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# The Relationship Between Alcohol Consumption and Depression: An Analysis of Secondary Affections and Therapeutic Interventions

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**Abstract:** Alcohol use disorders are associated with many negative mental health outcomes such as the aggravation of symptoms of depression and anxiety disorders and, notably, increased suicidality. The nearly reciprocal relationship between the two entities makes treatment much more complex and necessitates care pathways that are integrated. The present research addressed linking levels of alcohol use to the common mood disorders depression and anxiety and evaluating the feasibility of psychological interventions in reducing consumption and relieving the associated psychiatric symptoms. The sample comprised 147 patients hospitalized in a psychiatric facility (2021–2023) that were diagnosed according to DSM-5 criteria with alcohol dependence and depressive or anxiety comorbidities. The baseline and follow-up assessments utilized AUDIT (alcohol use), BDI (depression), and GAD-7 (anxiety) questionnaires. The psychological interventions included cognitive-behavioral techniques and motivational interviews. Of the participants, 33.8% presented with comorbid depression, 32.8% with anxiety disorders, and 33.4% with cognitive impairments. The psychological interventions significantly reduced alcohol consumption and the severity of depressive and anxiety symptoms. Superlative clinical outcomes came about with longer intervention time. The results call for the need for co-treating associated alcohol use as well as said mental conditions to optimize therapeutic results and improve quality of life for patients. These major implications lend themselves to the development of public health policies and tailored interventions to combat the concurrent battles between alcohol consumption and depression.

**Keywords:** alcohol consumption; depression; anxiety; psychology; cognitive-behavioral techniques



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## 1. Introduction

Depression and alcohol consumption are significant public health issues with profound implications for global and local populations. According to the World Health Organization

(WHO [1]), the global prevalence of depression in 2015 was 4.4%, affecting approximately 322 million people. In Romania, depression had a prevalence of 5% in the same year, accounting for an estimated 931,842 cases. Globally, the average alcohol consumption was 6.4 L of pure alcohol per capita among individuals aged 15 and older (2016). Nationally, 80% of men and 55% of women consumed alcohol, out of which 67% of men and 31% of women reported excessive consumption [1–3].

These data underline the profound impacts of depression and alcohol consumption on physical and mental health. The WHO has identified depression as the second leading cause of disability worldwide, affecting over 300 million individuals [4–6]. Approximately 800,000 people commit suicide every year, and suicide is associated with severe depression or secondary mental affections [7,8]. Concurrently, alcohol use accounts for 5.3% of global deaths, or approximately 3 million annually, as a significant factor in the development of mental and physical health disorders [9,10].

The relationship between alcohol consumption and depression is complex and bidirectional [11]. Alcohol's effects on the brain range from pleasant feelings of relaxation and happiness to severe intoxication (memory loss and impaired motor skills) and withdrawal symptoms (headaches, anxiety, and tremors). The amount and duration of alcohol use, frequency of consumption, and use of other substances influence the severity of these effects. Alcohol disrupts the neurotransmitters GABA (linked to relaxation) and dopamine (involved in reward). Chronic alcohol use creates a GABA imbalance, leading to negative physical and psychological consequences during withdrawal and potentially including delirium tremens. Alcohol's dopamine release reinforces its use through the brain's reward system, contributing to dependence [12,13]. Patients sometimes use alcohol to cope with depression and anxiety or to improve sleep, but this can backfire. While alcohol may provide temporary relief, it disrupts sleep and energy levels. This creates a harmful cycle: initial alcohol use eases depression but then depletes serotonin, leading to increased depression and a need for more alcohol, perpetuating the problem [14]. Excessive alcohol consumption may worsen depressive symptoms, while depression increases the likelihood of problematic alcohol consumption [15,16]. The comorbidity of these conditions is often associated with more severe symptoms, an increased risk of suicide, and significantly reduced quality of life [17]. Further, co-morbidity management exercises are especially challenging because of the mutual dependence of the core elements [18,19]. Composite therapies that blend short- and long-term interventions are proving more effective for patients with alcohol dependence who also suffer from depression and anxiety disorders.

For instance, CBT has proven effective in treating substance use disorders and dependencies by offering strategies to modify alcohol-related behaviors and thoughts [20,21].

Furthermore, pharmacological treatments such as naltrexone or acamprosate can be combined with psychotherapeutic interventions to enhance efficacy in preventing relapse and maintaining abstinence in patients with alcohol dependence [22].

A key perspective in the treatment of patients with alcohol dependence and co-occurring depression and anxiety is the importance of tailoring interventions to the individual's unique needs and circumstances [23]. While short-term interventions may be suitable for patients with mild symptoms and strong social support, those with severe and chronic conditions may benefit more from intensive long-term treatments [24]. Personalized care that accounts for specific challenges, strengths, and treatment preferences can enhance intervention efficacy and promote better outcomes.

This study analyzed the relationship between alcohol consumption and depression/anxiety and evaluates the effectiveness of psychological interventions, such as cognitive-behavioral therapy (CBT) and motivational interviews. It aimed to identify

integrated and effective strategies for treating these comorbidities, ultimately improving patient quality of life and informing public health policies [2,3,25].

## 2. Materials and Methods

The study was conducted at the “Elisabeta Doamna” Clinical Psychiatric Hospital and utilized a quasi-experimental design with a pre-post analysis. It aimed at evaluating the relationship between alcohol consumption, depression, and anxiety and the effectiveness of psychological interventions by comparing symptom severity before and after the interventions.

The “Elisabeta Doamna” Clinical Psychiatric Hospital provides specialized inpatient care for individuals with severe psychiatric conditions, including alcohol dependence. Patients are admitted based on criteria that include acute psychiatric decompensation, pronounced withdrawal symptoms, or significant comorbid medical conditions requiring close supervision. Standard protocols for detoxification and monitoring are employed, involving routine laboratory tests (including markers for liver function), daily clinical evaluations, and psychosocial assessments. The average length of stay for these patients typically ranges from 20 to 30 days depending on illness severity, response to initial detoxification, and the presence of co-occurring psychiatric or medical issues. By detailing this context, we aimed to clarify the clinical environment in which the data were collected and the scope of support these hospitalized patients received.

### 2.1. Short-Term Interventions

Short-term treatment options for patients with alcohol dependence depression and anxiety often involve brief counseling aimed at enhancing motivation, enhancing coping skills, and providing psychoeducation. Usually, short-term interventions are of great help in stabilizing and relieving the immediate symptoms. These include brief courses of cognitive-behavioral therapy (CBT), motivational interviewing, and crisis intervention [20,26].

### 2.2. Long-Term Interventions

Long-term interventions comprise more encompassing treatment programs that address the underlying concerns and difficulties of alcohol-use disorders, depression, and anxiety. These treatment methods rely heavily on intensive psychotherapy, medications, group therapy, family therapy, and other multidisciplinary approaches. The main goal of these long-term interventions is to facilitate sustained changes in behavior and emotional regulation (which are easily visible as improved overall functioning).

### 2.3. Sample Description and Data Collection

All clinical and psychometric data were collected with a retrospective study design during the period 2021–2023.

The study sample consisted of 147 inpatients diagnosed with alcohol-related disorders according to DSM-5 criteria, along with depressive or anxiety symptoms. The short-term treatment options for patients with alcohol dependence depression and anxiety often involve brief periods of counseling aimed at enhancing motivation, enhancing coping skills, and providing psychoeducation. Usually, short-term interventions are of great help in stabilizing and relieving the immediate symptoms. Among these are brief periods of cognitive-behavioral therapy (CBT), motivational interviewing, and crisis intervention [20,26]. The inclusion criteria required participants to be at least 18 years old, have a confirmed diagnosis of alcohol dependence, and present relevant scores on standardized depression and anxiety scales. The exclusion criteria included significant psychotic disorders, cognitive deficits preventing evaluation, or insufficient clinical data.

Alcohol consumption severity was assessed using AUDIT (the Alcohol Use Disorders Identification Test) [27]. At the same time, the depressive and anxiety symptoms were evaluated using the Beck Depression Inventory (BDI) [28] and the Generalized Anxiety Disorder 7-item (GAD-7) questionnaire [29], respectively. Additionally, biological markers such as gamma-glutamyl transferase (GGT) [30] and carbohydrate-deficient transferrin (CDT) [31] were utilized to obtain objective data on chronic alcohol consumption. These data, along with sociodemographic information, were integrated into a secure database.

#### 2.4. Psychological Interventions

The psychological interventions included cognitive-behavioral techniques and motivational interviewing, which were categorized into short-term and long-term approaches based on the total duration and the number of sessions allocated to each patient [26,32,33]. The therapeutic team determined the assignment to a specific type of intervention by considering the severity of disorders, comorbidities, and resource availability.

#### 2.5. Statistical Analyses

All statistical analyses, including the correlation tests, group comparisons, and linear or logistic regression modeling, were performed using MATLAB R2021b (MathWorks, Natick, MA, USA). We selected MATLAB for its flexible scripting environment, advanced data manipulation capabilities, and comprehensive statistical toolboxes, which enabled the customization of the inferential tests within our quasi-experimental design. Additionally, MATLAB's robust graphical features were advantageous for generating high-quality boxplots and heatmaps, which enhanced the clarity of our data presentation. Both descriptive and inferential methods were applied to explore the relationships between alcohol consumption and the severity of depressive and anxiety symptoms, as well as to evaluate the impact of psychological interventions on patient outcomes. A significance threshold of  $p < 0.05$  was used for all analyses.

### 3. Results

The descriptive analysis of the sample revealed a mean age of 42.31 years (SD = 13.51) for the participants, with a median of 42 years, indicating a relatively balanced age distribution (Figure 1). The gender distribution showed similar proportions of men and women, suggesting no significant gender imbalance among the participants (Figure 1). Regarding the types of interventions, approximately 40% of patients underwent short-term interventions, while 60% received longer term ones, demonstrating the slight predominance of the latter.

At baseline (pre-intervention), the depression and anxiety scores marked moderate symptom severity in the participants. A significantly reduced review was successful for both variables post-intervention ( $p < 0.001$ ), which implied that the clinically beneficial regions in healing on their scales were there. These differences were depicted in graphical comparisons of the participants' depression and anxiety scores (pre- and post-intervention for the intervention types), showing a post-intervention reduction.

Comparing the short- and long-term interventions showed no statistically significant differences for the depression scores ( $p > 0.05$ ), while a significant difference was noted for anxiety ( $p = 0.018$ ), suggesting that the long-term interventions may not have had much more of an effect on reducing anxiety (Figure 2). These findings are visually represented through adjacent boxplots, highlighting the downward trend in scores by intervention type.

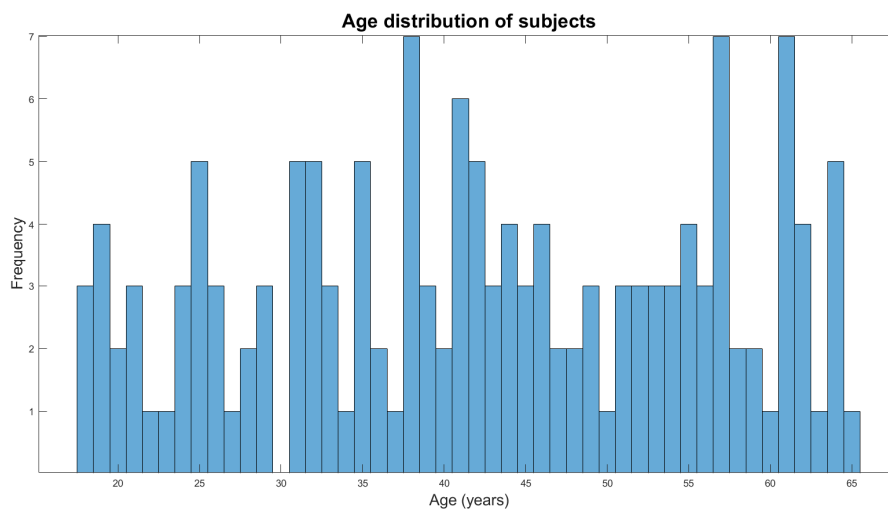


Figure 1. Patient distribution according to age.

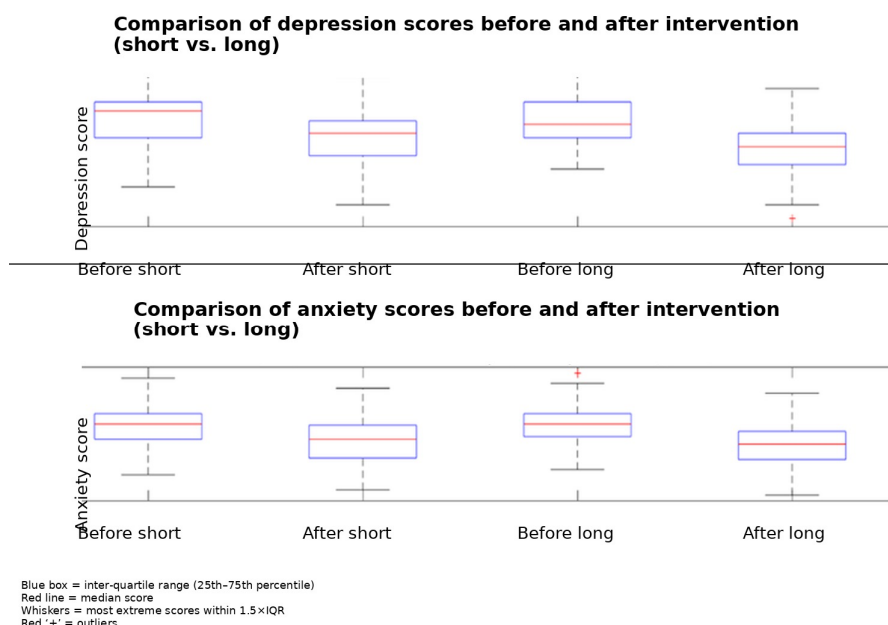
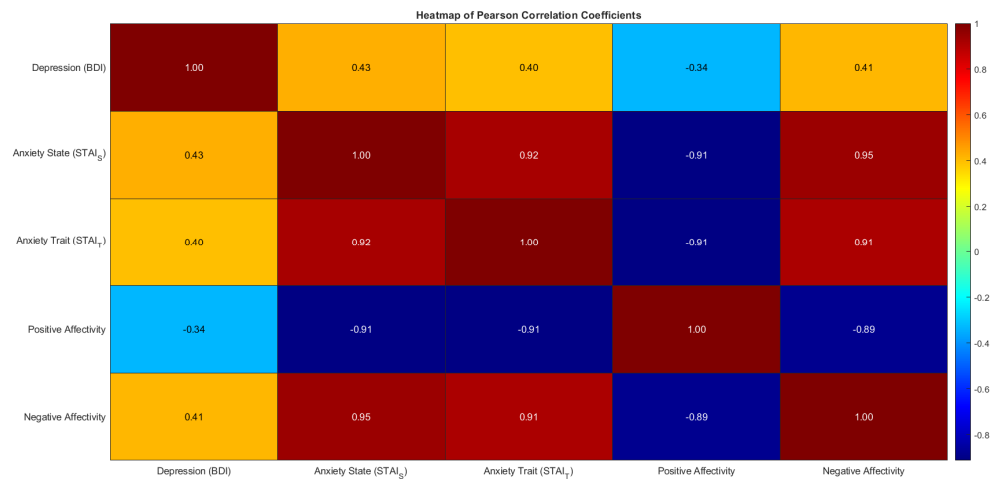


Figure 2. Score comparisons between the pre- and post- short- and long-term interventions.

*Correlation Analysis of the Psychological Variables*

To investigate the relationships among the depressive symptoms, anxiety (state and trait), and positive/negative affect, we conducted a Pearson’s correlation analysis. The findings, presented as a heatmap (Figure 3), revealed strong associations between anxiety (STAI\_S and STAI\_T) and negative affect ( $r = 0.91–0.95$ ), as well as significant negative correlations between anxiety and positive affect ( $r = -0.91$ ). Additionally, the BDI scores showed moderate associations with both anxiety and negative affect ( $r = 0.40–0.43$ ), suggesting that the depression severity was closely linked to the anxiety levels and negative affect, albeit not as strongly as the robust connection observed between anxiety and negative affect. These results underscored the interdependence of emotional dimensions in the patients presenting both depressive and anxiety symptoms, which was in line with previous literature, indicating a frequent co-occurrence of heightened negative affect alongside reduced positive affectivity.



**Figure 3.** Heatmap of the Pearson correlation coefficients between alcohol consumption and psychological variables (depression, anxiety state, anxiety trait, and positive and negative affectivity).

Linear regression models were constructed to identify the factors associated with post-intervention depression score variation, but they failed to identify significant predictors ( $p > 0.05$ ), highlighting the difficulty in explaining the result variability solely through the included variables. The low  $R^2$  values suggested a limited capacity for modeling score changes. Another distinction that was noticeably observed in these logistic regression models that attempted to predict the probability of a therapeutic response (which was defined as a more than five-point reduction in depression score) was data separation, leaving open the possibility of the further pursuit of large-sample studies or socio-economic covariate integrated studies from which potential predictive factors could be identified.

One potential explanation for the absence of significant predictors was the relatively small sample size (number = 147), which may have limited the statistical power to detect subtle but clinically relevant associations. Additionally, the unmeasured confounders (e.g., social support, medication adherence, and genetic predispositions) might have contributed to the outcome variability not captured by our current model. Future research should incorporate larger, more diverse samples and consider additional psychosocial and biological covariates to better elucidate the multifactorial nature of comorbid substance use and affective disorders. Furthermore, advanced statistical techniques such as mediation and moderation analyses may offer deeper insights into the pathways linking alcohol consumption, depression, and anxiety. We have noted these considerations and proposed them as directions for further study.

The results emphasized the efficacy of the psychological interventions in alleviating depressive and anxiety symptoms while failing to identify clear clinical, demographic, or biological predictors of a therapeutic response. The observed differences in anxiety reduction between the short- and long-term interventions necessitate further research for confirmation and better understanding.

#### 4. Discussion

The results of this study highlight the efficacy of psychological interventions in reducing alcohol consumption and alleviating depressive and anxiety symptoms among patients diagnosed with alcohol dependence. The observed differences between short- and long-term interventions indicated opportunities for optimizing therapeutic approaches, although further studies are required for a better understanding of the mechanisms underlying the benefits of each type of intervention. This study showed significant improvements in depression and anxiety scores post-intervention.

Although the short- and long-term interventions did not differ significantly in improving depression, the latter appeared moderately more effective for anxiety. These findings underscored the strong interdependence of depressive symptoms, anxiety dimensions, and affective states, as demonstrated by the robust associations between state (STAI\_S) and trait (STAI\_T) anxiety and negative affect, coupled with their negative correlations with positive affect. This pattern suggested that heightened negative affectivity is closely tied to anxious experiences while simultaneously reducing one's capacity for positive emotional engagement. The moderate yet clinically meaningful correlations between the BDI depression scores and both state and trait anxieties highlighted overlapping affective dysregulation in the patients presenting both conditions, which was in line with prior research showing the frequent co-occurrence of elevated negative affect alongside diminished positive affectivity.

From a therapeutic perspective, interventions aimed at mitigating negative affect (e.g., cognitive restructuring to counter catastrophic thinking) could concurrently alleviate anxiety, whereas approaches promoting positive affect (e.g., behavioral activation and mindfulness-based techniques) may bolster resilience and protect against depressive relapse. Future research with larger and more diverse samples is warranted to confirm these patterns and further elucidate the underlying mechanisms that drive such pronounced interrelations among anxiety, negative affect, and reduced positive affectivity.

Moreover, the observed differences were insufficient to conclusively determine the trajectory of these relationships. This may reflect fundamental variations in how depressive symptoms and anxiety co-develop or potentially indicate constraints on the effectiveness of short-term versus longer term interventions. Our results aligned with those of Hofmann et al. [34], who reported greater reductions in anxiety symptoms with extended CBT protocols compared to shorter formats. Similarly, Kay-Lambkin et al. [35] found that a nine-session, therapist- or computer-led treatment—paired with an initial motivational interview (MI) and case formulation session—was more effective than a single-session approach at reducing both alcohol use and depressive symptoms, although their study's small sample size and limited data on treatment adherence posed constraints on making broader generalizations.

Baker et al. [36] compared a brief intervention, single-focus therapy protocol (targeting either depression or alcohol) and an integrated therapy strategy for 284 participants. The integrated approach was superior for addressing both depression and alcohol, whereas the single-focus strategy yielded the following gender-specific outcomes: men benefited more from alcohol-focused therapy, while women responded better to depression-focused therapy. The authors proposed a stepped-care model, starting with brief interventions and escalating as needed, although the short-term follow-up period limited their conclusions. Other investigations [35,37–39] have likewise indicated that MI and CBT protocols targeting depression can improve alcohol-related measures. In particular, therapist-led MI/CBT protocols, group MI therapy for mixed psychiatric disorders, and combined CBT therapy for alcohol and anxiety produced the strongest results, while anxiety improvements were generally smaller yet comparable in magnitude to the changes seen in alcohol measures [36–39].

No association was found between alcohol consumption, biological markers (GGT and CDT), and the severity of depressive and anxiety scores, raising questions about the utility of these biomarkers in predicting the severity of psychiatric disorders in individuals with alcohol dependence, suggesting the multifactorial nature of psychological disorders. Several confounding variables may have contributed to the lack of correlation between the biological markers (GGT and CDT) and psychiatric symptoms. The patients may have had unrecognized liver conditions or were taking psychotropic or hepatotoxic medications that altered their marker levels. Additionally, individual genetic variations can influence

enzyme activity, further complicating the direct relationship between these biomarkers and the severity of depressive or anxious symptomatology. Future studies should incorporate comprehensive medical histories, including medication use and liver function assessments, to delineate how these potential confounders affect clinical outcomes and biomarker readings.

The metabolic complications arising from chronic alcohol consumption can further compound the risk of depressive and anxiety disorders, adding complexity to an already multifaceted clinical picture. For instance, advanced hepatic pathologies (e.g., cirrhosis) may induce or exacerbate psychological stress, while persistent neurotoxic effects and an increased likelihood of certain malignancies can heighten anxiety regarding health status. Recognizing these intertwined physical and mental health challenges underscores the importance of future research aimed at fully integrating metabolic factors and psychiatric comorbidities, ultimately providing a more holistic approach to the prevention and treatment of alcohol-related disorders.

Previous studies' results on the correlation between these markers and psychiatric symptoms are conflicting. Our results were consistent with some previous studies [40–44] which reported weak or inconsistent links between biological measures and psychological outcomes, which were often mediated by psychosocial factors, while GGT and AST and GGT along with the Beck Depression Inventory and the Clinician-Administered PTSD Scale may have been significantly useful in predicting verbal learning and memory (Hopkins Verbal Learning Test-Revised—HVLT-R %Retention T Score) [40]. Some indirect biomarkers of alcohol use may help to identify patients at high risk of cognitive impairment. However, these biomarkers are not a substitute for a full neurocognitive assessment; rather, they are a potentially useful screening tool when a comprehensive evaluation is not possible. However, some studies have suggested that demographic factors, particularly age and socioeconomic status, might indirectly influence therapy outcomes via access to care or adherence from a public health perspective, and implementing targeted screening programs for early detection of both alcohol misuse and emerging depressive or anxiety symptoms could significantly improve intervention outcomes. Educational campaigns aimed at adolescents and young adults may raise awareness about the risks of alcohol dependence and encourage help-seeking behaviors. Additionally, integrating mental health services into primary care settings—along with motivational interviewing and cognitive-behavioral therapy—can offer timely psychosocial support and reduce the stigma surrounding psychiatric comorbidities. Collaborative efforts between healthcare providers, policymakers, and community stakeholders are essential for ensuring that preventive strategies and treatment resources are both accessible and culturally appropriate. By focusing on early identification, comprehensive education, and multidisciplinary care, public health measures can more effectively address the complex interplay between alcohol consumption and affective disorders [31,45,46]. Recent studies have highlighted the role of specific dietary patterns—such as high sugar intake, low omega-3 fatty acid consumption, or poor micronutrient status (e.g., deficiencies in folate, vitamin D, or magnesium)—in the development and persistence of depressive symptoms. These nutritional imbalances may also indirectly influence alcohol cravings and relapse vulnerability, suggesting that dietary assessments could enhance holistic care in comorbid cases [47]. Moreover, we acknowledge that gender differences play a significant role in both the prevalence and manifestation of depressive symptoms and alcohol use disorders. Men may respond better to alcohol-focused interventions, while women tend to benefit more from depression-focused treatments [36]. These findings support the importance of tailoring interventions by gender to optimize therapeutic outcomes.



Accumulating evidence has suggested the influence of dietary factors on depression and alcohol consumption in the general population. Recently, studies investigating these two health problems have reported similar results, especially regarding the increased risk of a high-fat diet as a risk factor for depression and alcohol consumption. However, few studies have evaluated the interplay of dietary factors and gender differences in depression and alcohol consumption [48]. A disturbed emotional balance facilitates the adoption of a nutrition pattern that can be classified under the category of an eating disorder, attracting a procession of somatic and psychological consequences including alcohol consumption and depressive disorders [49].

Another important aspect is the lack of clear predictor factors for a response to treatment. Although linear and logistic regression models were tested, none of them identified any significant variable that could provide a correct explanation of the variance in depression scores or the likelihood of a substantial reduction in the symptoms. This may be attributed to several factors such as heterogeneity within the sample, the relatively small sample size for a study with interventional analysis potential, or confounding variables (such as social support, somatic comorbidities, or family history) that were not included in the models.

#### *Limitation of the Study*

A notable limitation of the current study is the relatively small sample size ( $n = 147$ ), which may limit the generalizability of our findings to broader populations. Future research involving larger, possibly multicentric samples is recommended to validate and extend these findings.

Additionally, the quasi-experimental design employed, lacking a control group, limited the ability to control confounding variables such as medication use, physical health status changes, or environmental stressors. Previous studies [50,51] have highlighted limitations associated with traditional statistical models in predicting treatment outcomes in depression, primarily due to individual variability and multiple interacting factors. This could explain the modest predictive power of our statistical models.

Nevertheless, our findings argue for the efficacy of an integrated therapeutic approach addressing concurrent alcohol dependence and depression and anxiety symptoms. Our results corroborate the effectiveness of cognitive-behavioral therapy (CBT) and motivational interviewing (MI), which aligned with existing literature.

One notable limitation is that a detailed alcohol consumption history was not comprehensively captured for every participant, reducing our ability to fully correlate the nuances of intake (e.g., age at onset, beverage type, and cumulative quantities) with depressive or anxiety symptom profiles. Additionally, the absence of significant correlations between biological markers and psychiatric symptom severity did not exclude the potential influence of other biological or psychosocial factors, such as cortisol levels, neuroinflammatory markers, or specific genetic polymorphisms. Future investigations should therefore incorporate more robust, standardized methods of documenting longitudinal drinking behaviors while also examining these additional variables.

Moreover, larger, possibly multicentric samples and controlled study designs are recommended to validate and extend the present findings. Exploring alternative therapeutic modalities—such as online interventions, telepsychiatry, and group therapies—could further enhance our understanding of effective strategies across diverse clinical contexts.

## **5. Conclusions**

The findings underscore the importance of psychological interventions, particularly cognitive-behavioral and motivational approaches, in reducing alcohol consumption and

alleviating depressive and anxiety symptoms. Both the short- and long-term interventions proved beneficial, although the extended interventions were slightly more effective for anxiety. Conversely, depression severity responded favorably regardless of the intervention type, suggesting a need for a personalized approach tailored to a patient's social support level and clinical characteristics.

The absence of significant correlations between alcohol consumption, biological markers (GGT and CDT), and the intensity of the depressive and anxiety symptoms highlights the complexity and multidimensionality of these disorders. Additional psychosocial and genetic factors may influence patient outcomes and account for the variability in therapeutic responses. Moreover, the attempts to identify predictive variables for therapeutic success were limited, indicating the necessity for future studies with larger samples and more complex analytical models.

Overall, the findings confirmed the utility of integrated approaches that simultaneously address both alcohol dependence and depressive and anxiety symptoms. These results can guide the development of personalized treatment programs and inform public health policies aimed at reducing the burden of alcohol–depression comorbidities and improving the quality of life for affected patients.

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**Informed Consent Statement:** Informed consent was obtained from all subjects involved in the study.

**Data Availability Statement:** This paper is part of the doctoral study of Psychologist Simona Dana Mitincu-Caramfil titled “The role of ethanolic consumption in the depressive syndrome development”.

**Conflicts of Interest:** The authors declare no conflicts of interest.

## Abbreviations

The following abbreviations are used in this manuscript:

CBT	Cognitive-behavioral therapy
CDT	Carbohydrate-deficient transferrin
GGT	Gamma-glutamyl transferase
HVLT-R	Hopkins Verbal Learning Test—Revised
MI	Motivational interviewing
PTSD	Post-traumatic stress disorder

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