Diet Quality and Mental Health Amongst Acute Inpatient Psychiatric Patients

Raveena Gill (✉ gillraveena@gmail.com)  
SUNY Upstate Medical University  https://orcid.org/0000-0001-5618-3625

Sean F Tyndall  
State University of New York Upstate Medical University

Darshini Vora  
Zucker Hillside Hospital

Rashedul Hassan  
State University of New York Upstate Medical University

James Megna  
State University of New York Upstate Medical University

Luba Leontieva  
State University of New York Upstate Medical University

Research article

Keywords: KIDMed, population, mental

DOI: https://doi.org/10.21203/rs.3.rs-41750/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License
Abstract

**Background:** Poor diet quality has been found to be prevalent in people with mental illness [71] and even contribute to mental illness [71]. The purpose of this exploratory study is to examine the relationship between diet quality, mental health problems, socioeconomic variables, physical activity level, and body mass index amongst acute psychiatric inpatients.

**Method:** The study sample were 100 adult inpatients. Tools administered were Eating Habit Questionnaires (Dana Farber Institute) and the KIDMed index to assess food compliance in accordance with a Mediterranean diet. The Index marks all individuals as either having an “unhealthy score” or “healthy score”. The study utilized a descriptive correlation research design.

**Results:** The results showed that 75% of the patients displayed an unhealthy score as per the KIDMed index. The findings displayed an inverse relationship of BMI and the KIDMed score. There was a significant positive correlation between the level of education and the KIDMed score. There is a positive correlation towards increased consumption of sugar in schizophrenic patients. There was no statistical significance between age, gender, income and physical activity with regards to the KIDMed score.

**Conclusion:** Psychiatric inpatients display unhealthy eating habits which is likely both an effect of and a contributing factor to the various mental illnesses. Prospective interventional randomized controlled studies are necessary for clarification of cause-effect relationships.

**Background**

Essential vitamins, minerals, and omega-3 fatty acids are often deficient in the general population in America and other developed countries; and are exceptionally deficient in patients suffering from mental disorders [71]. Studies have shown that daily supplements of vital nutrients often effectively reduce patients’ symptoms [72]. Supplements that contain amino acids also reduce symptoms, because they are converted to neurotransmitters that alleviate depression and other mental disorders. Based on emerging scientific evidence, this form of nutritional supplement treatment may be appropriate for controlling major depression, bipolar disorder, schizophrenia and anxiety disorders, eating disorders, attention deficit disorder/attention deficit hyperactivity disorder (ADD/ADHD), addiction, and autism [13]. In the last few years there have been a number of studies identifying an inverse association between diet quality and the common mental disorders, depression and anxiety, in adults [1], [2], [3] and two prospective studies suggesting that diet quality influences the risk for depressive illness in adults over time [4], [5].

Mood disorders (i.e. depression, bipolar condition) are very common on acute psychiatric units [73]. Depression has for some time now been known to be associated with deficiencies in neurotransmitters such as serotonin, dopamine, noradrenaline, and GABA [14-20]. As reported in several studies, the amino acid tryptophan, tyrosine, phenylalanine, and methionine are often helpful treating many mood disorders, including depression [21-25]. Tryptophan is a precursor to serotonin and is usually converted to serotonin.
when taken alone on an empty stomach. Therefore, tryptophan can induce sleep and tranquility and in cases of serotonin deficiencies, restore serotonin levels leading to diminished depression [26, 23].

Since the consumption of omega-3 fatty acids from fish and other sources has declined in most populations, the incidence of major depression has increased [27]. Several mechanisms of action may explain how eicosapentaenoic acid (EPA) which the body converts into docosahexaenoic acid (DHA), the two omega-3 fatty acids found in fish oil, elicit antidepressant effects in humans. Most of the proposed mechanisms involve neurotransmitters and, of course, some have more supporting data than others. For example, antidepressant effects may be due to EPA being converted into prostaglandins, leukotrienes, and other chemicals the brain needs [13].

While psychological stress is known to increase the pro-inflammatory cytokines, the relationship appears to be bi-directional, with inflammation suggested as a direct contributor to the risk for depressive illness [6]. Inflammation is accompanied by an accumulation of highly reactive oxygen species, and increased oxidative stress is also implicated as a factor in depressive illnesses [7]. Consumption of diets rich in antioxidants, vitamins, minerals and fiber is associated with reduced systemic inflammation [8]. Conversely, diets that are low in essential nutrients, such as magnesium and western type dietary patterns [9] are associated with increased systemic inflammation.

An important aspect of the shift in habitual diets globally is that of an increase in refined carbohydrate consumption. Hyperglycemia promotes an inflammatory state and high glycemic load (GL) diets are also associated with increased systemic inflammation [10]. Dietary factors, such as refined sugars and saturated fats, have a detrimental impact on the expression of neurotrophic factors [11] that are particularly salient to depressive illness. Thus, it is plausible to speculate that, by modifying inflammatory, oxidative, and neurotrophic factors, diet quality influences the genesis and progression of depressive illnesses. These hypotheses remain to be tested [12]. These hypotheses however remain largely untested [12].

The most consistent correlation found in one study that involved the ecological analysis of schizophrenia and diet concluded that increased consumption of refined sugar results in a lack of substantial improvement in patients with schizophrenia, as measured by both the number of days spent in the hospital and poor social functioning [37]. In the study performed there was a very strong association with consumption of sugar and increased mean hospital time and worsening of social functioning based upon two calculative scores with a Pearson coefficient of 0.94 and 0.89 respectively. That study also concluded that the dietary predictors of the outcome of schizophrenia and prevalence of depression are similar to those that predict illnesses such as coronary heart disease and diabetes [13]. There are preliminary data from RCTs suggesting that anti-inflammatory nutrients, such as omega-3 and folate-based compounds, may also be effective for other SMIIs, including bipolar disorder and schizophrenia [43]. Because inflammation is particularly elevated during onset of psychotic disorders, these adjunctive treatments may have neuroprotective effects in the early stages of illness among young people [44] [45].
potentially improving cognitive outcomes for some patients. However, the extent to which their effects are due specifically to their anti-inflammatory properties is not fully ascertained [46].

Mediterranean dietary patterns are comprised of: abundant plant foods (fruits, vegetables, breads, other forms of cereals, pulses, nuts and seeds); minimally processed, seasonally fresh and locally grown foods; fresh fruits as the typical daily dessert with sweets elaborated from nuts, olive oil and concentrated sugars or honey that are consumed during feast days; olive oil as the principal source of dietary lipids; dairy products (mainly cheese and yoghurt) consumed in low to moderate amounts; fewer than four eggs consumed per week; red meat consumed in low frequency and amounts; wine consumed in low to moderate amounts, and generally taken with meals. Such a dietary pattern assures a sufficient intake of certain nutrients that have been related in some way with a reduced risk of several chronic diseases. Various scores or indexes have been developed to assess the adherence to the Mediterranean diet pattern in the population and to link such patterns with several nutrient-related diseases [47].

A trial conducted in clinically depressed participants [41] observed large reductions in depressive symptoms from a 12-week modified Mediterranean diet with 32.3% of participants achieving remission from dietary intervention versus 8.0% in the social support control condition (p= 0.028) Subsequent RCTs have replicated these findings of the Mediterranean diet reducing symptoms in people with moderate to severe depression [42]. As a meta-analysis of 50 studies has shown, the Mediterranean diet significantly reduces inflammatory markers in other (i.e., non-psychiatric) populations, and it is possible that the benefits in people with depression are linked to the anti-inflammatory effects.

Changing established dietary behaviors is challenging, and this is attributed to factors such an obesogenic environment [52] and the addictive nature of high-fat high-sugar foods [53]. However, there is evidence that neural reward thresholds can be changed in favor of preferring healthy over unhealthy food [54]. A Mediterranean diet not only has demonstrated health benefits but is also a highly palatable diet and thus more likely to become a sustainable part of a healthy lifestyle [55].

As seen above, the role of diet plays a crucial role contributing to changes in mental health. This is an aspect less addressed by researchers [50] and further not widely acknowledged by physicians. Thus, the aim of this study is to increase the data pool to answer the question whether dietary patterns have any effect on mental health, particularly the acute psychiatric inpatients. This study seeks to contribute to the growing school of thought that a holistic approach is required for psychiatric treatment, especially healthier diet control.

**Study Design And Methodology**

The study was conducted on an acute inpatient psychiatric unit of an academic hospital. The study was approved by the institutional review board. 100 non-consecutive inpatients were approached by the researchers and after their verbal agreement the Eating Habits Questionnaire (Dana Fiber Institute) [56] was administered. The questionnaire was employed owing to its concise set of questions addressing major food groups and multiple options for recording frequency of consumption. The questionnaire also
entailed some basic questions regarding demographic characteristics of the study population. Explanation was offered regarding the purpose of the study and confidentiality was ensured.

The KIDMED questionnaire was used to evaluate the adherence to a Mediterranean diet in adolescents. It consists of 16 items, where there are 4 questions denoting a negative connotation to the Mediterranean diet (consumption of fast food, baked goods, sweets, and skipping breakfast) and 12 questions denoting a positive connotation (consumption of oil, fish, fruits, vegetables, cereals, nuts, pulses, pasta or rice, dairy products, and yoghurt). Questions denoting negative connotation are scored with −1, while positive connotation questions are scored with +1. According to the KIDMED index, a score of 0–3 reflects poor adherence to the Mediterranean diet, a score of 4–7 describes average adherence, and a score of 8–12 good adherence. However, the score was modified as “healthy” with a score of over 4 and “unhealthy” as 3 and under [48].

The study was conducted as a descriptive, cross-sectional, correlational design, to examine the relationship between KIDMed score index (a measure of healthy dietary habits) and BMI, gender, level of education, smoking, income and exercise. The data was analyzed using SPSS. The Mediterranean diet index KIDMed was used to categorize all patients as having a healthy or an unhealthy score which corresponded to their dietary habits.

Results

Descriptive analysis using frequency and percentages were used to describe the demographic data of this study. Correlation analysis between the KIDMed score (dependent variable) and biosocial variables, age, gender, level of education, race, income, and physical activity (input variables) was done using Chi Square and Spearman's ranked univariate analysis. Further univariate analysis using ANOVA was utilized to evaluate for associations between KIDMed scores and Grouped Mental Health conditions. Our study did not correct for confounders and statistical significance in view of the exploratory nature of the study. We did not correct for multiple tests of statistical significance since our study is an exploratory one.

Sample Characteristics

Demographic characteristics of the surveyed population are shown in Table 1. The majority of respondents were between 18yrs. and 50yrs. old (65%) with a mean age of 43yrs. Fifty-five (55%) percent of the surveyed population were females. Overall, seventy-nine (79%) percent of those surveyed identified as Caucasian.

The mean body mass index (BMI) computed from weights and heights taken at the time of admission was 29.8. Sixty-two percent of those studied were classified as either overweight or obese, with 37% in the latter category.

Overall, less than one in two received as much as a high school diploma. Surprisingly, however, 39% reported having obtained a college or associate college degree.
There was an almost equal distribution between smokers and non-smokers, with the latter predominating at 54%.

Sixty-four percent (64%) of those studied admitted some level of physical exercises on a weekly basis, with nearly 50% exercising at least once weekly.

With reference to household income, 1 in 2 grossed less than $30,000 yearly (55%) as combined household earnings.

**Figure 1** shows that majority of the participants (75%) are engaged in poor eating habits according to the KIDMED score which is (the frequency distribution of Mediterranean diet)

**Correlations**

A positive correlation between Education level and KIDMed score (0.196) was found using Spearman's Ranked analysis. Conversely, a negative correlation coefficient (-0.124) was found between BMI and KIDMed score. Both findings yielded a statistical significance with p values of 0.05 and 0.04 respectively. (See Table 2)

Figure 1 reveals a graphic illustration of ‘Eating Habits” assessed using KIDMed Score.

Seventy-five percent of respondents were categorized as having a poor dietary pattern based on a KIDMed score no greater than 3. The study failed to show any statistical significance for correlations between age and KIDMed score (p value = 0.223) using Spearman's coefficient analysis. Similarly, no significance was found between KIDMed score (dependent variable) and gender, level of education, BMI, Income, and physical activity, using Chi Square analysis.

Figure 3 is a Bar chart/ Histogram of KIDMed score and BMI.

**Grouped mental health disorders and KIDMed score.**

Analysis for correlation between Grouped Mental Health conditions and KIDMed score using the univariate ANOVA test failed to show any statistically significant association (p value =0.463). Table 4 is an illustration of Grouped Mental Health conditions and the concomitant mean KIDMed score for each group. Overall, the mean KIDMed scores for all the groups were poor, and failed to identify any trends with an increasing number of concomitant mental health conditions (multiple diagnoses).

**Correlation Between Sugar consumption and psychosis.**

Significantly, the study revealed a strong positive correlation between sugar consumption (number of daily teaspoonfuls of added sugar) and Psychotic individuals using the Chi Square analysis (p value = 0.001). This result closely identifies with those found in existing literature showing an inverse relationship between increased sugar consumption and worsening psychosis in schizophrenic patients (66).
Discussion

The frequency and the mean scores in this study identified a majority of acute psychiatric inpatients exhibiting unhealthy dietary habits that are known to cause deficiencies in essential nutrients and minerals that can exacerbate mental illnesses. The study further highlights that the causes of mental health conditions are multifactorial, and not all biosocial factors show strong direct association. Confounders such as the concomitant administration of antipsychotic medications, known to cause metabolic disturbances, may also impact the role of diet directly [51]. Importantly, the apparent underreporting of alcohol consumption cannot be excluded from the overall accuracy and veracity of data collected. In our study 69 patients were diagnosed as having a substance use/abuse problem, while 70 claimed liquor consumption of less than once weekly. Although 57 admitted that they drank one bottle of beer 1–3 times per month, an accurate determination for other liquor consumption could not be made.

In a patient population with determined mental illnesses, the prevalence of a constant low mean KIDMed score (2.9) and covalently, poor eating standards, can be partially attributed to social impairment and diminished quality of self-care as a result of their mental illness.

Evidence exists that Substance abuse is rampant; as well as in our population (64% of population). Mean KIDMed scores are 2.9 and mean BMI is 29.8. In active addiction, Malnutrition occurs in 5–30% of the cases [58]. While in Recovery, substance abuse patients report concerns with unhealthy eating patterns, unhealthy weight gain and development of obesity. With weight gains of approximately 3 kilograms over 12 weeks common [59].

70% of the study population were suffering from depression, which is accompanied often by decreased psychomotor state and appetite changes. Thus, further contributing to poor eating habits in our patient population. The overlay of prominent mood disorder (70%) and poor eating habits (mean KIDMed of 2.9) can be attributed to the existing evidence of a bidirectional involvement of depression and poor diet [50]. Additionally, there is compelling evidence that regular exercise is protective against depression [60]. Our analysis comparing the mean levels of physical activity in depressive patients are seen as 58% of patients in our sample population with mood disorders “don’t exercise or exercise < 1 time / week”. Thereby imploring intervention of physical activity in the active treatment of depressive inpatients.

Unhealthy food and inactivity have been part of the culture of mental health treatment. The implicit acceptance of these practices is likely to be a result of practitioners seeing them as normal or as self-comfort strategies, with little clinical relevance. Snack food vending machines are common in inpatient mental health units, exercise programs are rare, and smoking areas are still commonplace [61]. Although mental health practitioners may not feel competent to provide advice on diet and exercise improvements, the evidence suggests that specific and detailed advice may not be necessary. Recommendations and encouragement to follow national guidelines for dietary and exercise practices should be part of care for all people with mental illness and especially depression [62].
75% of the overall population displayed an unhealthy diet based on their KIDMed scores. The results of the Chi square tests comparing Biosocial factors vs KIDMed scores revealed correlation between level of education, BMI and the KIDMed index. However, the other biosocial factors (age, gender, income and physical activity) did not yield significant results. (Refer to Table 2). Several factors served as limitations in the current study. I) The low sample size did not accommodate the derivation of correlations. II) The lack of a control group contributed towards not being able to draw effective correlations. III) The KIDMed index gives an estimated score ranging from −2 till 8. This scale, though standard for an effective implementation of a Mediterranean diet, is limited in the inclusion of the detailed variety of diet types in Dana-Farber Institute Nutritional Questionnaire. IV) The Mean KIDMed Score of our sample group is (2.9) and a median of (2.0). This prevents the sample size to be stratified effectively into healthy and unhealthy groups. V) Recall bias exhibited by the patients and overestimating the consumption of foods they understand to have higher nutritional value. VI) Methods for accurately measuring people's dietary intakes remain problematic. Diet quality is most often measured using a priori dietary quality index derived from recommended dietary guidelines or Mediterranean indices. They commonly identify two main dietary patterns that reflect dietary habits; often labeled as (Healthy) and (Unhealthy). An issue with employing a priori diet quality score, such as a Mediterranean-style dietary index is problematic in non-Mediterranean cultures, as in our study. However newer methods such as the use of DII [57] are less influenced by cultural contexts and need to be more widely applied. VII) Studies based on diet intake are fraught in general with limitations. Covariance between health behaviors such as diet, physical activity and smoking are all associated with depression in a bidirectional manner, as well as being correlated with each other. Understanding how each interacts with the other can complicate the interpretation of the results of observational studies [50].

Patients with Schizophrenia are known to have poor dietary habits. Forty to 62% of people with schizophrenia are obese or overweight [67, 68]. High morbidity and mortality in schizophrenia may be attributed to an unhealthy lifestyle such as poor diet, lack of exercise, smoking, and substance abuse [69]. Increased sugar and processed diet consumption are thought to be characteristic of people with schizophrenia. Moreover, multiple studies have shown that patients on clozapine have nearly twice the refined sugar consumption than the regular populous [70]. An ecological study using data from the WHO had shown negative outcomes over a two-year span in schizophrenic patients with high levels of refined sugar consumption [66]. These assumptions hold true even in our sample population. 55.1% of the patients with psychosis are obese or overweight, with a mean BMI of 27.36. There is marked increase in sugar consumption on a daily basis in our patients suffering with psychosis with a p value of 0.01.

**Conclusion/limitations**

The findings of this study established the prevalence of unhealthy eating habits in psychiatric inpatients. There is a significant association between ascending levels of education with improved diet, and of lower BMI and the better diet. While the remainder of biosocial factors did not yield significant associations, it may be attributed to low sample size and other confounding factors. Moreover, the study has shed light
upon the limitations in performing nutritional based studies on mentally ill patients and the need for further prospective/interventional randomized controlled trials.

Our finding suggests that improved dietary habits (e.g., Mediterranean diet) may contribute to more rapid symptoms resolution and acute stabilization on a short-stay inpatient unit.

**Abbreviations**

RCT- Randomized controlled trial

SMI - severe mental illness

BMI - body mass index

ANOVA- analysis of variance.

**Declarations**

**Ethical Approval and consent to participate:** Yes. It was approved by Institutional Review Board

**Consent for publication:** Given by each participant verbally before questionnaire. Participants were informed that their details are confidential and participation would not affect care provided.

**Availability of data and materials:** Data available on request from the author.

**Competing interests:** Not applicable.

**Funding:** Not applicable

**Authors’ contributions:** RG - data collection, analysis, paper writing; ST - study design, IRB submission, data collection, paper writing; DV- study design, IRB submission; RH - data collection, JM - study design, paper correction, LL - study design and oversight, paper correction. All authors have read and approved the manuscript.

**Acknowledgements:** we acknowledge patients and staff of the inpatient unit for support of this project

**Author information:** Raveena Gill. Upstate Medical University. gillrav@upstate.edu

**References**

1. Ballenger, J.c. “Association of Western and Traditional Diets with Depression and Anxiety in Women.” Yearbook of Psychiatry and Applied Mental Health, vol. 2011, 2011, pp. 315–316., doi:10.1016/j.yopsy.2010.10.069.
2. Nanri, A, et al. “Dietary Patterns and Depressive Symptoms among Japanese Men and Women.” *European Journal of Clinical Nutrition*, vol. 64, no. 8, 2010, pp. 832–839., doi:10.1038/ejcn.2010.86.

3. Jacka, Felice N., et al. “The Association Between Habitual Diet Quality and the Common Mental Disorders in Community-Dwelling Adults.” *Psychosomatic Medicine*, vol. 73, no. 6, 2011, pp. 483–490., doi:10.1097/psy.0b013e31822831a.

4. Sánchez-Villegas, Almudena, et al. “Association of the Mediterranean Dietary Pattern With the Incidence of Depression.” *Archives of General Psychiatry*, vol. 66, no. 10, 2009, pp. 1090., doi:10.1001/archgenpsychiatry.2009.129.

5. Akbaraly, Tasnime N., et al. “Dietary Pattern and Depressive Symptoms in Middle Age.” *British Journal of Psychiatry*, vol. 195, no. 5, 2009, pp. 408–413., doi:10.1192/bjp.bp.108.058925.

6. Pasco, Julie A., et al. “Association of High-Sensitivity C-Reactive Protein with De Novo Major Depression.” *British Journal of Psychiatry*, vol. 197, no. 5, 2010, pp. 372–377., doi:10.1192/bjp.bp.109.076430.

7. Ng, Felicity, et al. “ChemInform Abstract: Oxidative Stress in Psychiatric Disorders: Evidence Base and Therapeutic Implications.” *ChemInform*, vol. 40, no. 22, 2009, doi:10.1002/chin.200922265.

8. Chrysohoou, Christina, et al. “Adherence to the Mediterranean Diet Attenuates Inflammation and Coagulation Process in Healthy Adults.” *Journal of the American College of Cardiology*, vol. 44, no. 1, 2004, pp. 152–158., doi:10.1016/j.jacc.2004.03.039.

9. King, Dana E., et al. “Dietary Magnesium and C-Reactive Protein Levels.” *Journal of the American College of Nutrition*, vol. 24, no. 3, 2005, pp. 166–171., doi:10.1080/07315724.2005.10719461.

10. Lopez-Garcia, Esther, et al. “Major Dietary Patterns Are Related to Plasma Concentrations of Markers of Inflammation and Endothelial Dysfunction.” *The American Journal of Clinical Nutrition*, vol. 80, no. 4, 2004, pp. 1029–1035., doi:10.1093/ajcn/80.4.1029.

11. Liu, Simin, et al. “Relation between a Diet with a High Glycemic Load and Plasma Concentrations of High-Sensitivity C-Reactive Protein in Middle-Aged Women.” *The American Journal of Clinical Nutrition*, vol. 75, no. 3, 2002, pp. 492–498., doi:10.1093/ajcn/75.3.492.

12. Molteni, R, et al. “A High-Fat, Refined Sugar Diet Reduces Hippocampal Brain-Derived Neurotrophic Factor, Neuronal Plasticity, and Learning.” *Neuroscience*, vol. 112, no. 4, 2002, pp. 803–814., doi:10.1016/s0306-4522(02)001239.

13. Lakhan, Shaheen E, and Karen F Vieira. “Nutritional Therapies for Mental Disorders.” *Nutrition Journal*, vol. 7, no. 1, 2008, doi:10.1186/1475-2891-7-2.

14. National Institute of Mental Health Depression. National Institute of Mental Health, National Institutes of Health. 2000. US Department of Health and Human Services, Bethesda(MD) [Reprinted September 2002]

15. Rush, A John. “The Varied Clinical Presentations of Major Depressive Disorder.” *The Journal of Clinical Psychiatry*, U.S. National Library of Medicine, 2007, ncbi.nlm.nih.gov/pubmed/17640152.

16. Stockmeier, Craig A. “Neurobiology of Serotonin in Depression and Suicide.” *Annals of the New York Academy of Sciences*, vol. 836, no. 1 Neurobiology, 1997, pp. 220–232., doi:10.1111/j.1749-
17. Praag, H.m. Van. “Depression, Suicide and the Metabolism of Serotonin in the Brain.” *Journal of Affective Disorders*, vol. 4, no. 4, 1982, pp. 275–290., doi:10.1016/0165-0327(82)90025-8.
18. Diehl, David J., and Samuel Gershon. “The Role of Dopamine in Mood Disorders.” *Comprehensive Psychiatry*, vol. 33, no. 2, 1992, pp. 115–120., doi:10.1016/0010-440x(92)90007-d.
19. Firk, Christine, and C. Rob Markus. “Review: Serotonin by Stress Interaction: a Susceptibility Factor for the Development of Depression?” *Journal of Psychopharmacology*, vol. 21, no. 5, 2006, pp. 538–544., doi:10.1177/0269881106075588.
20. Briley, Mike, and Chantal Moret. “The Role of Noradrenaline in Depression and Its Therapy.” *Neurochemistry*, 1997, pp. 193–196., doi:10.1007/978-1-4615-5405-9_32.
21. Petty, Frederick. “GABA and Mood Disorders: a Brief Review and Hypothesis.” *Journal of Affective Disorders*, vol. 34, no. 4, 1995, pp. 275–281., doi:10.1016/0165-0327(95)00025-i.
22. Mclean, Andrew, et al. “The Effects of Tyrosine Depletion in Normal Healthy Volunteers: Implications for Unipolar Depression.” *Psychopharmacology*, vol. 171, no. 3, 2004, pp. 286–297., doi:10.1007/s00213-003-1586-8.
23. Agnoli, A., et al. “Effect of S-Adenosyl-L-Methionine (SAMe) upon Depressive Symptoms." *Journal of Psychiatric Research*, vol. 13, no. 1, 1976, pp. 43–54., doi:10.1016/0022-3956(76)90008-x.
24. Young, Simon N., et al. “The Effect of Tryptophan on Quarrelsomeness, Agreeableness, and Mood in Everyday Life." *International Congress Series*, vol. 1304, 2007, pp. 133–143., doi:10.1016/j.ics.2007.07.037.
25. Hoes MJ. L-Tryptophan in Depression. Journal of Orthomolecular Psychiatry. 1982;4:231.
26. Buist R. The therapeutic predictability of Tryptophan and Tyrosine in the treatment of Depression. Int J Clinical Nutritional Review. 1983;3:1-3.
27. Hibbeln JR. Fish Consumption and major depression. The Lancet. 1998; 351;1213.
28. Skutsch, Gilliam M. “Manic Depression — A Disorder of Central Dopaminergic Rhythm.” *Medical Hypotheses*, vol. 7, no. 6, 1981, pp. 737–746., doi:10.1016/0306-9877(81)90085-2.
29. Skutsch, Gilliam M. “Manic Depression: A Multiple Hormone Disorder?” *Biological Psychiatry*, vol. 20, no. 6, 1985, pp. 662–668., doi:10.1016/0006-3223(85)90101-5.
30. Naylor, Graham J. “Vanadium and Manic Depressive Psychosis.” *Nutrition and Health*, vol. 3, no. 1-2, 1984, pp. 79–85., doi:10.1177/02601068400300206.
31. Naylor, Graham J., and Anne H. W. Smith. “Vanadium: a Possible Aetiological Factor in Manic Depressive Illness.” *Psychological Medicine*, vol. 11, no. 2, 1981, pp. 249–256., doi:10.1017/s0033291700052065.
32. Castle, David, et al. “The Incidence of Operationally Defined Schizophrenia in Camberwell, 1965–84.” *British Journal of Psychiatry*, vol. 159, no. 6, 1991, pp. 790–794., doi:10.1192/bjp.159.6.790.
33. M. M. A. Van Der Heijden, et al. “Amino Acids in Schizophrenia: Evidence for Lower Tryptophan Availability during Treatment with Atypical Antipsychotics?” *Journal of Neural Transmission*, vol.
112, no. 4, 2004, pp. 577–585., doi:10.1007/s00702-004-0200-5.

34. “Amelioration of Negative Symptoms in Schizophrenia by Glycine.” American Journal of Psychiatry, vol. 151, no. 8, 1994, pp. 1234–1236., doi:10.1176/ajp.151.8.1234.

35. Leiderman, Eduardo, et al. “Preliminary Investigation of High-Dose Oral Glycine on Serum Levels and Negative Symptoms in Schizophrenia: an Open-Label Trial.” Biological Psychiatry, vol. 39, no. 3, 1996, pp. 213–215., doi:10.1016/0006-3223(95)00585-4.

36. Javitt, Daniel C., et al. “Adjunctive High-Dose Glycine in the Treatment of Schizophrenia.” The International Journal of Neuropsychopharmacology, vol. 4, no. 04, 2001, doi:10.1017/s1461145701002590.

37. Peet M. International Variations in the Outcome of Schizophrenia and The Prevalence of Depression in Relation to National Dietary Practices: An Ecological Analysis. British Journal of Psychiatry 2004.

38. American Psychiatric A. Quick Reference to the Diagnostic Criteria From DSM IV-TR. Arlington, VA. 2000.

39. Fontenelle LF, Nascimento AL, Mendlowicz MV, Shavitt RG, Versiani M. An update on the pharmacological treatment of obsessive-compulsive disorder. Expert Opin Pharmacother. 2007;8:563–583. doi: 10.1517/14656566.8.5.563

40. Yaryura-Tobias JA, Bhagavan HN. L-tryptophan in obsessive-compulsive disorders. Am J Psychiatry. 1977;134:1298–1299.

41. Jacka FN, O'Neil A, Opie R, Itsiopoulos C, Cotton S, Mohebbi M, et al. A randomised controlled trial of dietary improvement for adults with major depression (the ‘SMILES’ trial). BMC Med (2017) 15(1):23. doi: 10.1186/s12916-017-0791-y

42. Parletta N, Zarnowiecki D, Cho J, Wilson A, Bogomolova S, Villani A, et al. A Mediterranean-style dietary intervention supplemented with fish oil improves diet quality and mental health in people with depression: a randomized controlled trial (HELFIMED). Nutr Neurosci (2017) 1–14. doi: 10.1080/1028415X.2017.1411320

43. Firth J, Teasdale S, Allot K, Siskind D, Marx W, Cotter J, et al. The efficacy and safety of nutrient supplements in the treatment of mental illness: a systematic synthesis of 33 meta-analyses of randomized controlled trials. World Psychiatry(2019) in press.

44. Firth J, Rosenbaum S, Ward PB, Curtis J, Teasdale SB, Yung AR, et al. Adjunctive nutrients in first-episode psychosis: a systematic review of efficacy, tolerability and neurobiological mechanisms. Early Interven Psychiatry(2018). doi: 10.1111/eip.12544

45. Pawełczyk T, Piątkowska-Janko E, Bogorodzki P, Gębski P, Grancow-Grabka M, Trafalska E, et al. Omega-3 fatty acid supplementation may prevent loss of gray matter thickness in the left parieto-occipital cortex in first episode schizophrenia: a secondary outcome analysis of the OFFER randomized controlled study. Schizophr Res(2018) 195:168–75. doi: 10.1016/j.schres.2017.10.013

46. Firth, Joseph R., et al. “What Is the Role of Dietary Inflammation in Severe Mental Illness? A Review of Observational and Experimental Findings.” Frontiers in Psychiatry, vol. 10, 2019, doi:10.3389/fpsyt.2019.00350.
47. Serra-Mayem L., Ribas L., Ngo J., Ortega R.M. Food, youth and the Mediterranean diet in Spain. Development of KIDMED, Mediterranean Diet Quality Index in children and adolescents. Public Health Nutr. 2004;7:931–935.

48. Štefan, Lovro, et al. “The Reliability of the Mediterranean Diet Quality Index (KIDMED) Questionnaire.” Nutrients, vol. 9, no. 4, 2017, p. 419., doi:10.3390/nu9040419

49. O’Donnell, T., et al. “Effects of Chronic Lithium and Sodium Valproate on Concentrations of brain Amino Acids.” European Neuropsychopharmacology, vol. 13, no. 4, 2003, pp. 220–227., doi:10.1016/s0924-977x(03)00070-1.

50. Jacka, Felice N. “Nutritional Psychiatry: Where to Next?” EBioMedicine, vol. 17, 2017, pp. 24–29., doi:10.1016/j.ebiom.2017.02.020.

51. Blouin, Mélissa, et al. “Adiposity and Eating Behaviors in Patients Under Second Generation Antipsychotics.” Obesity, vol. 16, no. 8, 2008, pp. 1780–1787., doi:10.1038/oby.2008.277.

52. Kleinert S, Horton R. Rethinking and reframing obesity - comment. Lancet 2015. doi:1016/S0140-6736(15)60163-5.

53. Johnson PM, Kenny PJ. Addiction-like reward dysfunction and compulsive eating in obese rats: role for dopamine D2 receptors. Nat Neurosci 2010;13(5):635–41. doi: 10.1038/nn.2519

54. Deckersbach T, Das SK, Urban LE, Salinardi T, Batra P, Rodman AM, et al. Pilot randomized trial demonstrating reversal of obesity related abnormalities in reward system responsivity to food cues with a behavioural intervention. Nutr Diab 2014;4:e129. doi:1038/nutd.2014.26 doi: 10.1038/nutd.2014.26

55. Bach-Faig A, Berry EM, Lairom D, Reguant J, Trichopoulou A, Dernini S, et al. Mediterranean diet pyramid today. Science and cultural updates. Pub Health Nutr 2011;14(12A):2274–84. doi: 10.1017/S1368980011002515

56. Behera, Binati, et al. “Physical Activity and Eating Habits: The Major Elements to BMI among Indian Undergrads.” International Journal of Pharmacology, 2017.

57. O’Neil, A., Shivappa, N., Jacka, F., Kotowicz, M., Kibbey, K., Hebert, J., & Pasco, J. (2015). Pro-inflammatory dietary intake as a risk factor for CVD in men: A 5-year longitudinal study. British Journal of Nutrition,114(12), 2074-2082. doi:10.1017/S0007114515003815

58. Sæland M, Haugen M, Eriksen F-L, Smehaugen A, Wandel M, Böhmer T, et al. Living as a drug addict in Oslo, Norway – a study focusing on nutrition and health. Public Health Nutr. 2008;12:630.

59. Emerson MH, Glovsky E, Amaro H, Nieves R. Unhealthy weight gain during treatment for alcohol and drug use in four residential programs for Latina and African American women. Subst Use Misuse. 2009;44:1553–65.

60. Lucas M, Mekary R, Pan A, et al. Relation between clinical depression risk and physical activity and time spent watching television in older women: a 10-year prospective follow-up study. Am J Epidemiol 2011; 174: 1017-1027.

61. Phongsavan P, Merom D, Bauman A, Wagner R. Mental illness and physical activity: therapists’ beliefs and practices [letter]. Aust N Z J Psychiatry 2007; 41: 458-459.
62. Jacka, Felice N, and Michael Berk. “Depression, Diet and Exercise.” *The Medical Journal of Australia*, vol. 1, no. 4, Jan. 2012, pp. 21–23., doi:10.5694/mjao12.10508.

63. S. Opie, C. Itsiopoulos, N. Parletta, A. Sanchez-Villegas, T.N. Akbaraly, A. Ruusunen & F.N. Jacka (2017) Dietary recommendations for the prevention of depression, Nutritional Neuroscience, 20:3, 161-171, DOI: 10.1179/1476830515Y.0000000043

64. Natalie Parletta, Dorota Zarnowiecki, Jihyun Cho, Amy Wilson, Svetlana Bogomolova, Anthony Villani, Catherine Itsiopoulos, Theo Niyonsenga, Sarah Blunden, Barbara Meyer, Leonie Segal, Bernhard T. Baune & Kerin O’Dea (2019) A Mediterranean-style dietary intervention supplemented with fish oil improves diet quality and mental health in people with depression: A randomized controlled trial (HELFIMED), Nutritional Neuroscience, 22:7, 474-487, DOI: 1080/1028415X.2017.1411320

65. Jacka, F.N., O’Neil, A., Opie, R. et al. A randomised controlled trial of dietary improvement for adults with major depression (the ‘SMILES’ trial). *BMC Med* 15, 23 (2017). https://doi.org/10.1186/s12916-017-0791-y

66. Peet, M. (2004). International variations in the outcome of schizophrenia and the prevalence of depression in relation to national dietary practices: An ecological analysis. *British Journal of Psychiatry*, 184(5), 404-408. doi:10.1192/bjp.184.5.404

67. Taylor DM, McAskill R: Atypical antipsychotics and weight gainasystematic review. Acta Psychiatr Scand 2000; 101:416–32

68. Allison DB, Mentore JL, Heo M, et al.: Antipsychotic-induced weight gain: A comprehensive research synthesis. Am J Psychiatry-try 1999; 156:1686–96

69. Brown S, Birtwistle J, Roe L, et al.: The unhealthy lifestyle of people with schizophrenia. Psychol Med 1999; 29:697–701

70. Stokes C, Peet M. Dietary sugar and polyunsaturated fatty acid consumption as predictors of severity of schizophrenia symptoms. Nutritional Neuroscience. 2004 Aug;7(4):247-249. DOI: 10.1080/10284150400010012.

71. American psychiatric A: Diagnostic and statistical manual of mental disorders.4th ed. Washington DC: 2000.

72. Shaheen Lakhan SE, Vieira KF. Nutritional therapies for mental disorders. Nutr Jr. 2008;7:2.

73. Leontieva, Luba, et al. “Attention, Concentration and Planning Ability Improvement in Response to Depression Treatment during Acute Psychiatric Hospitalization.” *Clinical Depression*, vol. 03, no. 02, 2017, doi:10.4172/2572-0791.1000123.

Tables

Table 1: Sample Characteristics (n=100)
| Item                        | Frequency (N) | Percentage (%) |
|-----------------------------|---------------|----------------|
| Age in years                |               |                |
| 18-30                       | 25            | 25%            |
| 31-40                       | 24            | 24%            |
| 41-50                       | 16            | 16%            |
| 51-60                       | 22            | 22%            |
| >60                         | 13            | 13%            |
| Race                        |               |                |
| White                       | 79            | 79%            |
| Black                       | 13            | 13%            |
| Asian or Pacific Islander   | 1             | 1%             |
| American Indian             | 2             | 2%             |
| Other                       | 5             | 5%             |
| Gender                      |               |                |
| Male                        | 55            | 55%            |
| Female                      | 45            | 45%            |
| Smoker                      |               |                |
| Yes                         | 46            | 46%            |
| No                          | 54            | 54%            |
| Body Mass Index             |               |                |
| <18.5                       | 2             | 2%             |
| 18.5-24.9                   | 36            | 36%            |
| 25-29.9                     | 25            | 25%            |
| >30                         | 37            | 37%            |
| Level of Education          |               |                |
| Less than High School       | 16            | 16%            |
| High School Diploma         | 25            | 25%            |
| Vocation/Trade School       | 4             | 4%             |
| College/Associate Degree    | 39            | 39%            |
| Bachelor's Degree           | 16            | 16%            |
| Income          |       |     |
|-----------------|-------|-----|
| Less than 10K   | 18    | 18% |
| 10-29K          | 37    | 37% |
| 30-49K          | 27    | 27% |
| 50-69K          | 12    | 12% |
| 70K and above   | 6     | 6%  |

| Exercise        |       |     |
|-----------------|-------|-----|
| Do not exercise | 36    | 36% |
| Exercise once a week | 5    | 5%  |
| Exercise 2-4 times/week | 32  | 32% |
| Exercise 5-7 times/week | 14  | 14% |
| Not regular     | 13    | 13% |

Table 2

Correlation between KIDMED score and age, gender, education, race, BMI, income and physical activity. Chi-Square Analysis.

| KIDMED Score | Variable   | S. Coef | P value |
|--------------|------------|---------|---------|
|              | Age        | -0.124  | 0.223   |
|              | Gender     | 0.43    | 0.407   |
|              | Education  | 1.98**  | 0.05*   |
|              | Race       | 0.71    | 0.480   |
|              | Income     | 0.04    | 0.970   |
|              | BMI        | -0.124**| 0.04*   |
|              | Physical Activity | 0.149 | 0.138 |

* indicates p value <0.05

** indicates Spearman Coefficient of range >1/-1
Table 3
Frequency of Individual Mental Health illness in our patient population.

| Mental Illness                | N  |
|------------------------------|----|
| Mood Disorders               | 70 |
| Psychotic Disorders          | 29 |
| Anxiety                      | 50 |
| Substance Abuse              | 64 |
| Others*                      | 23 |
| Total N                      | 100|

Others*: Neurocognitive decline, Tourette's Syndrome, Intellectual disability, Gender dysphoria, Mild cognitive impairment, Depression due to a medical condition, Eating disorder, ADHD, Conversion disorder, Autism Spectrum and Insomnia

Table 4
Overlap of Mental Illness in the patient population + Mean KIDMed Scores

| Mental Illness                          | Frequency | Mean KidMed Score | Std. Deviation |
|-----------------------------------------|-----------|-------------------|----------------|
| Mood + Anxiety                          | 14        | 2.07              | 1.54           |
| Mood + Substance Abuse                  | 16        | 2.06              | 1.61           |
| Psychotic + Substance Abuse             | 6         | 2.83              | 2.22           |
| Anxiety + Substance Abuse               | 6         | 2.17              | 1.16           |
| Psychotic + Anxiety + Substance Abuse   | 4         | 2.25              | 0.95           |
| Mood + Psychotic + Substance Abuse      | 5         | 2.60              | 1.67           |
| Mood + Anxiety + Substance Abuse        | 11        | 2.82              | 2.04           |
| Mood + Anxiety + Substance Abuse + Others | 4       | 1.50              | 2.51           |

>4 KIDMed Classified “Healthy”

<4 KIDMed Classified “Unhealthy”

Figures
Figure 1

Percentage distribution of study sample according to KIDMED score.
Figure 2

Bar Histogram showing the Level of Education vs KIDMed Score.
Figure 3

Bar Histogram showing BMI groups vs KIDMed Score.
Figure 4

Bar Histogram showing the average consumption of sugar in our sample population.