



## THE ASSOCIATION BETWEEN LONG TERM ALCOHOL CONSUMPTION AND DEMENTIA

Baozhen Dai<sup>1</sup>

Joseph Marfoh<sup>2\*</sup>

<sup>1</sup>Jiangsu University, P. R. China.

<sup>2</sup>Jiangsu University, School of Management, Department of Policy and Management, Jiangsu Province, P. R. China.

Email: [marfohjoseph1@gmail.com](mailto:marfohjoseph1@gmail.com)



(+ Corresponding author)

### ABSTRACT

#### Article History

Received: 21 December 2020

Revised: 5 February 2021

Accepted: 24 February 2021

Published: 16 March 2021

#### Keywords

Alcohol consumption

Dementia

Alcohol/Anxiety disorder

Depression disorder

Bipolar disorder

Schizophrenia.

The study assessed the association between long-term alcohol consumption and risk of dementia in a panel study of 177 countries from 2000 to 2009. The study used econometric techniques to analyze the association between long-term alcohol consumption and risk of dementia. However, it employed the ordinary least square regression method and the fully modified ordinary least square regression method for robust inference. The study found that alcohol consumption is heterogeneously associated with dementia regarding the associated risk factors such as alcoholic disorder, anxiety disorder, depression disorder, bipolar disorder, and Schizophrenia. Moreover, some contributing factors such as age, eating disorder, smoking, and educational level play a significant role in attributing to dementia with the risk factor. However, it is recommended that alcohol consumption be reasonably taken to avoid its consequences. More specifically, ethanol is neurotoxic, crosses the blood-brain barrier to enter neurons directly, and can cause pathological processes leading to brain damage in high concentrations and its metabolite acetaldehyde.

**Contribution/Originality:** This study uses an econometric methodology to assess the association between long-term alcohol consumption and risk of dementia in a panel study. The study found that alcohol consumption is heterogeneously associated with dementia regarding the associated risk factors such as alcoholic disorder, anxiety disorder, depression disorder, bipolar disorder, and Schizophrenia.

### 1. INTRODUCTION

The estimated global prevalence of dementia by 2030 is projected to exceed 74.7 million [1]. This development would have substantial effects on public health and social care, with the expense of caring for people coping with dementia estimated to escalate from USD 818 billion in 2015 to USD 2 trillion in 2030 [1]. Drinking alcohol is a modifiable activity that has emerged as a possible dementia preventive factor. Three high-quality systematic reviews that met their inclusion criteria were listed in a recent analysis of systematic reviews that examined the relationship between alcohol use and dementia or cognitive decline [2]. A meta-analysis was performed in two of the three systematic reviews and concluded that light-to-moderate alcohol intake was associated with a 25-38 percent reduction in risk of Alzheimer's disease (AD), vascular dementia (VaD), and all-cause dementia (ACD) relative to abstainers [3, 4]. The ingestion of alcohol is recognized for its psychotropic effects. The behavioral changes in the nervous system after alcohol consumption differ according to the level of ethanol ingested: psycho-

stimulation if the amount is below 0.5 g/l, and sedation above that dose. A relaxation of inhibitions is associated with psycho-stimulation: cognitive tasks are performed more rapidly, with a sensation of ease, but with a higher rate of error. Notably when driving a car, there is indeed a risk-taking attitude. In heavy drinkers, cognitive dysfunction is often found and memory, visuomotor ability or abstract thinking are influenced. Excessive alcohol intake, also called alcoholic dementia, is also responsible for Korsakoff's syndrome. This condition is actually due to a deficiency of vitamin B1, which is often related to malnutrition in heavy drinkers [5]. Some pathologies like dementia are becoming a public health issue with the aging of the population. The deterioration of fully interconnected cognitive functions, including memory, characterizes dementia. Alzheimer's disease is the most prevalent dementia, and is a degenerative dementia characterized by a gradual worsening of cognitive functions and constitutes 2/3 of all dementia. The prevalence of dementia rises with age and ranges from 1% in people aged 65 years to even more than 28% in people aged 90 years and older [6, 7]. The etiology is still unclear and it is still under way to look for risk factors. One of these risk factors is alcohol intake, and it has been investigated in clusters of subjects aged 65 years and older [6].

There is an increased risk of dementia for individuals with alcohol use disorder [8] and alcohol misuse is a priority for avoiding dementia [9, 10]. Alcohol can cause cognitive impairment with neuronal loss, especially in the frontal cortex [11, 12]; dysfunction of the central nervous system; hypoglycemia; epilepsy; and depression, all of which contribute to the risk of dementia [8, 13, 14]. Although the potential for dementia to be affected by clinical alcohol problems seems evident, the role of total alcohol consumption in dementia development in the overall population is unclear. Meta-analyses of community studies indicate a high prevalence of dementia in people with extreme alcohol consumption relative to moderate alcohol consumption [15, 16] while this result is not universal and has not been confirmed in linear regression studies using alcoholic disorder, anxiety disorder, depression disorder, bipolar disorder, Schizophrenia and alcoholic consumption per capita as dementia and alcoholic consumption proxies, respectively [2, 17, 18]. This present study seeks to establish an association between long-term alcohol consumption and risk of dementia in a panel study adopting econometric techniques.

## 2. METHOD AND DATA

### 2.1. Method

This present study employed an econometric approach in assessing the association between alcohol consumption and the risk of dementia. The econometric model developed in that pursuit can be found below:

$$Dementia = f(\text{alcoholic consumption, eating habit, smoking, age, educational level}) \quad (1)$$

$$\begin{aligned}
 Dementia_{it} & \begin{bmatrix} ALC\_DISOR \\ ANX\_DISOR \\ BIPO\_DISOR \\ DEP\_DISOR \\ SHCZ \end{bmatrix} \\
 & = \beta_0 + \beta_1 ALC\_CON_{it} + \beta_2 EAT\_DISOR_{it} + \beta_3 LOW\_EDU_{it} + \beta_4 HIGH\_EDU_{it} \\
 & + \beta_5\_15\_24_{it} + \beta_6\_25\_64_{it} + \beta_7 OVER\_65_{it} + \beta_8 SMOKING_{it} \\
 & + \varepsilon_{it}
 \end{aligned} \quad (2)$$

Equation 1 simplifies the association between dementia and alcoholic consumption, and it implies that dementia is a function of alcoholic consumption, eating disorder, smoking habit, age, and educational level. In furtherance, Equation 2 elaborates on the econometric model proposed for the study to assess the association between the risk of dementia and alcoholic consumption. In Equation 2, dementia is measured by proxy of alcoholic disorder (ALC\_DISOR), anxiety disorder (ANX\_DISOR), bipolar disorder (BIPO\_DISOR), depression disorder (DEP\_DISOR), and Schizophrenia (SHCZ). Where i represents the cross-section of 177 countries, t represents the

period from 2000 to 2009,  $\beta_1$  to  $\beta_8$  are coefficients of the parameters (independent variables) to be estimated,  $\beta_0$  represents the intercept or constant term, and  $\varepsilon$  represents the error term. Empirically, the study followed some econometric techniques to achieve its objectives. The econometric techniques are (1) unit root test; (2) cointegration test; (3) correlation matrix; (4) ordinary least square regression method, and fully modified ordinary least square regression method. Firstly, the variables are tested for unit root to ascertain their stationarity status. Evidence of unit root in the variables does not warrant further investigation or analysis; it would amount to spurious analysis. Subsequently, after confirmation of no unit root, a cointegration test is performed to identify the long-run relationship or cointegration relationship between the independent and the dependent variables. Therefore, at a 5% significance level, the null hypothesis of cointegration should be rejected. The next approach is to perform a correlation matrix to check for the correlation association among the variables and further check for no multicollinearity evidence. Evidence of no multicollinearity suggests that none of the independent variables has a correlation coefficient of  $-/+0.70$  or above with the dependent variable [19]. However, the final approach is to perform the regression analysis; hence, the ordinary least square regression method. The ordinary least square has a limitation to resolve cross-sectional heterogeneity and simultaneity issues in a panel Kao and Chiang [20]. Pedroni [21]; Pedroni [22] suggests that the fully modified ordinary least square is the best estimator to robust check the ordinary least square method. Therefore, the fully modified ordinary least square method is used as the study's robust check method.

**Table-1. Variable descriptions and measurement.**

Variable	Measurement	Description
ALC_CON	Alcoholic consumption	Alcohol, recorded per capita (15+) consumption (in litres of pure alcohol)
EAT_DISOR	Eating Habit	Prevalence - Eating disorders - Sex: Both - Age: Age-standardized (Percent)
LOW_EDU	low educational level	Share of students achieving the minimum threshold [23]
HIGH_EDU	High educational level	Share of students achieving the advanced threshold [23]
_15_24	AGE 15 TO 24 years	Estimates: Total population by broad age group, both sexes combined (thousands) - Population aged 15-24
_25_64	AGE 25 TO 64 years	Estimates: Total population by broad age group, both sexes combined (thousands) - Population aged 25-64
OVER_65	Age over 65 years	Estimates: Total population by broad age group, both sexes combined (thousands) - Population aged 65 or over
SMOKING	Smoking habit	Age-standardised prevalence of daily smoking in populations aged 10 and older (%) - Past - Unscaled
ALC_DISOR	Dementia	Prevalence - Alcohol use disorders - Sex: Both - Age: Age-standardized (Percent)
ANX_DISOR	Dementia	Prevalence - Anxiety disorders - Sex: Both - Age: Age-standardized (Percent)
BIPO_DISOR	Dementia	Prevalence - Bipolar disorder - Sex: Both - Age: Age-standardized (Percent)
DEP_DISOR	Dementia	Prevalence - Depressive disorders - Sex: Both - Age: Age-standardized (Percent)
SHCZ	Dementia	Prevalence - Schizophrenia - Sex: Both - Age: Age-standardized (Percent)

## 2.2. Data

The study's data were sourced from the World Health Organisation database and the World Bank's World Development Indicators from 2000 to 2009. However, 177 countries are considered in a panel study to critically assess the association between long-term alcohol consumption and risk of dementia. This study's dependent variable is dementia, measured by the proxy of alcoholic disorder, bipolar disorder, anxiety disorder, depression disorder, and Schizophrenia. The independent variable is alcoholic consumption, measured by proxy of alcoholic consumption per capita in liters. Moreover, other variables such as age, eating habits, educational level, and smoking habit are used as control variables. More details about the variables are presented in Table 1. The selection of these variables is on the backdrop of dementia's psychological implication of dementia, such as changes in behavior and personality,

anxiety, depression, mood swings, hallucination, apathy, and agitation. Moreover, alcoholic consumption, age, smoking, etc., inevitably contribute to dementia, hence their selection as control variables [6].

### 3. FINDINGS AND DISCUSSION

#### 3.1. Descriptive Statistics of Variables

Table 2 presents the descriptive statistics of the selected variables for the study. From the table, the mean of variables suggests that alcohol disorder increases by 0.308% annually, and alcoholic consumption per capita increases by 0.876% annually. Meanwhile, bipolar disorder and schizophrenia decrease by 0.343% and 1.594% annually. Moreover, eating disorder decreases by 1.636% annually, but smoking increases by 2.640% annually. The variables' standard deviation suggests that they are homogeneously related, and the Jarque-Bera test confirms that the variables are not normally distributed except for depression disorder.

#### 3.2. Unit Root Test

This section presents the results of the unit root tests performed outlined in table 8. Four unit root tests were performed; thus Levin, et al. [24]; Im, et al. [25], ADF-Fisher Chi-square, and PP-Fisher Chi-square tests [26]. From the results, the variables could not pass all the unit root tests at level form because Levin, Lin & Chu tests showed a higher p-value. Nevertheless, at the first difference, all the tests confirmed a 1% significance level, signaling no evidence of unit root. Therefore, the null hypothesis that there is a unit root in the variables is rejected.

Table-3. Unit root test.

Group unit root test					
Level Form				Cross-	
Method	Statistic	Prob.**	Sig.	sections	Obs
Levin, Lin & Chu t*	35.411	1.000		13	22757
Im, Pesaran and Shin W-stat	-23.126	0.000	***	13	22757
ADF - Fisher Chi-square	607.492	0.000	***	13	22757
PP - Fisher Chi-square	1156.280	0.000	***	13	22997
First Difference					
Method	Statistic	Prob.**		Cross-	Obs
Levin, Lin & Chu t*	-32.524	0.000	***	13	22753
Im, Pesaran and Shin W-stat	-58.095	0.000	***	13	22753
ADF - Fisher Chi-square	2119.980	0.000	***	13	22753
PP - Fisher Chi-square	381.470	0.000	***	13	22984

Note: \*\*\* denotes 1% significance level.

#### 3.3. Johansen Combined Unrestricted Cointegration Test

Evidence from Table 4 confirms that the independent variables are cointegrated with the dependent variable at a 1% significance level from at most 1 to At most 12. Both trace and Max-Eigen tests confirmed statistical significance for the cointegration test. Therefore, the null hypothesis that there is no evidence of cointegration relationship among the variables is rejected. Hence, there is a long-run relationship between the variables.

#### 3.4. Correlation Matrix

The results of the correlation matrix are presented in Table 5. Regarding correlation, alcoholic consumption positively and significantly correlates with the alcoholic disorder, bipolar disorder, anxiety disorder, and Schizophrenia, but negatively and significantly correlates with depression disorder. Consistently, eating disorder and age over 65 years positively and significantly correlates with the alcoholic disorder, anxiety disorder, bipolar disorder, depression disorder, and Schizophrenia, respectively. Also, age between 25 and 64 years positively and significantly correlates with anxiety disorder, bipolar disorder, depression disorder, and Schizophrenia, but insignificantly correlates with the alcoholic disorder. However, for ages between 15 and 24 years, it has a positive and significant correlation with depression disorder and Schizophrenia. Smoking habit positively and significantly

correlates with anxiety disorder, alcoholic disorder, and Schizophrenia, but insignificantly correlates with depressive disorder and bipolar disorder. With respect to educational background, high educational level negatively and significantly correlates with alcoholic disorder, anxiety disorder, and bipolar disorder, but insignificant with depression disorder and Schizophrenia.

**Table-4 Johansen Combined Cointegration test.**

Unrestricted Cointegration Rank Test (Trace & Maximum Eigenvalue)							
Hypothesized	Trace			Hypothesized	Max-Eigen		
No. of CE(s)	Statistic	Prob.**	Sig.	No. of CE(s)	Statistic	Prob.**	Sig.
At most 1 *	1362.013	0.000	***	At most 1 *	149.183	0.000	***
At most 2 *	1212.830	0.000	***	At most 2 *	142.420	0.000	***
At most 3 *	1070.410	0.000	***	At most 3 *	138.436	0.000	***
At most 4 *	931.975	0.000	***	At most 4 *	134.771	0.000	***
At most 5 *	797.204	0.000	***	At most 5 *	123.421	0.000	***
At most 6 *	673.783	0.000	***	At most 6 *	115.116	0.000	***
At most 7 *	558.667	0.000	***	At most 7 *	111.174	0.000	***
At most 8 *	447.493	0.000	***	At most 8 *	102.273	0.000	***
At most 9 *	345.220	0.000	***	At most 9 *	98.492	0.000	***
At most 10 *	246.728	0.000	***	At most 10 *	93.833	0.000	***
At most 11 *	152.895	0.000	***	At most 11 *	83.709	0.000	***
At most 12 *	69.186	0.000	***	At most 12 *	69.186	0.000	***

Note: \*\*\* denotes 1% significance level.

On the other hand, low educational level positively and significantly correlates with Schizophrenia, but negatively and significantly correlates with alcoholic disorder and bipolar disorder. To check for multicollinearity, the results displayed in Table 5 convincingly suggest that there is no evidence of multicollinearity because the independent variable with the highest coefficient values is eating disorder; thus, 0.769 and 0.669 with an anxiety disorder and bipolar disorder, respectively. The second highest value of the coefficient is 0.492 and 0.306; thus, alcoholic consumption with an alcoholic disorder and bipolar disorder. Conversely, the study confidently rejects the null hypothesis of multicollinearity.

### 3.5. Analysing the Association of Long-Term Alcohol Consumption and Dementia: OLS Estimation

The analyses are presented in table 6. The study found that alcohol consumption negatively and significantly associates with anxiety disorder, depression disorder, and Schizophrenia that triggers dementia in the ordinary least square estimation. However, alcohol consumption is positively and significantly associated with an alcoholic disorder, while it is insignificant with bipolar disorder. In the estimation, it was realized that a percentage point increase in alcoholic consumption per capita could lead to a decrease in anxiety disorder (0.038%), depression disorder (0.008%), and Schizophrenia (0.012%) but could increase alcoholic disorder by 0.151% in the long run. Considering the educational background, it is evident that low educational level is negatively associated with an anxiety disorder and bipolar disorder, but positively and significantly associated with depressive disorder and Schizophrenia. On the other hand, a high educational level positively and significantly associates with bipolar disorder but negatively and significantly associates with depression disorder. Eating habits or disorders positively and significantly associates with anxiety disorder, bipolar disorder, depression disorder, and Schizophrenia, but negatively and significantly associates with an alcoholic disorder in the long-run. Considering the magnitude, a percentage point increase in eating disorder could increase anxiety disorder, bipolar disorder, depression disorder, and Schizophrenia by 0.373%, 0.324%, 0.084%, and 0.169%, respectively.

Table-2. Descriptive statistics.

	ALC_DISOR	ANX_DISOR	BIPO_DISOR	DEP_DISOR	SHCZ	ALC_CON	EAT_DISOR
Mean	0.308	1.337	-0.343	1.230	-1.594	0.876	-1.636
Median	0.370	1.256	-0.355	1.246	-1.620	1.378	-1.724
Maximum	1.698	2.194	0.187	1.748	-0.981	2.883	-0.080
Minimum	-0.799	0.705	-1.143	0.786	-1.909	-4.605	-2.572
Std. Dev.	0.495	0.273	0.223	0.187	0.186	1.604	0.589
Skewness	0.230	0.567	-0.285	-0.023	0.845	-1.422	0.556
Kurtosis	3.064	2.674	3.312	2.758	3.648	4.879	2.322
Jarque-Bera	15.934	102.812	31.125	4.479	241.627	856.866	125.250
Probability	0.000	0.000	0.000	0.107	0.000	0.000	0.000
Observations	1770	1770	1770	1770	1770	1770	1770
	HIGH_EDU	LOW_EDU	OVER_65	_25_64	_15_24	SMOKING	
Mean	0.732	1.474	5.863	7.871	7.003	2.640	
Median	0.000	0.000	5.996	7.991	7.141	2.737	
Maximum	4.147	4.595	11.590	13.544	12.380	3.644	
Minimum	-2.303	0.000	1.025	3.472	2.415	0.737	
Std. Dev.	1.264	2.021	2.102	1.962	1.947	0.593	
Skewness	1.283	0.670	-0.101	-0.159	-0.198	-0.613	
Kurtosis	3.169	1.495	2.716	2.975	2.908	2.745	
Jarque-Bera	487.354	299.471	9.006	7.458	12.241	115.779	
Probability	0.000	0.000	0.011	0.024	0.002	0.000	
Observations	1770	1770	1770	1770	1770	1770	

Table-5. Correlation matrix.

Correlation													
Probability	ALC_DISOR	ANX_DISOR	BIPO_DISOR	DEP_DISOR	SHCZ	ALC_CON	EAT_DISOR	HIGH_EDU	LOW_EDU	OVER_65	_25_64	_15_24	SMOKING
ALC_DISOR	1												
ANX_DISOR	-0.174***	1											
BIPO_DISOR	0.102***	0.626***	1										
DEP_DISOR	-0.072**	0.313***	0.113***	1									
SHCZ	-0.082***	0.471***	0.269***	0.054**	1								
ALC_CON	0.493***	0.100***	0.306***	-0.046**	0.242***	1							
EAT_DISOR	0.058**	0.669***	0.769***	0.117***	0.682***	0.426***	1						
HIGH_EDU	-0.044*	-0.113***	-0.132***	-0.037	-0.017	-0.088***	-0.117***	1					
LOW_EDU	-0.044*	-0.038	-0.091***	0.028	0.092***	-0.038	0.00	0.659***	1				
OVER_65	0.099***	0.121***	0.127***	0.121***	0.277***	0.093***	0.191***	-0.015	0.005	1			
_25_64	0.010	0.065**	0.049**	0.135***	0.184***	-0.039	0.082***	0.010	0.018	0.968***	1		
_15_24	-0.021	-0.010	-0.033	0.143***	0.063**	-0.123***	-0.047**	0.011	0.009	0.919***	0.981***	1	
SMOKING	0.062**	0.219***	0.010	-0.007	0.457***	0.178***	0.247***	-0.029	-0.001	0.249***	0.160***	0.068**	1

Note: \*\*\* denotes 1% significance level, \*\* denotes 5% significance level, \* denotes 10% significance level.

But a percentage point increase in eating disorder could lead to a reduction in alcoholic disorder by 0.191%. However, smoking negatively and significantly associates with alcoholic disorder, and bipolar disorder, but positively and significantly associates with anxiety disorder, and Schizophrenia while it is insignificant with depression disorder.

Table-6. Ordinary least square estimations.

OLS	ALC_DISOR	ANX_DISOR	BIPO_DISOR	DEP_DISOR	SHCZ
ALC_CON	0.151 (20.238)***	-0.038 (-11.055)***	-0.003 (-1.442)	-0.008 (-2.550)**	-0.012 (-5.726)***
EAT_DISOR	-0.191 (-8.212)***	0.373 (34.345)***	0.324 (43.084)***	0.084 (8.279)***	0.169 (25.075)***
LOW_EDU	-0.003 (-0.501)	-0.005 (-1.739)*	-0.013 (-6.253)***	0.007 (2.353)**	0.008 (4.009)***
HIGH_EDU	0.002 (0.161)	-0.002 (-0.358)	0.008 (2.381)**	-0.009 (-1.933)**	-0.002 (-0.494)
_15_24	0.047 (1.091)	0.080 (3.966)***	0.054 (3.857)***	0.109 (5.745)***	-0.090 (-7.201)***
_25_64	-0.272 (-4.574)***	-0.092 (-3.308)***	-0.084 (-4.363)***	-0.101 (-3.865)***	0.095 (5.476)***
OVER_65	0.232 (8.816)***	0.010 (0.806)	0.031 (3.671)***	0.005 (0.403)	0.003 (0.334)
SMOKING	-0.045 (-2.316)**	0.050 (5.510)***	-0.069 (-10.992)***	0.006 (0.661)	0.076 (13.553)***
Constant	0.438 (3.891)***	1.962 (37.301)***	0.484 (13.280)***	1.359 (27.564)***	-1.643 (-50.268)***
R <sup>2</sup>	0.307	0.502	0.641	0.361	0.588
Adjusted R <sup>2</sup>	0.034	0.500	0.639	0.360	0.586
F-statistics	97.701***	222.196***	392.282***	15.263***	313.978***
Ramsey Reset test	82.832***	19.492***	121.325***	1.133**	8.011**
observation	1770	1770	1770	1770	1770

Note: \*\*\* denotes 1% significance level, \*\* denotes 5% significance level, \* denotes 10% significance level.

### 3.6. Robust Check: Fully Modified Ordinary Least Square Estimation

In the robust check estimation with the fully modified ordinary least square regression method, some variables showed different results compared to the ordinary least square method (see Table 7). Specifically, alcoholic consumption has an insignificant association with depression disorder; eating disorder insignificantly associates with bipolar disorder and depression disorder; age between 15 and 24 years insignificantly associates with bipolar disorder; age between 25 and 64 years insignificantly associates with an alcoholic disorder and bipolar disorder; age over 65 years insignificantly associates with bipolar disorder, and smoking insignificantly associates with an alcoholic disorder. Nonetheless, the other results remain the same.

### 3.7. Discussion

The study contends that alcohol consumption is heterogeneously associated with dementia regarding the associated risk factors such as alcoholic disorder, anxiety disorder, depression disorder, bipolar disorder, and Schizophrenia. Moreover, some contributing factors such as age, eating disorder, smoking, and educational level play a significant role in attributing to dementia with the risk factor. Specifically, the findings suggest that a high education level has no significant association with dementia, but low educational level associates with dementia significantly. This is in support of the contention of Leonard [6]. According to Watkins [27] there is a connection between alcohol consumption and anxiety, but excessive alcohol use could trigger another level of anxiety disorder and alcoholic disorder, respectively.



Table-7. Robust check estimations: FMOLS

FMOLS	ALC_DISOR	ANX_DISOR	BIPO_DISOR	DEP_DISOR	SHCZ
ALC_CON	0.160 (8.991)***	-0.038 (-4.580)***	-0.004 (-0.653)	-0.008 (-0.952)	-0.015 (-2.911)**
EAT_DISOR	-0.202 (3.651)***	0.379 (14.839)***	0.329 (18.663)***	0.083 (3.327)***	0.177 (10.974)***
LOW_EDU	-0.011 (-0.684)	-0.004 (-0.531)	-0.015 (-3.054)**	0.009 (1.218)	0.011 (2.422)**
HIGH_EDU	0.008 (0.337)	-0.005 (-0.390)	0.009 (1.107)	-0.011 (-0.996)	-0.004 (-0.596)
_15_24	0.002 (0.016)	0.099 (2.075)**	0.051 (1.546)	0.095 (2.032)**	-0.088 (-2.930)**
_25_64	-0.191 (-1.349)	-0.112 (-1.713)*	-0.076 (-1.685)*	-0.094 (-1.464)	0.087 (2.121)**
OVER_65	0.192 (3.065)***	0.014 (0.481)	0.025 (1.257)	0.010 (0.337)	0.007 (0.378)
SMOKING	-0.056 (-1.209)	0.047 (2.219)**	-0.059 (-4.010)***	-0.002 (-0.067)	0.075 (5.595)***
Constant	0.361 (1.344)	1.984 (16.013)***	0.465 (5.442)***	1.390 (11.480)***	-1.615 (-20.713)***
R <sup>2</sup>	0.304	0.502	0.640	0.362	0.586
Adjusted R <sup>2</sup>	0.301	0.500	0.638	0.368	0.584
Hansen Test	1.920	2.356	2.702	1.381	2.229
P-value	< 0.01	< 0.01	< 0.01	< 0.01	< 0.01
observation	1770	1770	1770	1770	1770

Note: \*\*\* denotes 1% significance level, \*\* denotes 5% significance level, \* denotes 10% significance level.

Meanwhile, Kivimäki, et al. [28] understand that individuals' lifestyles could lead to dementia such that healthy living is associated with the risk of dementia. This relates to the finding that eating disorders and smoking habits are significantly associated to the factors that lead to dementia, such as anxiety disorder, depression disorder, Schizophrenia, and alcoholic disorder. Moreover, Andrews, et al. [29] contended that alcohol consumption and Alzheimer's are significantly associated. Perhaps, Alzheimer's in the long-run leads to dementia and is considered a causal agent of dementia. Eating habit is pinpointed as one factor that leads to dementia. That notwithstanding, Killin, et al. [30] opined that lack of vitamin D is associated to the risk of dementia. Also, he understands that smoking is prevalent to the risk of dementia.

#### 4. CONCLUSION

The study assessed the association between long-term alcohol consumption and risk of dementia in a panel study of 177 countries from 2000 to 2009. The study used econometric techniques to analyze the association between long-term alcohol consumption and risk of dementia. However, it employed the ordinary least square regression method and the fully modified ordinary least square regression method for robust inference.

The study found that alcohol consumption is heterogeneously associated with dementia regarding the associated risk factors such as alcoholic disorder, anxiety disorder, depression disorder, bipolar disorder, and Schizophrenia. Moreover, some contributing factors such as age, eating disorder, smoking, and educational level play a significant role in attributing to dementia with the risk factor. Even though alcohol consumption has a negative impact on anxiety; basically, it can have a devastating effect on an individual when it is abused. However, it is recommended that alcohol consumption be reasonably taken to avoid its consequences. More specifically, ethanol is neurotoxic, crosses the blood-brain barrier to enter neurons directly, and can cause pathological processes leading to brain damage in high concentrations and its metabolite acetaldehyde.

**Funding:** This study received no specific financial support.

**Competing Interests:** The authors declare that they have no competing interests.

**Acknowledgement:** Both authors contributed equally to the conception and design of the study.

## REFERENCES

- [1] M. J. Prince, "World alzheimer report 2015: The global impact of dementia: An analysis of prevalence, incidence, Cost and Trends." vol. 2549, ed London: Alzheimer's Disease International, 2015.
- [2] J. Ilomaki, N. Jokanovic, E. CK Tan, and E. Lonroos, "Alcohol consumption, dementia and cognitive decline: An overview of systematic reviews," *Current Clinical Pharmacology*, vol. 10, pp. 204-212, 2015. Available at: <https://doi.org/10.2174/157488471003150820145539>.
- [3] K. J. Anstey, H. A. Mack, and N. Cherbuin, "Alcohol consumption as a risk factor for dementia and cognitive decline: Meta-analysis of prospective studies," *The American Journal of Geriatric Psychiatry*, vol. 17, pp. 542-555, 2009. Available at: <https://doi.org/10.1097/jgp.0b013e3181a2fd07>.
- [4] R. Peters, J. Peters, J. Warner, N. Beckett, and C. Bulpitt, "Alcohol, dementia and cognitive decline in the elderly: A systematic review," *Age and Ageing*, vol. 37, pp. 505-512, 2008. Available at: <https://doi.org/10.1093/ageing/afn095>.
- [5] L. Letenneur, "Risk of dementia and alcohol and wine consumption: A review of recent results," *Biological Research*, vol. 37, pp. 189-193, 2004. Available at: <https://doi.org/10.4067/s0716-97602004000200003>.
- [6] W. Leonard, "Risk factors of dementia. Healthline. Retrieved from: <https://www.healthline.com/health/dementia-risk-factors>," 2017.
- [7] A. Lobo, L. Launer, L. Fratiglioni, K. Andersen, A. Di Carlo, M. Breteler, J. Copeland, J. Dartigues, C. Jagger, and J. Martinez-Lage, "Prevalence of dementia and major subtypes in Europe: A collaborative study of population-based cohorts," *Neurology*, vol. 54, pp. S4-9, 2000.
- [8] M. Schwarzing, B. Pollock, O. Hasan, C. Dufouil, and J. Rehm, "QalyDays study group. Contribution of alcohol use disorders to the burden of dementia in France 2008-13: A nationwide retrospective cohort study," *Lancet Public Health*, vol. 3, pp. e124-e132, 2018. Available at: [10.1016/S2468-2667\(18\)30022-7](https://doi.org/10.1016/S2468-2667(18)30022-7).
- [9] National Academies of Sciences, *National Academies of Sciences, Engineering and medicine: Preventing cognitive decline and dementia—A way forward*: The National Academies Press, 2017.
- [10] WHO, *Risk reduction of cognitive decline and dementia: WHO guidelines*. Washington D.C: World Health Organization, 2019.
- [11] C. Harper, "The neuropathology of alcohol-related brain damage," *Alcohol*, vol. 44, pp. 136-140, 2009. Available at: [10.1093/alcalc/agn102](https://doi.org/10.1093/alcalc/agn102).
- [12] S. Weis and A. Büttner, "Alcohol-related diseases," *Handbook of Clinical Neurology*, vol. 145, pp. 175-180, 2018.
- [13] A. V. Samokhvalov, H. Irving, S. Mohapatra, and J. Rehm, "Alcohol consumption, unprovoked seizures, and epilepsy: A systematic review and meta-analysis," *Epilepsia*, vol. 51, pp. 1177-1184, 2010. Available at: [10.1111/j.1528-1167.2009.02426.x](https://doi.org/10.1111/j.1528-1167.2009.02426.x).
- [14] B. S. Diniz, M. A. Butters, S. M. Albert, M. A. Dew, and C. F. Reynolds, "Late-life depression and risk of vascular dementia and Alzheimer's disease: Systematic review and meta-analysis of community-based cohort studies," *The British Journal of Psychiatry*, vol. 202, pp. 329-335, 2013. Available at: <https://doi.org/10.1192/bjp.bp.112.118307>.
- [15] W. Xu, H. Wang, and Y. Wan, "Alcohol consumption and dementia risk: A dose-response meta-analysis of prospective studies," *European Journal of Epidemiology*, vol. 32, pp. 31-42, 2017. Available at: [10.1007/s10654-017-0225-3](https://doi.org/10.1007/s10654-017-0225-3).
- [16] J. Rehm, J. L. Molinuevo, A. Brugulat-Serrat, C. Falcon, O. Grau-Rivera, M. Suárez-Calvet, J. Pavia, A. Niñerola-Baizán, A. Perissinotti, and F. Lomeña, "Centiloid cut-off values for optimal agreement between PET and CSF core AD biomarkers," *Alzheimer's Research & Therapy*, vol. 11, pp. 1-12, 2019. Available at: [10.1186/s13195-018-0453-0](https://doi.org/10.1186/s13195-018-0453-0).
- [17] A. Ruitenberg, J. C. Van Swieten, and J. C. Wittman, "Alcohol consumption and risk of dementia: The Rotterdam Study," *Lancet*, vol. 359, pp. 281-286, 2002. Available at: [10.1016/S0140-6736\(02\)07493-7](https://doi.org/10.1016/S0140-6736(02)07493-7).
- [18] A. Paganini-Hill, C. H. Kawas, and M. M. Corrada, "Lifestyle factors and dementia in the oldest-old: The 90+ study," *Alzheimer Disease and Associated Disorders*, vol. 30, pp. 21-26, 2016. Available at: [10.1097/WAD.0000000000000087](https://doi.org/10.1097/WAD.0000000000000087).
- [19] Q. Sun, W. Tong, and Q. Yu, "Determinants of foreign direct investment across China," *Journal of International Money and Finance*, vol. 21, pp. 79-113, 2002. Available at: [https://doi.org/10.1016/S0261-5606\(01\)00032-8](https://doi.org/10.1016/S0261-5606(01)00032-8).

- [20] C. Kao and M. H. Chiang, "On the estimation and inference of a cointegrated regression in panel data," *Advances in Econometrics*, vol. 15, pp. 179–222, 2000.
- [21] P. Pedroni, "Fully-modified OLS for heterogeneous cointegration panel," *Advances in Econometrics*, vol. 15, pp. 93–130, 2000.
- [22] P. Pedroni, "Purchasing power parity tests in cointegrated panels," *Review of Economics and statistics*, vol. 83, pp. 727–731, 2001. Available at: <https://doi.org/10.1162/003465301753237803>.
- [23] N. Altinok, N. Angrist, and H. A. Patrinos, "Global data set on education quality (1965-2015) (January 23, 2018)," World Bank Policy Research Working Paper No. 8314, Available at SSRN: <https://ssrn.com/abstract=31081462018>.
- [24] A. Levin, C.-F. Lin, and C.-S. J. Chu, "Unit root tests in panel data: Asymptotic and finite-sample properties," *Journal of Econometrics*, vol. 108, pp. 1–24, 2002. Available at: [https://doi.org/10.1016/s0304-4076\(01\)00098-7](https://doi.org/10.1016/s0304-4076(01)00098-7).
- [25] K. S. Im, M. H. Pesaran, and Y. Shin, "Testing for unit roots in heterogeneous panels," *Journal of Econometrics*, vol. 115, pp. 53–74, 2003. Available at: [https://doi.org/10.1016/s0304-4076\(03\)00092-7](https://doi.org/10.1016/s0304-4076(03)00092-7).
- [26] G. S. Maddala and S. Wu, "A comparative study of unit root tests with panel data and a new simple test," *Oxford Bulletin of Economics and statistics*, vol. 61, pp. 631–652, 1999. Available at: <https://doi.org/10.1111/1468-0084.61.s1.13>.
- [27] M. Watkins, *The connection between anxiety and alcohol*. United States of America: American Addiction Centers, 2019.
- [28] M. Kivimäki, A. Singh-Manoux, G. D. Batty, S. Sabia, A. Sommerlad, S. Floud, M. Jokela, J. Vahtera, M. A. Beydoun, and S. B. Suominen, "Association of alcohol-induced loss of consciousness and overall alcohol consumption with risk for dementia," *JAMA Network Open*, vol. 3, pp. e2016084–e2016084, 2020.
- [29] S. J. Andrews, A. Goate, and K. J. Anstey, "Association between alcohol consumption and Alzheimer's disease: A Mendelian randomization study," *Alzheimer's & Dementia*, vol. 16, pp. 345–353, 2020. Available at: [10.1016/j.jalz.2019.09.086](https://doi.org/10.1016/j.jalz.2019.09.086).
- [30] L. O. J. Killin, J. M. Starr, and I. J. Shiue, "Environmental risk factors for dementia: A systematic review," *BMC Geriatr*, vol. 16, p. 175, 2016. Available at: <https://doi.org/10.1186/s12877-016-0342-y>.

Views and opinions expressed in this article are the views and opinions of the author(s), International Journal of Medical and Health Sciences Research shall not be responsible or answerable for any loss, damage or liability etc. caused in relation to/arising out of the use of the content.