

Analytical Assessment of the Negative Impacts of Marijuana Smoking on Human Population

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Abstract

Smoking is a practice in which a substance most commonly called cannabis is burnt and the smoke tasted or inhaled. The aim of this work is to design software that will predict the negative impact of marijuana smoking on consumers. This will help in recognizing the consequences of smoking marijuana and abuse on physical and mental health as well as socio-occupational life and also act as a necessary step for initiating appropriate action to reduce the harm/danger from smoking. 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 marijuana smoking. The methodologies for the execution of this research work were the statistical analysis of a sample population of people treated at the Imo State teaching hospital (IMSUTH from diseases emanating from marijuana smoking using Statistical Packages for Scientific Studies (SPSS) and deployment of the relevant coefficients from the analysis to develop a systematic forecasting software model used in simulating for the next 30 years. The simulation is intended to be predictive and to enable policy makers to see the impact and dangers of marijuana smoking between now and the next 30 years if current abuse is not controlled .That is the number of smokers likely to contract liver, brain and related diseases and who are most likely to die from these diseases. The software provides approximate prediction of likely deaths that may occur via diseases such as: liver, brain damage, heart and hypertension.

Keywords: Simulation, Model, Disease, Tetrahydrocannabinol, marijuana smoking.

1.0 Introduction

Smoking is a practice in which a substance, most commonly called cannabis, is burned and the resulting smoke (consisting of particle and gaseous) tasted or inhaled. German scientists identified a link between smoking and lung cancer in the late 1920s, leading to the first anti-smoking campaign in modern history, albeit one truncated by the collapse of the third Reich at the end of the second world war [1]. In 1950, British researchers demonstrated a clear relationship between smoking and cancer. This is primarily practiced as a route of administration for recreational drug use, as combustion releases the active substances in drugs such as Tetrahydrocannabinol and makes them available for absorption through the lungs. The most common method of smoking today is through pipes, joint, blunt, bubble, gravity bong and bongs. It has been suggested that smoking related disease kills one half of all long term smokers but these diseases may also be contracted by non-smokers. A 2007 report states that about 4.9 million people worldwide each year die as a result of smoking [2].

Smoking is one of the most common forms of recreational drug use. Marijuana smoking is today by far the most popular form of smoking and is practiced by over several people in the majority of all human societies. Opium is one of the less common drugs for smoking. Some of the substances are classified as hard narcotics, like heroin, but the use of these is very limited as they are often not commercially available [2].

The history of smoking can be dated to be as early as 5000 BC, and has been recorded in many different cultures across the world. Early smoking evolved in association with religion ceremonies, as offerings to doilies, in cleansing rituals or to allow shamans and priests to alter their minds for purposes of divination or spiritual enlightenment. After the European exploration and conquest of the Americas, the practices of (mostly of cannabis). In Europe, it introduced a new type of social activity and a form of drug intake which previously had been known perception surrounding smoking has varied over time and from one place to another, holy and sinful, sophisticated and vulgar [2]. Today, medical studies have proven that smoking marijuana is among the leading causes of many diseases such as lung cancer, heart attacks, and erectile dysfunction and can also lead to birth defects. The inherent health hazards of smoking have caused many countries to launched anti- smoking campaigns every year in an attempt to curb marijuana smoking.

2.0 Theoretical background

Marijuana refers to the dried leaves, flowers, stems, and seeds from the *Cannabis sativa* or *Cannabis indica* plant. The plant contains the mind-altering chemical THC and other related compounds. People use marijuana by smoking, eating, drinking, or inhaling it. Tetrahydrocannabinol or THC is a chemical found in marijuana known to be the psychoactive compound that creates a euphoric feel and atmosphere. The amount of THC affects how strong one feels the effects of smoking marijuana or ingesting it in other ways, in other words the more the THC the more effect you feel. The amount of THC on a marijuana plant is affected by how it is treated during its growth period and the genetics of the seeds used. When smoked the THC is carried by the smoke and enters the user's lungs where it is then absorbed by the alveoli and transported to the brain by following the bloodstreams path, after it reaches the brain it then travels through the rest of your body. This method takes merely seconds for it to travel through. Another form is by ingesting marijuana, which once it enters the stomach the blood carries it throughout your body. Although this method takes about thirty minutes to feel, the effects are longer lasting than if smoked. Kevin Bonsor claims, "An intravenous (IV) dose of only one milligram can produce serious mental and psychological effects" [3]. Once smoked it goes straight to your brain and effects can be felt within seconds. Bonsor also adds, "The user's eyes may dilate, causing colors to appear more intense, and other senses may be enhanced" [3]. It is important to keep in mind that the effects of marijuana are influenced by dose, mode of ingestion, prior experience with marijuana (including use expectancies), and by the social context in which use occurs [4]. Cannabis use has been implicated in an increased risk of schizophrenia. Longitudinal studies have clearly demonstrated that marijuana use increases the likelihood that schizophrenia will develop in at-risk individuals, with more frequent use linked to increased risk of diagnosis [4]. Researchers have estimated that 14% of schizophrenia diagnoses could be prevented if marijuana use was similarly prevented [5]. Meta-analysis has confirmed links between depression and marijuana use, but the evidence is not as strong as that seen between marijuana use and psychosis. Meta-analysis has also concluded that a link between suicide and marijuana use exists, but causation remains unclear (as cited in Moore et al., 2007) [5]. Survey research has indicated that acute adverse psychiatric reactions, which are most common in new users, can include anxiety (with or without panic attacks) and psychotic symptoms [4]. In fact, in a community sample, 22% of users reported experiencing acute anxiety or panic attacks following use, and 19% reported memory loss or "blackouts" for a period while intoxicated (Thomas, 1996) [6]. Acute marijuana intoxication may also be associated with impaired judgment, leading to risky behaviors like unprotected sexual intercourse or driving while intoxicated [7]. Marijuana smoke and tobacco smoke contain many of the same carcinogens [8]. Regular, heavy smokers of marijuana report chronic cough, throat irritation, and other symptoms of chronic bronchitis [9]. There is very clear and consistent evidence that marijuana smokers are more likely to experience a broad range of respiratory symptoms, including wheezing, phlegm, and exercise-induced shortness of breath, even after accounting for contributions from nicotine. Damages to the respiratory system may occur after only a short period of heavy cannabis use [10]. Most researchers agree that chronic, heavy use of marijuana increases cancer risk [9].

3.0 Methodology

The methods adopted in this work are:

Firstly, A sample population of people treated at the Imo state teaching hospital from diseases emanating from marijuana smoking were collected Statistically analyzed and relevant coefficient were deployed for the

coding of the simulation model. Secondly, simulation software was developed using the indices collected from the statistical software to assess the impact of marijuana smoking in the next 50 years on smokers.

3.1 Presentation and Analysis of Data

This is the analysis of the data collected from Imo State University Teaching Hospital Orlu (IMSUTH). The death rates from the smoking of marijuana will be estimate through the development of regression model in which the descriptive statistics of each of the variables will be calculated. These are the mean, the standard deviation and the correlation between the various marijuana smokings.

3.0 Data Source

The source of the data for the work is Imo State University Teaching Hospital, IMUSTH, Orlu. Data were collected on the following variable over a period of 24 months, from the records of patients suffering from marijuana smoking –related killer diseases.

- Liver disease
- Lung disease
- Hepatitis
- Brain damage

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

4.1 Data Arrangement

TABLE 1: 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 2015	5	4	0	1	0	18
4	April	4	2	0	0	0	23
5	May	1	1	2	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	0	0	0	18
12	Dec.	2	0	3	0	0	19
13	Jan. 2016	0	0	2	0	0	24
14	Feb	4	4	0	0	0	10
15	March	10	0	0	0	6	17
16	April	1	0	4	0	0	16
17	May	1	1	1	0	0	19
18	June	3	1	0	1	1	19
19	July	5	4	0	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 IMSUTH

4.2 Descriptive Statistics

A Preliminary descriptive statistics were done on the data collected for period of 24 months. The mean number of deaths, their correlation as well as standard deviations was identified. The four identified diseases caused by marijuana smoking were analyzed separately. Below are the computational formula used:

$$Y = \alpha\beta x_1 + \beta x_2 + \dots + \beta x_n$$

$$\text{Mean (X)} = \frac{\sum_{i=1}^n x_i^2}{n} \dots \dots \dots (1.1)$$

X-number of deaths in each month

$$\text{Standard deviation (SD)} = \sqrt{\frac{\sum_{i=1}^n x_i^2}{n} - \left(\frac{\sum_{i=1}^n x_i}{n}\right)^2} \dots \dots \dots (1.2)$$

And

$$\text{Correlation (r)} = \frac{n\sum xy - (\sum x)(\sum y)}{\sqrt{\left\{n\sum xy^2 - (\sum x)^2\right\} \left\{n\sum y^2 - (\sum y)^2\right\}}} \dots \dots \dots (1.3)$$

Where

X = no of deaths from one disease

Y = no of deaths from another disease

Using the software “statistic packages for scientific studies(SPSS)”, the following results were obtained.

Mean and Standard Deviation

Mean	Standard Deviation
Liver = x_1	= 1.6818 $SD_1 = 1.78316$
Lung disease = 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 $SD = 5.90179$

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, lung disease, hepatitis and Brain damage, out of the total number of patients. The proposed non-intercept model is of the form:

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

Where

- Y = Total number of patients
- X₁ = number of deaths from liver disease
- X₂ = number of deaths from lung disease
- X₃ = number of death from hepatitis
- X₄ = number of death from Brain
- E = error term.

B₁ β₂ β₃ and β₄ are the model parameters that will be estimated using the following formulas:

$$Y = \beta X + e, y = \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4 + e$$

Putting this in matrix form we have

Where

$$Y = X\beta + e$$

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

$$e = \begin{Bmatrix} e_1 \\ e_2 \\ \vdots \\ e_n \end{Bmatrix}$$

Using method of least squares, we can derive the estimate of β₁ as follows:

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

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

$$\begin{aligned}
 SSe - e^T e &= (Y - X\beta)^T (Y - X\beta) \\
 &= Y^T Y - Y^T X\beta - \beta^T X^T Y + \beta^T X^T X\beta
 \end{aligned}$$

$$SSe = y^T y - 2\beta^T X^T y + \beta^T X^T X\beta \dots\dots\dots (1.6)$$

Differentiating (4.6) wrt β and equating to zero, we obtain

$$\frac{dSSe}{d\beta} = 2X^T y - 2X^T X\beta = 0$$

$$X^T X\beta = X^T Y$$

$$\beta = (X^T X)^{-1} (X^T Y) \dots\dots\dots (1.7)$$

The fitted model will be tested for its adequacy using an ANOVA table.

Table 2: Anova Table to Test Model Adequacy

Source of variation	Df	Sum of Square	Means 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.

$$SSR = \sum_{i=1}^N (\hat{y}_i - y_i)^2 \dots\dots\dots (1.8a)$$

$$SSe = \sum_{i=1}^N (y_i - \hat{y}_i)^2 \dots\dots\dots (1.8b)$$

$$SST = \sum_{i=1}^N (y_i - \bar{y})^2 \dots\dots\dots (1.8c)$$

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

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

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

$$t = \frac{\beta_i}{\text{Se}(\beta_i)} = \frac{\beta_i}{\delta^2 C_{ii}} \dots\dots\dots (1.9)$$

$$\text{Se}(\beta_i) = \sqrt{\delta^2 C_{ii}}$$

Where

β_i = Coefficient of intercept

δ^2 = MSe

C_{ii} = Diagonal element of $(X^T X)^{-1}$

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

$$Y = 3.095 X_1 + 4.782 X_2 + 8.329 X_3 + 1.9.11 X_4 \dots \dots \dots (1.10)$$

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

Table 3: Anova table presented from SPSS

Model	Sum of Square	Df	Mean Sum of	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_0 and accept H_a saying that at least one of the parameters is significantly different from zero. To know which of these B_1 is significantly different from zero, we have the following t-test results.

Table 4: 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 5: Correlation Table

	R	P-value	Remark
Liver Vs Lung disease	0.257	0.124	Not significant
Liver Vs. Hepatitis	0.470	0.014	Significant
Liver Vs. Brain damage	0.125	0.290	Not Significant
Lung disease 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 person die monthly as a result of tobacco smoking –related liver disease, while an average of 0.8636 people dies monthly from lung disease 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 0.090 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 tobacco smoking 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. 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 lung disease. 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 show 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. Lung disease 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 I 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 the number of death from liver, lung disease, Hepatitis, and Brain damage. This 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 $Y = 3.095X_1 + 4.782X_2 + 8.329X_3 + 1.911 X_4$ shows that for every unit death as a result of liver disease, about 3.095 persons are patients suffering from any of the four disease. Also for every unit death as a result of lung disease, 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 lung disease 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.

4.0 Summary

A summary of the negative impact of marijuana smoking on human smokers has been carried out for about 30 years. It has been found that smoking of marijuana is the main source of liver, heart, kidney and lungs related diseases including hypertension and death. There is need to establish some level of control over marijuana smoking to prevent untimely deaths of the larger population of citizens.

5.1 Conclusion

Constant smoking of marijuana can result to untimely death to a community and a whole nation. If this dirty habit of smoking is not controlled, there is a tendency of decimation of the Nigerian population and productivity impotency of large proportion of population. To avert the possibility of human existence, government needs to take decisive measures.

5.2 Recommendation

I recommend the following:

- Knowledge of marijuana smoking should part of Secondary School Curriculum in Nigeria
- Seminars and workshops should be organized at federal, state and local levels by the ministry of health, national orientation Agency to create awareness of the negative impacts of marijuana smoking.
- There is need for the government to create job opportunities for unemployed youths who indulge in marijuana smoking as a source of reduction of depression or teased

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