Probiotics and Depression

Name

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Abstract

The aim of this research was to see whether there was a correlation between probiotic intake and depression levels. A self-report survey was used to gather data on depression levels (DV), probiotic intake (IV1), and alcohol consumption (IV2) as a possible confound. The data were analyzed using several bivariate and multivariate linear regression models in RStudio. The findings indicated that there was no significant connection between probiotic intake and depression, that there was no significant relationship between alcohol use and depression, and that there were no significant interactions between alcohol, probiotics, and depression. The paper recognizes numerous limitations associated with sample bias and controls and makes recommendations for future research aimed at establishing correlations between these factors.

Introduction

The microbiome is a self-regulating colony of bacteria found in the human gut. It has been characterized as "a sophisticated and ecologically active community that plays a critical role in health and illness" (Taggart, 2014, p.1). There is overwhelming data demonstrating depression's huge effect on society, ranging from low scholastic performance to job insecurity and higher mortality rates, all of which lead to a catastrophic economic loss (Kessler, 2011). Additionally, there is growing evidence indicating a link between the microbiome and depression (Winter, Hart, Charlesworth, & Sharpley, 2018). This is unsurprising, given that about 90% of our serotonin is generated in the stomach (Stoller-Conrad, 2015). This knowledge has sparked widespread interest in the use of probiotics to replenish the microbiome with good bacteria in the aim of alleviating the symptoms of mood disorders such as depression. The potential for probiotic use as a depression therapy requires further study on their connection. While many research demonstrate a favorable connection between probiotics and depression (McKean, Naug, Nikbakht, Amiet, & Colson, 2017), one meta-analysis shows that such studies' findings are unclear (Ng, Peters, Ho, Lim, & Yeo, 2018).

However, prior research indicates a dearth of controls for possible confounding variables and the interaction effects of popular dietary items, such as alcohol. This leads to the hypothesis that there is a connection between probiotic intake and depression, that this correlation is confounded by alcohol use, and that there is an interaction effect between alcohol, probiotics, and their effects on depression. This establishes the framework for my study and following hypotheses:

Hypothesis 1: Main Effect

H0: There is no correlation between probiotic consumption and depression.

Ha: There is a correlation between probiotic consumption and depression.

Hypothesis 2: Main Effect

H0: There is no correlation between alcohol consumption and depression.

Ha: There is a correlation between alcohol consumption and depression.

Hypothesis 3: Confound

H0: Alcohol consumption does not explain the correlation between probiotics and depression.

Ha: Alcohol consumption explains the correlation between probiotics and depression.

Hypothesis 4: Interaction Effect

H0: The relationship between probiotics and depression does not depend on alcohol consumption.

Ha: The relationship between probiotics and depression depends on alcohol consumption.

Methods

Participants:

The original population for this research was 132(N = 132) individuals. I eliminated eight individuals from this group due to their inability to complete the survey, leaving a sample size of 124 (n = 124) for data analysis. There were 52 men (42%), 71 females (57%), and 1 participant classified as "Other" in the sample size. Participants self-identified as being between the ages of "18 - 24" "25 - 34" "35 - 44" "45 - 54" "55 - 64" "65 - 74" or "75 - 84," with the "45-54" age group reporting the greatest frequency (33 percent). Participants were recruited via my partner's Facebook network and my network within the San Francisco Suicide Prevention organization, where I volunteer. Participants in the Facebook network were invited to participate in an academic survey on depression through their notifications wall. They were told that the survey results might provide some light on future mental health treatments. They received no extra compensation other than the satisfaction of contributing to the benefit of mental health services. San Francisco Suicide Prevention participants got an email from the volunteer organizer inviting them to participate in an academic survey performed by another volunteer. Additionally, they were told that survey results might possibly offer insight into future mental health treatments. They received no extra benefits.

Procedures:

The self-report survey was accessible by participants through a link that was either placed on their Facebook page or emailed to them. The link sent users to a 23-question Qualtrics survey. Three demographic questions were included: sex, age range, and race. Six questions regarding food consumption were addressed, including probiotic intake and frequency, coffee use, and alcohol consumption. The survey was designed in such a way that if a person indicated that they did not drink probiotics, they were not asked additional questions about the frequency or manner of probiotic intake; instead, they were asked about their alcohol and coffee use. The following questions assessed participants' depression levels in relation to a variety of variables, including melancholy, energy levels, sleep disruption, and general quality of life.

Measures and Manipulations:

Dependent Variable (DV)

Depression served as the dependent variable (DV) in my research. This was determined via a self-report survey utilizing the Clinically Useful Depression Outcome Scale (CUDOS), a frequently used scale by physicians to assess depression levels (Zimmerman, Chelminski, McGlinchey, & Posternak, 2008). Participants responded to questions on their perceived degrees of sadness, including "I felt sad or melancholy," "I was less interested in my normal activities," "I believed the future looked bleak," and "How would you rank your overall quality of life over the last week?" The responses of participants were rated on a five-point scale using the following guidelines: 0 indicates that nothing is true (0 days), 1 indicates that something is sometimes true (1-2 days), 2 indicates that something is occasionally true (3-4 days), 3 indicates that something is often true (5-6 days), and 4 indicates that something is nearly always true (every day). The last two questions of the CUDOS were scored using the following rating guideline: 0) excellent; my life could scarcely be much better. 1) quite good; most things are doing well; 2) fairly awful; most things are going badly; 4) very terrible; my life could scarcely be worse. I removed two items from the CUDOS about mortality and suicide thoughts merely to avoid emotional triggers, keeping in mind that this was not a professionally supervised survey.

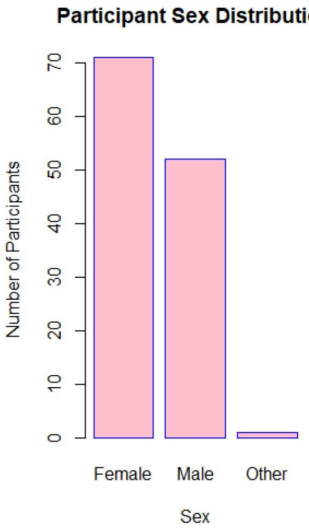
Independent Variable 1 (IV1)

Probiotic intake was the first independent variable in my research. Participants were asked whether they consumed probiotics and provided various options, including yogurt, kombucha, kimchi, miso, fermented foods, and pills. This was quantified using a self-report survey as a categorical variable with three levels: "Yes," "No," or "Not Certain."

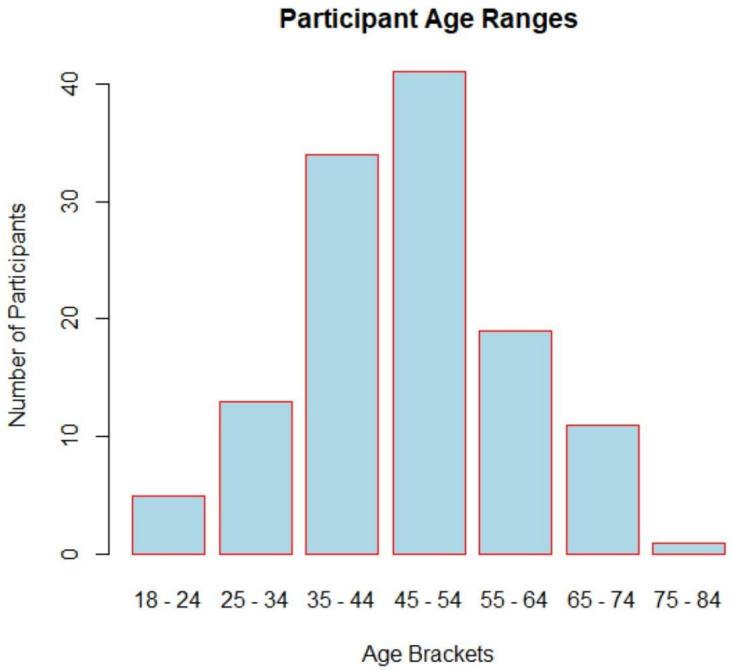
Independent Variable 2 (IV2)

Independent variable 2 for my study was alcohol consumption. Participants were asked whether or not they consumed alcohol. This was measured via the self-report survey as a categorical variable with two levels: “Yes or “No”.

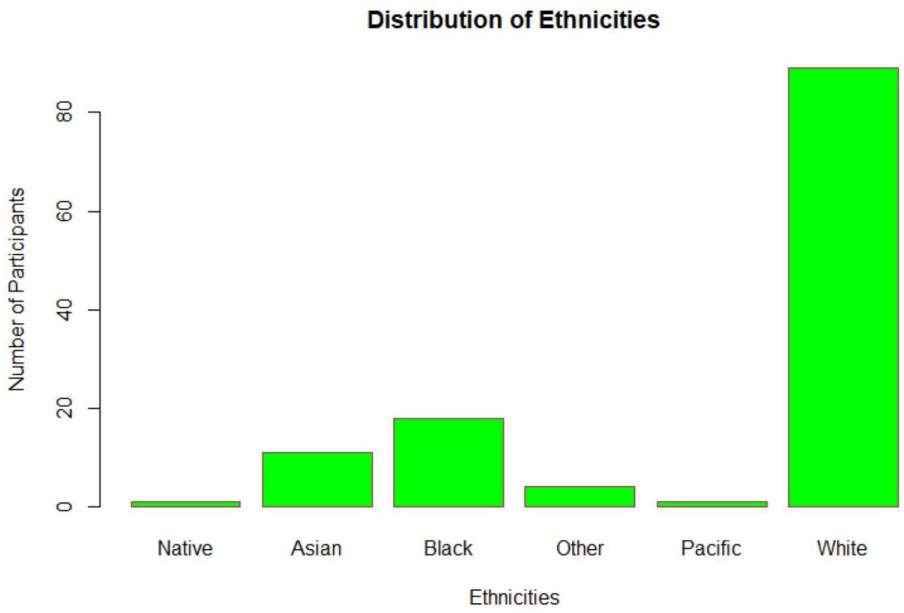
Results



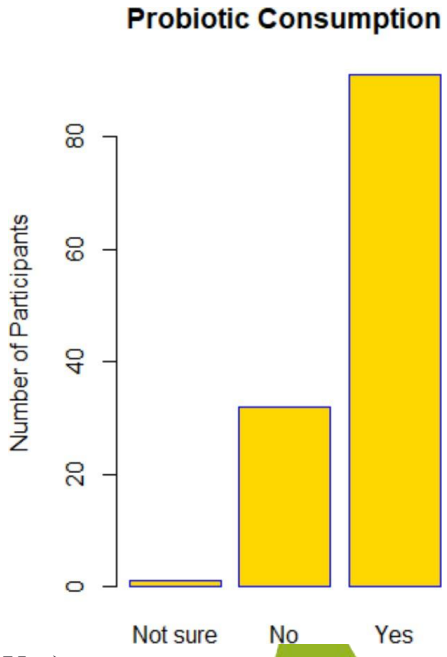
The variables measured in this study were sex (71 Female, 52 Male, 1 Other) as is illustrated in the following graph, age bracket (5 in the "18-24", 13 in the "25-34", 34 in the "35-44", 41 in the "45-54", 19 in the "55-64", 11 in the "65-74", 1 in the "75-84", mode = "45-54") as seen in the following graph,



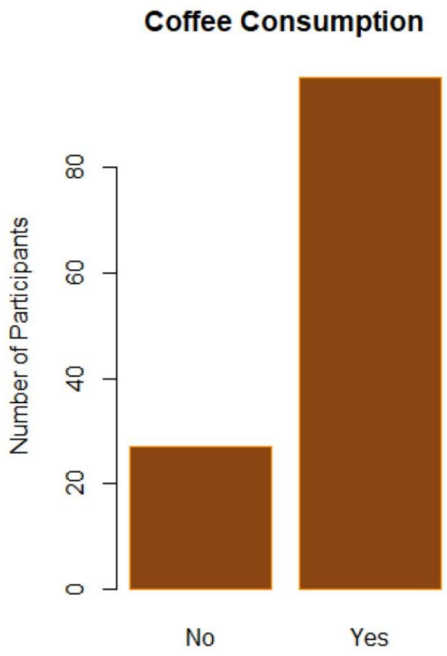
ethnicity (1 Native American/Alaska Native, 11 Asian, 18 Black/African American, 1 Pacific



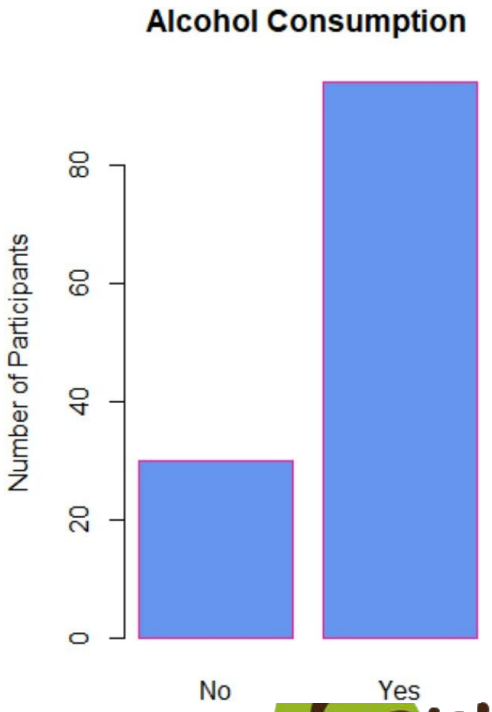
Islander, 89 White, 4 Other) as shown in the following graph, probiotic consumption (32 No, 91 Yes, 1 Not Sure),



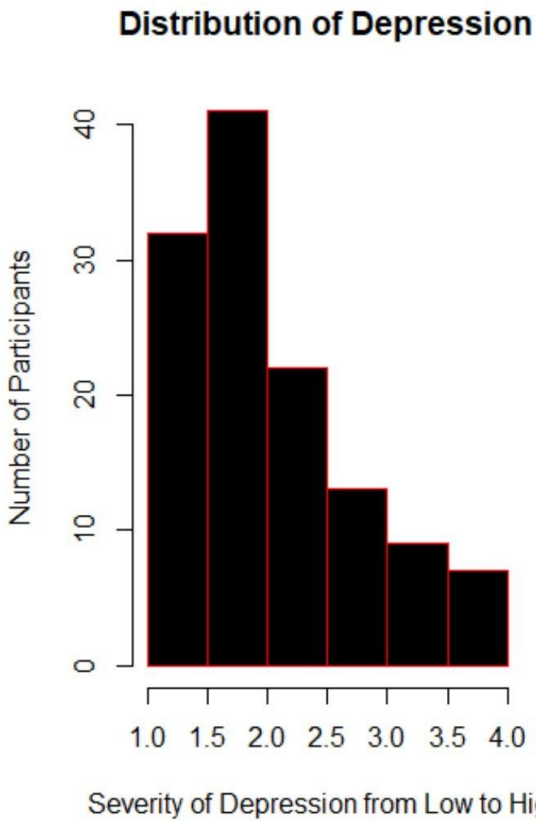
coffee consumption (27 No, 97 Yes),



alcohol consumption (30 No, 94 Yes),



and level of depression (mean = 2.07, SD = 0.72, range = 2.93, alpha = 0.92).

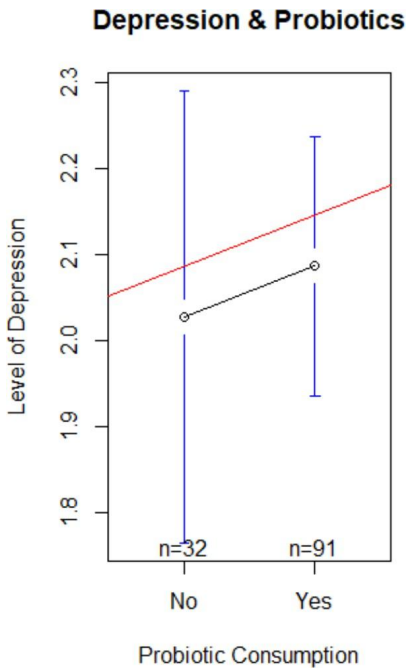


A series of linear regression models were utilized to predict relationships between depression and probiotic consumption, depression and alcohol consumption, and to see if there were any interaction effects between depression, probiotic consumption, and alcohol consumption.

Hypothesis 1 (Model 1)

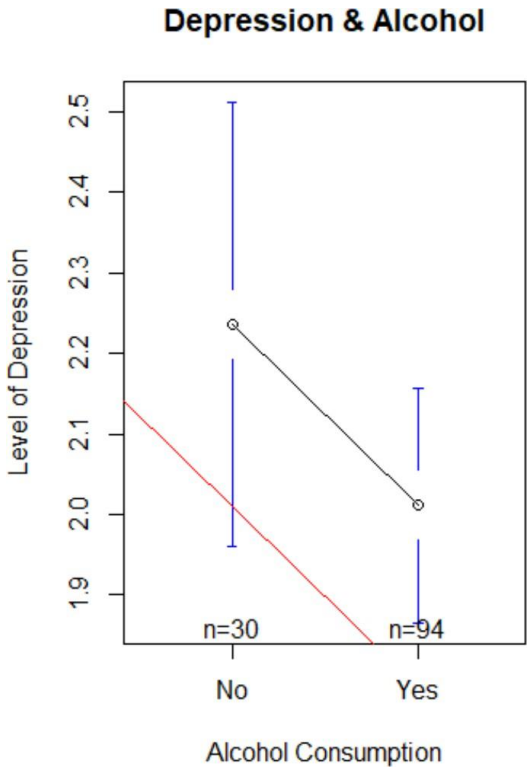
A bivariate linear regression model was used to predict the relationship between depression and probiotic consumption. This model shows that there is a slight correlation between depression and probiotic consumption, with people who take probiotics predicted to see an increase in their levels of depression, (slope = .06, 95% CI = [-.24, .35], $t(121) = 0.4$, $p = 0.7$, $R^2 = .001$).

However, this correlation is extremely weak, with the R^2 indicating that less than 1% of the variation in levels of depression is explained by probiotic consumption. The p-value of 0.7 is greater than .05 which means we fail to reject the null hypothesis. The power for this model is P = .04, meaning that if the alternative hypothesis were true, we could expect to find a sample that would allow us to reject the null hypothesis only about 4% of the time. So, based on this model, there is not a strong nor significant relationship between depression and probiotic consumption.



Hypothesis 2 (Model 2)

A bivariate linear regression model was used to predict the relationship between depression and alcohol consumption. This model shows that there is a negative correlation between depression and alcohol consumption, with people who drink alcohol predicted to see a decrease in their levels of depression (slope = -0.22, 95% CI = [-0.52, 0.07], t(122) = -1.5, p = 0.14, R^2 = 0.018). However, this correlation is extremely weak, with the R^2 indicating that only about 1.8% of the variation in depression is explained by alcohol consumption. The p-value of 0.14 is greater than .05 which means we fail to reject the null hypothesis. The power for this model is P = 0.04, meaning that if the alternative hypothesis were true, we could expect to find a sample that would allow us to reject the null hypothesis only about 4% of the time. Based on this model, there is not a strong nor significant relationship between depression and alcohol consumption.

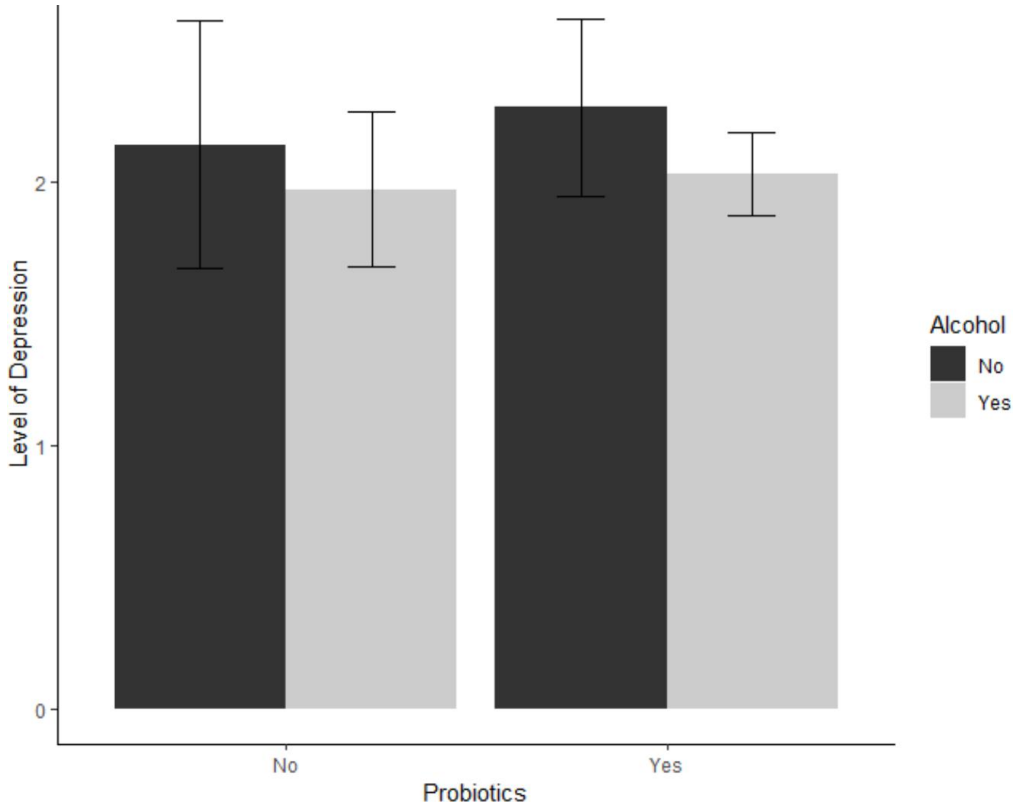


Hypothesis 3 (Model 3)

A multivariate linear regression model was used to determine whether the relationship between depression and probiotics could be explained by alcohol consumption. The model shows that the relationship between depression and probiotic consumption increased when alcohol consumption was included. However, the suppression by alcohol consumption was minimal and not significant (slope = 0.08, 95% CI = [-0.21, 0.37], t(120) = 0.54, p = 0.31, R^2 = 0.019). The p-value of 0.31 is greater than 0.05, so we fail to reject the null hypothesis. Based on this model, alcohol does not explain the relationship between probiotics and depression.

Hypothesis 4 (Model 4)

A model was used to determine if the relationship between depression and probiotics depends on alcohol consumption. There was no interaction effect of alcohol on the relationship between probiotic consumption and levels of depression. People who consumed probiotics were predicted to have higher levels of depression, regardless of whether they consumed alcohol or not (slope = -0.08, 95% CI = [-0.74, 0.57], t(119) = -0.25, p = 0.49, R^2 = 0.02). The p-value of 0.49 is greater than 0.05, so we fail to reject the null hypothesis.



Standardized βs

	Model 1	Model 2	Model 3	Model 4
Probiotic Consumption	0.06 [-.24, .35] t(121) = 0.4 p = 0.7 R^2 = .001		0.08 [-0.21, 0.37] t(120) = 0.54 p = 0.6 R^2 = 0.019	0.14 [-0.42, 0.69] t(119) = 0.5 p = 0.62 R^2 = 0.02
Alcohol Consumption		-0.22 [-0.52, 0.07] t(122) = -1.5 p = 0.14 R^2 = 0.018	-0.23 [-0.53, 0.08] t(120) = -1.49 p = 0.14 R^2 = 0.019	-0.17 [-0.71, 0.38] t(119) = -0.61 p = 0.54 R^2 = 0.019
Probiotic Consumption * Alcohol Consumption				-0.08 [-0.74, 0.57] t(119) = -0.25 p = 0.8 R^2 = 0.019

Discussion

Key Findings

Previous meta-analyses of research examining the association between depression and probiotic intake produced inconsistent results. One meta-analysis discovered a significant connection between probiotics and depression levels, whereas another found no correlation. This discrepancy served as the impetus for this research, which aimed to establish a significant link between probiotics and depression. However, this study's findings indicate that there is no connection between the two factors.

Given that serotonin, the neurotransmitter implicated in mood regulation, is mostly generated in the gut, researchers sought evidence that manipulating the gut microbiota would impact the serotonergic system. The idea that probiotic intake would have a negative connection with depression levels was disproved, with this research indicating that probiotic consumers are expected to have greater levels of depression ($r = 0.03$). The idea that alcohol use, a recognized depressive, would serve as a mediator between probiotics and depression was disproved, with this research demonstrating that alcohol intake instead strengthened the connection between probiotics and depression ($r = 0.14$). The hypothesis that alcohol consumption would have an interaction effect on probiotics and depression was also disproved, with this study demonstrating that people who consumed probiotics in general were predicted to have higher levels of depression regardless of whether they consumed alcohol or not ($r = 0.14$). None of the regression models had a p-value less than 0.05, indicating that none of the regression models were significant.

Limitations and Future Research

This research has many drawbacks. For one thing, the tiny sample size was drawn from a very limited segment of the overall population, introducing sampling bias. The sample drawn from the San Francisco Suicide Prevention Center, in particular, may have been skewed toward depression, leading in bias. Another issue is the difficulty of detecting and removing particular strains of bacteria present in probiotics. While some strains are utilized in manufacturing and naturally exist in foods, various brands of yogurt, meals, and supplements include a variety of bacteria strains. Additionally, there is a broad range of microorganisms present in various meals and supplements. Finally, this research did not account for the kind of alcohol ingested, which may have a varied effect on the microbiota based on how various forms of alcohol are metabolized. It is abundantly apparent that the available scientific data about the connection between depression and probiotic intake is inconclusive. While it has been established that serotonin is associated with depression, the majority of serotonin is produced in the gut, and the gut microbiome plays a role in serotonin production, additional research is required to establish a strong correlation or causation between these two variables. Future study may benefit from adjusting for differences in bacteria strains and quantities, as well as the interaction effects of a variety of typical dietary items that may influence the microbiota and serotonergic activity.

Works Cited

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