Analytical Assessment of the Effects of Alcohol Consumption on Human Population

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Abstract

Alcohol is psychoactive drug found in beer, wine and hard liquor, whose effects, both pleasant and unpleasant is well documented. Recognition of the consequences of alcohol and abuse on physical and mental health as well as socio-occupational life are necessary steps for initiating appropriate action to reduce the harm/dangers from alcohol consumption. This work was motivated by the observed and anticipated negative health burden with its concomitant socio-economic consequences which the nation is bound to face if systematic efforts are not made now to control the growing problem of alcohol consumption. Three methodologies have been combined in the execution of this research. The first methodology involved conducting the clinical test to determine the independent assessment of impact of alcohol using Digital Display Alcohol Tester (DDAT). Secondly, sample populations of people treated at the Jos University Teaching Hospital from diseases emanating from alcohol were collected, statistically analyzed using Statistical Packages for Scientific Studies (SPSS), and relevant coefficients were deployed for the coding of the simulation model. Thirdly, simulation software was developed using the indices collected from the statistical software to assess the impact of alcohol in the next 50 years on consumers as will be discussed in the next publication. This is to assist decision makers to formulate public policy to stem the inherent catastrophe associated with alcohol consumption. The software is capable of providing approximate prediction of likely deaths that may occur via diseases such as: liver, brain damage, heart and hypertension. It is expected that the deployment of this Alcohol Simulator will guide public policy trust on Alcohol Consumption.

Keywords: Alcohol consumption, software, disease, simulation, model

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1.0 Introduction

Alcohol is an organic chemical containing one or more hydroxyl groups. Common alcohol includes ethanol (the type found in alcohol beverages) **[1].** Alcohol is a colorless volatile flammable liquid, C_2H_5OH synthesized or obtained by fermentation of yeast, sugar and starches and is widely used, either pure or denatured, as a solvent and in drugs, cleaning solutions, explosives, and

intoxicating beverages. It is used as a recreational drug in most societies. Alcohol is a drug that is taken for no medical reasons – usually for mood – altering effects.

Ethyl alcohol, (ethanol) or simply alcohol is a psychoactive drug found in beer, wine, and hard liquor. Alcohol is considered a drug because a drug causes changes in a person's physical and emotional state. The changes caused by drinking alcohol are known as intoxication. The drinking of alcohol beverages or simply alcohol has two faces: one happy and other sad. The moderate drinking of alcohol can make the heart of man rejoice, says the Bible (Psalm 104:15) **[2].** The same Bible also warns that misuse can cause harm or can even be deadly, like the bite of a poisonous snake (Proverb 23:31, 32) **[2].**

Alcohol (ethanol) is the favorite mood-altering drug in most parts of the world and its effects both pleasant and unpleasant are well-documented. What may not be well known is the fact that alcohol is a toxic drug that produces pathological changes (cirrhosis) in liver tissues and can cause death.

Alcohol is a general term denoting a family of organic chemicals with common properties. Members of this family include ethanol, isopropanol, methanol, and others. The most commonly ingested among this group is ethanol. Alcohol (ethanol), is a clear volatile liquid that burns (oxidizes easily with a slight characteristic odour and is very soluble in water. Alcohol is an organic compound composed of carbon, oxygen and hydrogen. Its chemical formula is CH_3CH_2OH.

2.0 Theoretical Background

Many people associate the effect of alcohol on the body with the heart, lungs, liver, brain, memory, etc. If asked about effects of drinking alcohol on terms of physical fitness, the answer is development of beer belly. Drinking too much of alcohol will end up storing more calories as fat **[3].** Many people will choose low calorie alcohol drinks or low carbohydrate alcohol beverages in an attempt to avoid the fat storage issue. By making this choice, they feel that only bad effects of alcohol – increased fat storage, will be minimized. But what we do not know is that only 5% of the calories from alcohol are stored as fat **[3].**

The effects of alcohol on the body are far damaging than can be predicted by the number of empty calories in some alcohol beverages.

Alcohol really affects the amount of fat that can and will burn for energy. In a study done by the American Journal of Clinical Research**, [4]** they concluded that just a mere 24g of alcohol consumption showed whole-body lipid oxidation (the rate at which the body burns fat), decrease by a whopping 73%.

Alcohol consumption has far reaching Implications:

- The drinker consumes a couple of alcohol drinks or more.
- The liver metabolizes that into acetate
- The body uses the acetate for fat as fuel.

In another American Journal of Clinical Ntrition study, there was evidence to suggest that consumption of alcohol leads to an increase in appetite over that of any other carbohydrate type drink**. [4]**

A study of 8 healthy male–volunteers observed that after drinking alcohol, the effects of a significant decrease in testosterone and an increase in cortisol (a muscle destroying hormone) lasted up to 24 hours. **[5]**

A common side effect of alcohol is dehydration. Alcohol is a diuretic. Drinks

containing 4% alcohol tend to delay the recovery process **[6].**

Alcohol consumption, especially at the times when one would normally sleep, can have effects on the quality of sleep. Alcohol consumption can include sleep disorders by disrupting the sequences and duration of sleep states and by altering total sleep time as well as the time required to fall asleep **[7].**

The brain is extraordinarily sensitive to alcohol. Alcohol is a depressant drug that reduces the pace of brain activities by a combination of effects. Alcohol affects the function of neurotransmitters (NTs) by altering the communication between them **[8].** A sufficient dose can shut down brain function. This can lead to unconsciousness or death. Alcohol dependence occurs through changes in the brain caused by prolonged exposure. Prolonged exposure to alcohol can cause the brain to become dependent on alcohol in order to maintain an appropriate level of brain activity.

Ethanol facilitates the action for the major depressant neurotransmitter (Gama Aminobutyric Acid, GABA) in the brain and inhibits the action of the major excitatory neurotransmitter in the brain (Glutamates). By influencing the action of these receptors, ethanol "slows down" the functioning of the nervous system. Thus ethanol is called a central nervous system (CNS) depressant.

Using imaging with computerized tomography, two studies **[9,10]** compared brain shrinkage, a common indicator of brain damage, in alcoholic men and women and reported that male and female alcoholics both showed significantly greater brain shrinkage than control subjects. Studies also show that both men and women have similar learning and memory problem as a result of heavy drinking.

Up to 80 percent of alcoholics have a deficiency in thiamine and some of these people go on to develop serious brain disorders such as Wernicke-Korskoff Syndrome (WKS) [**11].** WKS is a disease that consists of two separate syndromes, a short-lived and severe condition called Wernick's encephalopathy and a long-lasting and debilitating condition known as korsakoff's psychosis.

Researchers studying the effects of alcohol use on the brain are aided by advanced technology such as Magnetic Resonance Imaging (MRI), Diffusion Tensor Imaging (DTI), Position Emission Tomography (PET), and Electrophysiological Brain Mapping. These tools are providing valuable insights into how alcohol affects the brain's structure and function.

Long-term heavy drinking may lead to shrinking of the brain and deficiencies in the fibres (white mater) that carry information between brain cells (gray matter). MRT and DTI are being used together to assess the brains of patients when they first stop chronic heavy drinking and again after long periods of sobriety, to monitor for possible relapse to drinking.[**12].**

The World Health Organization Technical Committee on Cardiovascular Disease asserted that the relationship between moderate alcohol consumption and reduced death from heart disease could no longer be doubted. [**13].**

Consumption of red wine may be particularly favourable, since red wines contain certain polyhenol antioxidants associated with cardiovascular health.

The cardiovascular effects of alcohol are not all beneficial; however, studies show that large quantity consumption of alcohol can lead to alcoholic cardiomyopathy, commonly known as "holiday heart syndrome". Heavy ethanol consumption

leads to dilated cardiomyopathy, which refers to low cardiac output and enlargement of the heart and its chambers **[14].**

Most people realize that heavy, long – term drinking of alcohol can damage the liver, the organ chiefly responsible for breaking down alcohol into harmless by-products and clearing it from the body. But people may not be aware, that prolonged liver dysfunction such as liver cirrhosis resulting from excessive alcohol consumption, can harm the brain, leading to a serious and potential fatal brain disorders known as hepatic encephalopathy [**15**]. In serious cases, patients may slip into a coma (i.e. hepatic coma), which can be fatal.

New imaging techniques have enabled researchers to study specific brain regions in patients with alcoholic liver disease, giving them a better understanding of how hepatic encephalopathy develops. These studies have confirmed that at least two substances, ammonia and manganese; have a role in the development of hepatic encephalopathy. Alcohol-damaged liver cells allow excess amount of these harmful by-products to enter the brain, thus, harming brain cells [**16**].

Researchers of Brigham and Women Hospital in Boston have found that consuming moderate amount of alcohol, about one drink a day, may prevent kidney function decline in men [**16].**

The researchers examined patient blood samples collected from more than 11,000 men enrolled in the then on-going physician's health study. They found that men who consumed at least seven drinks per week were at a 30 percent lower risk of elevated levels of a compound called creatinine in the blood, compared to men who had one or no drinks per week. High blood creatinine levels are a strong indicator of kidney dysfunction **[16].**

Fig 1.0 Parts of the Body Affected By Alcohol. Source: Awake October, 2005

Table 1: *Effects of Ethyl Alcohol on Electrolytes*

Problem	**Major Cause(s)**
Low Sodium Level (hyponatremia)	Massive intake of solute-free fluid (e.g bear)
Low potassium (hypokalemia)	Dietary deficiency, gastric loses, leaky membranes, shift from extracellular to intracellular
Low phosphorus level (hypophosphatemia)	Dietary deficiency, malabsorption
Low magnesium level (hyomagusamia)	Dietary deficiency, malabsorption, phosphorus deficiency

Source: [17]

A study shows that ALCOHOL Abuse is said to cost Ireland's four million people at least one billion dollars annually [**17**]. A source quoted in the Irish Times stated that this sum is equal to "the price of a new hospital, a sports stadium, and a jet for every Minister every year".

In 1998, the Minichi Daily News reported that the economic impact of heavy drinking in Japan was "more than 6 trillion yen [$55 million] yearly". A report to the U.S Congress declared "The estimated economic cost of alcohol abuse was $184.6 billion for 1998 alone, or roughly $638 for every man, woman and child living in the United States that years" **[2].**

In his study, Edebatu [**18]** stated that in Awka, Anambra State, out of ten different drinking spots, there are more than four persons who purchased at least four bottles of the drinks as shown in the table below for a period of 3 months.

Table 2: Cost Analysis of Alcohol Consumption

Drink	**Unit Price**	**Qty Taken**	**Total Cost**	**Total Per 6-day week**	**Total per 52-week year**
Star	N150	4	N600	N3600	N187,200=
Guilder	N150	4	N600	N3600	N187,200=
S/Stout	N150	6	N900	N5400	280,800=
Heineken	N180	5	N900	5400	N280,800=
Total Annual Cost					**N936,000=**

Source: [18]

This study reveals that these groups spend about N936,000.00 which, as at the period of study, was enough to train about ten students in the University.

Furthermore, the study informs that majority of the people understudied were Civil Servants. Analysis of the highest expected income from these groups to show the percentage of annual income spent on alcohol is shown in table 3 below.

Table 3: Percentage of Annual Income spent on Alcohol consumption

Monthly Income	Annual Income	Annual Cost of Alcohol Consumption	% of Annual Income on Alcohol
N60,000.00	N720,000.00	N187,200.00	26%
N60,000.00	N720,000.00	N280,800.00	39%
N70,000.00	N840,000.00	N187,200.00	22%
N70,000.00	N840,000.00	N280,800.00	33.4%
N80,000.00	N960,000.00	N187,200.00	20%
N80,000.00	N960,000.00	N280,800.00	29.3%

Source: [18]

It is suicidal for anybody who earns less than N60,000.00 monthly to be part of these groups.

3.0 Methodology

Three types of methods were adopted in this work:

The first methodology involved conducting the clinical test to determine the independent assessment of impact of Alcohol Consumption. This was carried out with *Digital Display Alcohol Tester (DDAT).*

Secondly, a sample population of people treated at the Jos University1: Teaching Hospital from diseases emanating from Alcohol Consumption2: were collected, statistically analyzed and relevant coefficient were deployed for the coding of the simulation model. 3:

The third methodology which is the development of a simulation model to predict the negative impact of Alcohol Consumption will be discussed in the next edition of this journal.

3.1 Steps Involved In Using Digital Display Alcohol Tester

To use the Digital Display Alcohol test the following steps are involved: 2:

Step1: ***Warm up step***

1: Press the power button; the tester is turned on with one beep sound.

2: The tester starts the 'countdown timer from 20 to 00 on the display and finally the LCD displays 0.00. At this point it is ready for Blood Alcohol Level (BAL) test.

Step 2: Test

Near and blow into the breath inhaler for seconds

Read the test result on the LCD (the level of Alcohol in the consumer's blood in mg/L

The buzzer continuously sounds alarm quickly if the Alcohol concentration or level is up to the level:

- Over and equal to 0.05% BAC
- Over and equal to 0.08% BAC
- Over and equal to 0,25mg/L
- Over and equal to 0.50mg/L

Step 3: Power Off

1: Press the power button to turn off the tester

The "OFF" is displayed on the LSD if the device is idle for 100 seconds. At that point the power button is pressed to turn off the equipment.

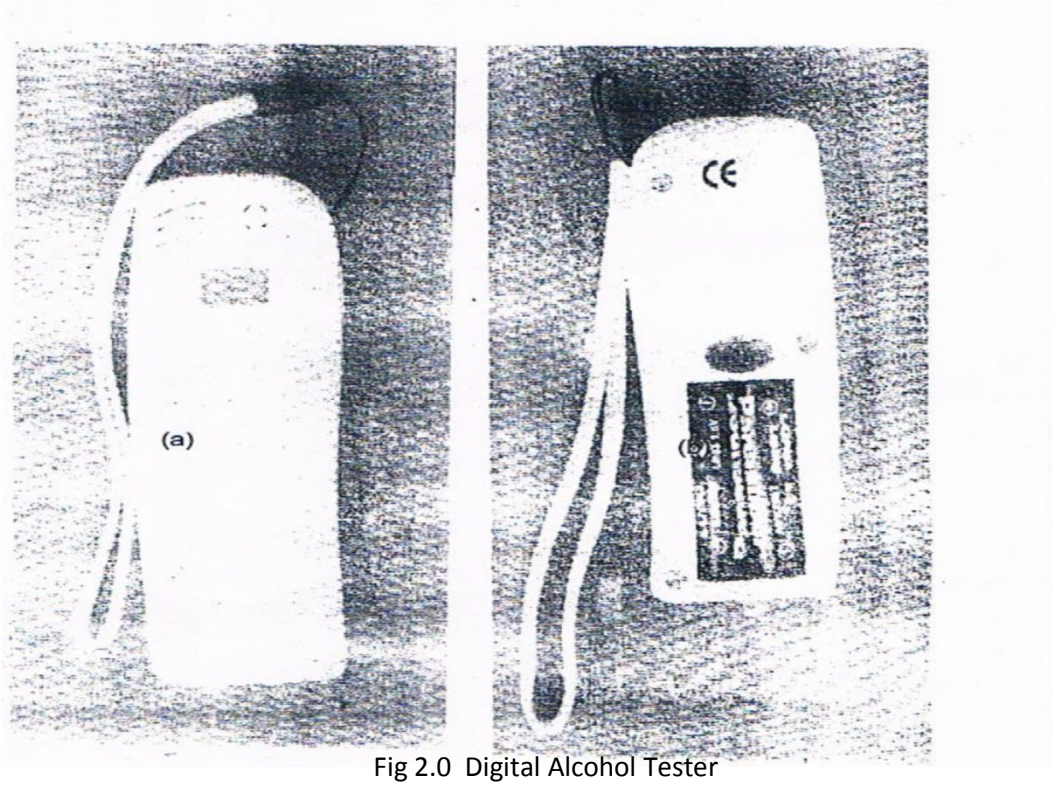

Fig 2.0 Digital Alcohol Tester

3.2 Recording Format For Experimental Result

The table of comparison for Alcohol in the blood between the mg/l and BNC is shown below:

Table 4: Comparison of Experimental Result With Actual BAC Values

The level of Alcohol display in mg/l mg/L	The level of Alcohol display in BAC
0.05	0.01
0.10	1.02
0.20	0.04
0.25	0.05
0.30	0.06
0.40	0.08
0.55	0.11
0.60	0.12
0.70	0.14
0.80	0.16
0.90	0.18
1.00	0.20

Source: JUTH [18]

The blood Alcohol level as recorded on the LCD display is in mg/L. The actual Blood Alcohol Concentration (BAC) is obtained by dividing the value on the LCD display by five (5).

3.3 Presentation and Analysis of Data

This section deals with the analysis of the collected (secondary) data from the Jos University Teaching Hospital, JUTH. Regression model will be developed to estimate the death rates resulting from the Alcohol Consumption in the society. The descriptive statistics of each of the variables will be calculated. These include: the mean, the standard deviation and the correlation between the various consumption of Alcohol –impact

4.0 Data Source

The source of the data for the work is Jos University Teaching Hospital, JUTH, Data is collected on the following variable over a period of 24 months, from the records of patients suffering from Alcohol related killer diseases.

- Liver disease
- Hypertension
- Hepatitis
- Brain damage

From the records, the total number of deaths resulting from these diseases was also recorded. Under the period, about 363 patients were found to be suffering from these diseases. Information was also collected on age of these patients

4.1 Data Arrangement

Table 5: Number of Deaths From Four Killer Diseases

S/no	Month	No of death	Liver x_1	Lung disease x_2	Hepatitis x_3	Brain damage x_4	Total No of patients
1	Jan.	1	1	0	0	0	19
2	Feb.	2	1	0	1	0	18
3	March 2005	5	4	0	1	0	18
4	April	4	2	2	0	0	23
5	May	1	1	0	0	0	22
6	June	0	0	0	0	0	11
7	July	1	1	0	0	0	19
8	August	2	1	0	1	0	18
9	Sep.	4	1	0	2	1	16
10	Oct.	3	2	0	1	0	20
11	Nov.	3	0	3	0	0	18
12	Dec.	2	0	2	0	0	19
13	Jan. 2006	0	0	0	0	0	24
14	Feb	4	4	0	0	0	10
15	March	10	0	4	0	6	17
16	April	1	0	1	0	0	16
17	May	1	1	0	0	0	19
18	June	3	1	0	1	1	19
19	July	5	4	1	0	0	17
20	August	8	6	1	1	0	28
21	Sept.	5	5	0	0	0	12
22	Oct.	3	2	1	0	0	7
23	Nov	2	1	1	0	0	6
24	Dec.	1	0	1	0	0	5

Source: JUTH [18]

4.2 Descriptive Statistics

A preliminary descriptive statistics were done on the data collected for the period of 24 months. The mean number of deaths, their correlation as well as standard

deviations were identified. The four identified diseases caused by cigarette smoking were analyzed separately. Below are the computational formula used:

$Y = \beta_1 x1 + B_2 x_2 + \ldots \beta_n x_n$

Mean (X) = $\sum_{i=1}^{n} xi^2$ (1.1)

X-number of deaths in each month

Standard deviation (SD) =

$$\sqrt{\frac{1}{N}\sum_{i=1}^{N} x_i^2 - \left(\frac{1}{N}\sum_{i=1}^{N} x_i\right)^2}. \quad (1.2)$$

And

$$\text{Correlation (r)} = \frac{n\sum x_i y_i - \sum x_i \sum y_i}{\sqrt{n\sum x_i^2 - (\sum x_i)^2}\ \sqrt{n\sum y_i^2 - (\sum y_i)^2}}. \quad (1.3)$$

Where

X = no of deaths from one disease

Y = no of deaths from another disease

.

Mean and Standard Deviation

Liver = x_1 = 1.6818

SD_1 = 1.78316

Hypertension = x_2 = 0.8636

SD_2 = 1.20694

Hepatitis = x_3 = 0.3182

SD_3 = 0.56790

Brain Damage = x_4 = 0.0909

SD_4 = 0.29424

Total number of patients x = 16.5455

$Y = \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4 + e$ (1.4)

Where

Y = Total number of patients

X_1 = number of expected deaths from liver disease

X_2 = number of expected deaths from hypertension

$Y = \beta x + e$, $y = \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \beta_4 x_4 + e$

Using the software "statistical packages for scientific studies (SPSS)", the following results were obtained

SD = 5.90179

4.3 Regression Analysis

A regression model intended to develop is a non-intercept multiple regression model. This model will help to explain the proportion of deaths that can be attributed to liver disease, hypertension, hepatitis and Brain damage, out of the total number of patients. The proposed non-intercept model is of the form:

X_3 = number of expected death from hepatitis

X_4 = number of expected death from Brain

E = error term.

β_1 β_2 β_3 and β_4 are the model parameters that will be estimated using the following formulas:

Putting this in matrix form we have,

$Y = \beta x + e$

Where

$$Y = \begin{Bmatrix} y_1 \\ y_2 \\ \vdots \\ y_n \end{Bmatrix} \quad X \quad = \begin{Bmatrix} X_{11}\ X_{12}\ X_{13}\ X_{14} \\ X_{21}\ X_{22}\ X_{23}\ X_{24} \\ X_{31}\ X_{32}\ X_{33}\ X_{34} \\ \cdots\cdots\cdots\cdots \\ X_{n1}\ X_{n2}\ X_{n3} \ldots X_{n4} \end{Bmatrix}$$

$$e = \begin{Bmatrix} e_i \\ e_2 \\ \vdots \\ e_n \end{Bmatrix}$$

Using method of least squares, we can derive the estimate of β_1 as follows:

$$e=Y-\beta X \qquad (1.5)$$

It is intended to minimize the sum of squares of the error term.

$$SSe - e^1e = (Y - X\beta)^1 (Y-X\beta)$$
$$= Y^1Y-Y^1X\beta -\beta^1X^1Y -\beta^1X^1X\beta$$

$$SSe = y^1y-2\beta^1x^1y+\beta^1x^1x\beta \qquad (1.6)$$

Differentiating (1.6) wrt β and equating to zero, we obtain

$$\frac{dSSe}{d\beta} = 2x\ ^1y-2\beta^1x^1x=0$$
$$2\beta^1= 2\ X\ ^1Y$$
$$\beta= X^1Y \quad = \frac{(X^1X)^{-1}\ (X^1Y)}{X^1X} \qquad (1.7)$$

The fitted model will be tested for its adequacy using an ANOVA table.
The hypothesis of interest is:
H_0: $B_1= B_2= B_3= B_4=0$
H_a: $B_1\neq B_2\neq B_3\neq B_4\neq 0$

Table 6: Anova Table to Test Model Adequacy

Source of variation	Df	Sum of square	Mean sum of square	F-ratio
Regression	K	SSR	MSR	MSR/MSe
Error	N-K-1	SSe	MSe	
Total	N-1	SST		

Where
K = number of parameters in the model
N = number of observations per variable

$$SSR = \hat{\beta}^1 X^1 Y - NY^2 \quad . (1.8a)$$

$$SSe = Y^1 Y - \hat{\beta} X^1 Y \quad (1.8b)$$

$$SST = Y^1 Y - NY^2 \quad . (1.8c)$$

$$MSR = SSR/K \quad (1.8d)$$

$$MSe = SSe/(N-K-1). \quad (1.8e)$$

The model is significant if F-ratio > F table at N-K-1, N-1 degrees of freedom and 5% level of significance or if the P-value given by the computer is less than 5%.,

If the model is significant it does not mean that all the parameters are significantly different from zero. So, we still need to test for each individual parameter significance using t-test given by

$$t = \frac{\beta_i}{\sqrt{Se(\beta)}} = \frac{\beta i}{\sqrt{\delta^2 C_{ii}}} \quad (1.9)$$

Where
β = Coefficient of intercept
δ^2 = MSe
C_{ii} = Diagonal element of $(X^1X)^{-1}$

The whole analyses were done using SPSS. The results are hereby presented.

$$Y=3.095X_1+4.782X_2+8.329X_3+1.911X_4 \quad (1.10)$$

This model was tested for significance using the Anova table below.

Table 7: Anova table presented from SPSS.

Model	**Sum of square**	**Df**	**Mean sum of squares**	**F**	**P-value**
Regression	4558.205	4	1139.551	9.341	0.000
Error	2195.795	18.	121.989		
Total	6754.000	22			

Using a significance level of 5%, the computed model is significant. That is, we reject H_o and accept H_a saying that at least one of the parameters is significantly different from zero. To know which of these B_i is significantly different from zero, we have the following t-test results.

Table 8: T- Test table for test of significance

Coefficient	t-value	P-value	Remark
B_1 =3.095	2.556	0.020	Significant
B_2 =4.782	2.856	0.010	Significant
B_3 =8.329	1.337	0.198	Not significant
B_4 =1.911	0.163	0.872	Not significant

4.4 Correlation

Table 9:Correlation Table

	R^2	P-value	Remark
Liver Vs Hypertension	0.257	0.124	Not significant
Liver Vs. Hepatitis	0.470	0.014	Significant
Liver Vs. Brain damage	0.125	0.290	Not significant
Hypertension Vs Hepatitis	0.049	0.415	Not significant
Hepatitis Vs Brain Damage	0.000	0.5	Not significant
Hepatitis Vs Brain Damage	0.707	0.000	Significant

4.5 Discussion of Results

The above result shows that during the period under study, an average of 1.6818 persons died monthly as a result of Alcohol related liver disease, while an average of 0.8636 persons died monthly from hypertension with standard deviations of 1.78316 and 1.20694 respectively. The average numbers of persons dying from hepatitis and Brain Damage monthly with their standard deviations are 0.3182 with SD of 0.5680 and 0.0909 with SD of 0.294 respectively. This shows that more deaths are recorded from liver disease than any other disease. This may be due to the fact that Alcohol directly has an impact on the liver since it makes the liver to over work.

On the total number of patients suffering from the four killer diseases monthly, an average of 16.5455 with standard deviation of 5.9079, this figure is alarming since it may lead to more deaths being recorded monthly if not checked.

Correlation shows the degree of linear (one on one) relationship between two variables. Thus, when a correlation value is recorded, it simply shows the strength of linear relationship between a pair **[18].**

There may be other strong or more powerful relationship between the pair that is not linear. Our analysis is purely on linear relationship. From the above correlation table, a correlation value of 0.257 was computed between the number of deaths recorded from liver disease and Hypertension. This value was not significant at 5% level of significance, but

shows a positive weak correlation between the pair. This value further shows that the number of deaths recorded from both diseases either increases or decreases together over the period under study. A very significant value of correlation was recorded between liver disease and Hepatitis. The figure of 0.470 with a P-value of 0.014 which shows that it is significant at 5% indicates that there is a moderate positive correlation, between the number of deaths recorded from liver disease and that recorded from hepatitis. Both diseases are moving in the same pattern. Deaths recorded from liver and Brain damage had no significant relationship as a value of 0.125 and a P-value of 0.290 were recorded. Hypertension and Hepatitis also recorded a poor relationship as well as Hypertension and Brain damage which recorded correlation values of 0.049 and 0.000 respectively. The highly significant pair is between Hepatitis and Brain damage which recorded a correlation value of 0.707 with a P-value of 0.000. This shows that there is a strong positive correlation between Hepatitis and Brain damage.

4.6 Coefficient of Multiple Determinations (R^2)

The R^2, tells us about the amount of variation in Y (total number of patients) that is accounted for by the number of death from liver, hypertension, Hepatitis, and Brain damage. Thus for our model, the R^2 computed is 0.603, which shows that about 60.3% of the variation in y can be accounted for by X_1,X_2,X_3 and X_4.

4.7 Interpretation of Results

The model $Y = 3.095X_1 + 4.782X_2 + 8.329X_3 + 1.911X_4$ shows that for every unit death as a result of liver disease, about 3.095 persons are patients suffering from any of the four diseases. Also for every unit death as a result of hypertension, about 4.782 patients are suffering from any of the four diseases. Also, for every unit death in hepatitis, 8.329 patients are suffering from the four diseases. In a similar manner, for every unit death as a result of brain damage, about 1.911 persons will be suffering from the four diseases as a result of a unit of death from brain damage.

The test for the parameter significance shows that only death from liver disease and hypertension are significant at 5%. The number of deaths from hepatitis and brain damage are not significant at 5%. This does not mean that there are no deaths recorded in these diseases, but that the number of deaths recorded as a result of these diseases is not significant. This shows that B_1 and B_2 can be used for future predictions with certainty, but the prediction to be made with B_3 and B_4 may not be accurate.

5.0 Summary

A study of the impact of Alcohol has been carried out for about 50years. It has been found that Alcohol consumption in the main source of liver, heart, kidney and related diseases including Hypertension and death. There is need to establish some level of control over Alcohol consumption to prevent untimely deaths of the larger population of citizens.

5.1 Conclusion

Abuse of Alcohol can threaten the longevity and existence of a community and the whole nation. The study reveals that if Alcohol abuse is not controlled, the consequences on the Nigerian population are far–reaching and have the potential of decimating the Nigerian population or rendering a large proportion of the population productively impotent. It has the same potency like a nation in a state of war.

5.2 Recommendation

There is an overwhelming evidence from the study that the negative consequences of Alcohol abuse is enormous and therefore requires government intervention. We therefore recommend the following:

- There is immediate need for federal, state and local governments to create or expand job opportunities for our teaming unemployed youths who have taken to Alcohol as the only source of stress reduction or escape.
- Knowledge of health implications of Alcohol abuse and Alcoholism should form part of secondary school curriculum.
- The Federal government should enact laws restricting the importation of hard liquor and local production and consumption of hot drinks. This will invariably reduce the number of Liver Cirrhosis patients in Nigeria.
- There should be federal government policy trust on the sale and consumption of Alcohol at prohibited areas such as motor parks and secondary schools' environment. Liquor licenses should attract heavier licensing fee.
- The federal government should as a matter of policy implement the use of Digital Display Alcohol Tester (DDAT) by the Federal Road Safety Commission (FRSC) worked in all Nigerian motor parks. There should be enactment and enforcement of Driving Under Intoxication (DUI) law to provide strict sanctions for any driver whose Blood Alcohol Concentration (BAC) level is found to exceed maximum standard for driving.

5.3 Area of further work

The follow up article will focus on the development of Computer Simulation model where SSADM and OOADM will be deployed to study software design structure and eventual simulation.

References

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