th, 2016

From: Lochen, Heidi
To: Irwin, Jennifer

Hello Dr. Irwin,

My name is Heidi Lochen and I am a second-year graduate student at the University of Wisconsin-Stout completing my masters in Food and Nutritional Science. I am conducting research this spring semester dealing with Vitamin D Consumption and Knowledge in university students during the winter months. I had read your paper published in the *Journal of Nutrition Education and Behavior* and would like to do a continuation off of your research. With your permission and cooperation, I would be interested in using the questions that you developed to assess vitamin D knowledge in my graduate thesis as a guide with proper acknowledgement and citation. Please let me know if this would be at all possible. I appreciate your time and attention.

Thank you,

Heidi Lochen
Appendix F: Vitamin D-Related Knowledge Questionnaire

1. Where do you think vitamin D comes from? (check all that are correct)
   a. Fruits
   b. Vegetables
   c. Water
   d. Fatty fish*
   e. Sun*
   f. Air
   g. Fortified cereals*
   h. Milk/dairy*
   i. Nuts
   j. Cod liver oil*
   k. Chicken
   l. Eggs*
   m. Mushrooms*

2. Vitamin D is important for which of the following? (select all that apply)
   a. Bone health*
   b. Heart health
   c. Cancer prevention
   d. Vision
   e. Wound healing
   f. Blood clotting
   g. None of the above
3. What is the recommended daily allowance (RDA) of vitamin D for adults (aged 19-50)?
   a. 200 International Units (IU)
   b. 400 IU
   c. 600 IU*
   d. 800 IU
   e. 1,000 IU
   f. 2,000 IU

4. How much time would the average fair-skinned (i.e., Caucasian) person need to spend in the sun during the summer to get enough vitamin D, if their bare legs and arms were exposed?
   a. Less than 10 minutes per week
   b. About 10-60 minutes a week*
   c. About 1-6 hours per week
   d. More than 6 hours per week

5. What factors that can decrease the amount of vitamin D a person can get? (Select all that apply)
   a. Skin pigmentation (skin color)*
   b. Shade or clouds*
   c. Time of day*
   d. Latitude*
   e. Season*
   f. Age*
   g. Fatty diets
h. Sunscreen use*

i. Vegan/vegetarian diets*

j. Lactose intolerance*

k. Dairy allergy*

l. Pollution*

m. Wind

n. Smoking

o. Body mass index (BMI)*

p. Other (please specify__)

6. What percentage of the United States population is estimated to be vitamin D insufficient (i.e., has less vitamin D in their body than recommended)?
   a. 1%
   b. 22%
   c. 42%*
   d. 98%

*Denotes correct responses

Scoring:
**Question 1: Food Sources** (add 0.077 for each correct selection, deduct 0.077 points for each incorrect selection) Total= 1 point

**Question 2: Health Benefits** (add .143 for each correct selection, deduct 0.143 points for each incorrect selection) Total= 1 point

**Question 3, 4, and 6: Recommendations** (add 0.334 for the correct selection for each question) Total= 1 point

**Question 5: Barriers** (add 0.0667 for each correct selection, deduct 0.0667 for each incorrect selection, “other” is not included in scoring) Total= 1 point
Appendix G: Patient Health Questionnaire-9

Over the last 2 weeks, how often have you been bothered by any of the following problems?

<table>
<thead>
<tr>
<th>Problem</th>
<th>Not at all</th>
<th>Several Days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Little interest or pleasure in doing things</td>
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<tr>
<td>Feeling down, depressed, or hopeless</td>
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<tr>
<td>Trouble falling asleep or staying asleep, or sleeping too much</td>
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<tr>
<td>Poor appetite or overeating</td>
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<tr>
<td>Feeling bad about yourself- or that you are a failure or have let yourself or your family down</td>
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<tr>
<td>Trouble concentrating on things, such as reading the newspaper or watching television</td>
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<tr>
<td>Moving or speaking so slowly that other people could have noticed? Or the opposite- being so fidgety or restless that you have been moving around a lot more than usual</td>
<td></td>
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<tr>
<td>Thoughts that you would be better off dead or of hurting yourself in some way</td>
<td></td>
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</tr>
</tbody>
</table>

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?

<table>
<thead>
<tr>
<th>Difficulty</th>
<th>Not difficult at all</th>
<th>Somewhat difficult</th>
<th>Very difficult</th>
<th>Extremely difficult</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>□</td>
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<td>□</td>
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</tbody>
</table>

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