A Computational Analysis of the Negative Impact of Cigarette Smoking on Human Population In Imo State

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Abstract

Smoking is a practice in which a substance most commonly called Tobacco or Cannabis is burnt and the smoke tasted or inhaled. Recognition of the consequences of cigarette smoking and abuse on physical and mental health as well as socio-occupational life are necessary steps for initiating appropriate action to reduce the harm or dangers resulting 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 cigarette smoking. 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 smoking using Digital Display Nicotine Tester (DDNT). Secondly, sample populations of people treated at the Imo State University Teaching Hospital from diseases emanating from smoking were collected, statistically analyzed using Statistical Packages for Social Sciences (SPSS).Relevant coefficients were extracted and 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 smoking on the population in the next 50 years. This is to assist policy formlators and decision makers on what public policy should be in place to stem possible health catastrophe that may occur as a result of uncontrolled consumption. The software simulation follows a stochastic model.

Introduction

The issue of smoking and associated health risks in human beings have become a crucial matter for discussion. Most people today engage in one type of Tobacco smoking or the other without knowing its negative impact on human beings. The inhalation of products of tobacco may cause serious respiratory complications.

According to WHO [12] as many as one-third of patients admitted to burn treatment unit have pulmonary injury from smoke inhalation. Morbidity and deaths due to smoke inhalation exceed those attributed to the burns themselves. This same report also shows that the death rate of patients with both severe body burns and smoke inhalation exceeds 50%.

In 1612, six years after the settlement of Jamestown, John Rolfe was credited as the first settler to successfully raise tobacco as a cash crop. The demand quickly grew as tobacco, referred to as "golden weed", reviving the Virginia join stock company from its failed gold expeditions [7]. In order to meet .demands from the old world, tobacco was grown in succession, quickly depleting the land. This became a motivator to settle west into the unknown continent, and likewise an expansion of tobacco production [3]. Indentured servitude became the primary labor force up until Bacon's Rebellion, from which the focus turned to slavery. This trend abated following the American Revolution as slavery became regarded as unprofitable. However the

practice was revived in 1794 with the invention of the cotton gin [7].

A Frenchman named Jean Nicot (from whose name the word Nicotine was derived) introduced tobacco to France in 1560. From France tobacco spread to England. The first report of a smoking Englishman was a sailor in Bristol in 1556, seen "emitting smoke from his nostrils" [11]. Like tea, coffee and opium, tobacco was just one of many intoxicants that were originally used as a form of medicine [5]. Tobacco was introduced around 1600 by French merchants in what today is modern-day Gambia and Senegal. At the same time caravans from Morocco brought tobacco to the areas around Timbuktu and the Portuguese brought the commodity (and the plant) to southern Africa, establishing the popularity of tobacco throughout all of Africa by the 1650s.

Soon after its introduction to the Old World, tobacco came under frequent criticism from state and religious leaders. Murad IV, *sultan of the Ottoman Empire 1623-40* was among the first to attempt a smoking ban by claiming it was a threat to public moral and health [5]. The Chinese emperor Chongzhen issued an edict banning smoking two years before his death and the overthrow of the Ming dynasty. Later, the Manchu of the Qing dynasty, who were originally a tribe of nomadic horse warriors, would proclaim smoking "a more heinous crime than that even of neglecting archery". In Edo period Japan, some of the earliest tobacco plantations were scorned by the shogunate as being a threat to the military economy by letting valuable farmland go to waste for the use of a recreational drug instead of being used to plant food crops [8].

The most common method of smoking today is through cigarettes, primarily industrially manufactured but also hand-rolled from loose tobacco and rolling paper. Other smoking tools include pipes, cigars, bidis, hookahs 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**.[16]**

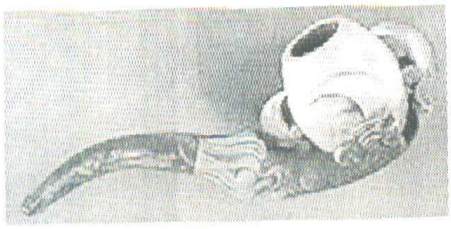

Fig 1: *An elaborately decorated pipe. Source:* ***Proctor R. N., (2000).***

Smoking is one of the most common forms of recreational drug use. Tobacco smoking is today by far the most popular form of smoking and is practiced by over one billion people in the majority of all human societies. Less common drugs for smoking include cannabis and opium [7].

The history of smoking can be dated to as early as 5000 BC, and has been recorded in many different cultures across the world. Early smoking evolved in association with religious ceremonies; as offerings to deities, 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 Americans, the practice of smoking tobacco quickly spread to the rest of the world. In regions like India and sub-Saharan Africa, it merged with existing practices of smoking (mostly of cannabis) [6]. In Europe, it introduced a new type of social activity and a form of drug intake which previously had been unknown. Perception surrounding smoking has varied over time and from one place to another; holy and sinful, sophisticated and vulgar, a panacea and deadly health hazard. Only relatively recently, and primarily in industrialized Western countries-, has smoking come to be viewed in a decidedly negative light. Today medical studies have proven that smoking tobacco 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 institute high taxes on tobacco products and anti-smoking

campaigns are launched every year in an attempt to curb tobacco smoking [6].

Many ancient civilizations such as the Babylonians, Indians and Chinese burnt incense as a part of religious rituals, as did the Israelites and the later Catholic and Orthodox Christian churches. Smoking in the Americas probably had its origins in the incense-burning ceremonies of shamans but was later adopted for pleasure or as a social tool. The smoking of tobacco and various other hallucinogenic drugs was used to achieve trances and to come into contact with the spirit world.

Substances such as Cannabis, clarified butter (ghee), fish offal, dried snake skins and various pastes molded around incense slicks dates back at least 2000 years. Fumigation *(dhupa)* and fire offerings *(homa)* are prescribed in the Ayurveda for medical purposes and have been practiced for at least 3,000 years while smoking, *dhumrapana* (literally "drinking smoke"), has been practiced for at least 2,000 years. Before modern times, these substances have been consumed through pipes, with stems of various lengths or chillums [3].

Cannabis smoking was common in the Middle East before the arrival of tobacco, and was early on a common social activity that centered on the type of water pipe called a hookah. Smoking, especially after the introduction of tobacco, was an essential component of Muslim society and culture and became integrated with important traditions such as weddings, funerals and was expressed in architecture, clothing, literature and poetry [5].

Cannabis smoking was introduced to Sub-Saharan Africa through Ethiopia and the east African coast by either Indian or Arab traders in the 13th century or earlier or spread on the same trade routes as those that carried coffee, which originated in the highlands of Ethiopia [13]. It was smoked in calabash water pipes with terra cotta smoking bowls, apparently an Ethiopian invention which was later conveyed to eastern, southern and central Africa.

At the time of the arrivals of Reports from the first European explorers and conquistadors to reach the Americas tell of rituals where native priests smoked themselves into such high degrees of intoxication that it is unlikely that the rituals were limited to just tobacco [17]. Religious leaders have often been prominent among those who considered smoking immoral or outright blasphemous. In 1634 the Patriarch of Moscow forbade the sale of tobacco and sentenced men and women who flaunted the ban to have their nostrils slit and their backs whipped until skin came off their backs. The Western church leader Urban VII likewise condemned smoking in a papal bull of 1590. Despite many concerted efforts, restrictions and bans were almost universally ignored. When James I of England, a staunch anti-smoker and the author of *A Counterblast to Tobacco,* tried to curb the new trend by enforcing a whopping 4000% tax increase on tobacco in 1604, it proved a failure, as London had some 7.000 tobacco sellers by the early 17th century. Later, scrupulous rulers would realize the futility of smoking bans and instead turned tobacco trade and cultivation into lucrative government monopolies [10].

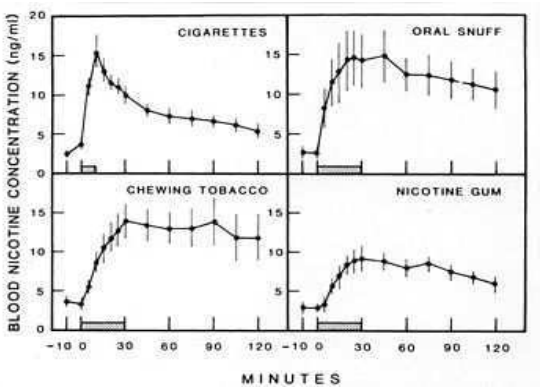

Fig.2 *A graph that shows the efficiency of smoking as a way to absorb any different one. Source:* ***Proctor R. N., (2000)***

TABLE 1.1:

Cardiovascular Disease
Coronary artery disease
Peripheral vascular disease
Aortic aneurysm
Stroke (at younger ages)
Cancer
Lung
Larynx, oral cavity, esophagus
Bladder, kidney
Pancreas, stomach
Lung Disorders
Cancer (as noted above)
Chronic bronchitis with airflow obstruction
Emphysema
Complications of Pregnancy
Infants—small for gestations age, higher perinatal mortality
Maternal complications— placenta previa, abruptio placenta
Gastrointestinal Complications
Peptic ulcer
Esophageal reflux

Increased Risks for Cigarette Smokers

Source: ***Burns, (1985.)***

Methodology

Three types of methods are adopted in this work:

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

Secondly, a sample population of people treated at the Imo State University Teaching Hospital from diseases emanating from smoking were collected, statistically analyzed and relevant coefficient were deployed for the coding of the simulation model. The third methodology which is the development of a simulation model to predict the negative impact of Cigarette Smoking will be discussed in a follow-up paper to be published in the next edition of this journal.

Steps Involved In Using Digital Display Nicotine Tester

To use the Digital Display Nicotine test the following steps are involved:

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 Nicotine Level (BNL) test.

Step 2: Test

1: Near and blow into the breath inhaler for seconds
2: Read the test result on the LCD (the level of Nicotine in the consumer's blood in mg/L)
3: The buzzer continuously sounds alarm quickly if the Nicotine concentration or level is up to the level:

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

1: Press the power button to turn off the tester
2: 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.

Step 3: Power Off

Recording Format For Experimental Result

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

Table: 2 Comparison of Experimental Result With Actual BNC Values

The level of Nicotine display in mg/l mg/L	The level of Nicotine display in BNC
0.05	0.01
0.10	1.02
0.20	0.04
0.25	0.05S
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: IMSUTH, ORLU

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

The second methodology is guided by the Structured System Analysis and Design Methodology (SSADM). This enables the researcher to study the existing methodology of Nicotine testing for purposes of identifying gaps and weaknesses in order to deploy the improved methodology using the DDNT Clinical testing tool. The SSADM study enables the acquisition of data that will enhance the development of a computer simulated solution of cigarette smoking addiction model.

To change the current system used in determining the Blood Nicotine Concentration (BNC) using blood serum test, to a better method using a device called Digital Display Nicotine Tester (Fig. 2). It is pertinent to carry out an in-depth system's investigation and analysis of the old system. A High Level Model was developed from the study of the present system to solve the problems identified at the analysis stage.

The Structured Systems Analysis And Design Methodology (SSADN) Steps

The methodology adopted here in the second phase of the study is the Structured Systems Analysis and Design Methodology (SSADM). The SSADM is the standard information system development method. It takes a top-down approach to system development, in which a high level picture of system requirements is built and then gradually refined into a detailed and rigorous system design. Its steps include:

i. Problem identification
ii. Feasibility study
iii. Analysis
iv. Design
v. Implementation
vi. Post implementation maintenance

Discussion of Findings

This section deals with the analysis of the collected (secondary) data from the Imo State University Teaching Hospital, IMUSTH, Orlu. Regression model was developed to estimate the death rates resulting from the tobacco smoking in the society. The descriptive statistics of each of the variables was calculated. These include: the mean, the standard deviation and the correlation between the various consumption of Tobacco impact

Data Source

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

- Liver disease
- Lung disease
- 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

Data Arrangement
The Data Collected Are Rearranged In Table 3

Table 3: 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 2007	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. 2008	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: IMSUTH Orlu

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:

$$Y = a\beta x_1\ 4Bx_2 + \ldots\ \beta x_n$$

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

X-number of deaths in each month

Standard deviation (SD) =

And

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

n n

Correlation (r) =

$$\frac{n\sum x_i y_i - \sum x_i \sum y_i}{\sqrt{n\sum x_i^2 - (\sum x_i)^2}\ \sqrt{n\sum y_i^2 - (\sum y_i)^2}}. \qquad (1.3)$$

Where

X = no of deaths from one disease

Y = no of deaths from another disease

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

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

Regression Analysis

A regression model proposed 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:

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

Where

Y	=	Total number of patients
X_1	=	number of expected deaths from liver disease
X_2	=	number of expected deaths from lung disease
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:

$$Y = \beta X + e,\ y = \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \beta_4 x_4 + e \quad (1.5)$$

Putting this in matrix form we have

Where $Y = X\beta + e$

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

$$e = \begin{pmatrix} e_i \\ e_2 \\ \vdots \\ e_n \end{pmatrix} \quad (1.6)$$

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

$$e = Y - \beta X \quad (1.7)$$

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

$$SSe - e^1 e = (Y - X\beta)^1 (Y - X\beta)$$
$$= Y^1 Y - Y^1 X\beta - \beta^1 X^1 Y - \beta^1 X^1 X\beta$$
$$SSe = y^1 y - 2\beta^1 x^1 y + \beta^1 x^1 x\beta \quad (1.8)$$

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

$\frac{dSSe}{d\beta} = 2x\,{}^{1}y - 2\beta^{1}x^{1}x = 0$

$2\beta^{1} = 2\,X\,{}^{1}Y$

$$\beta = X^{1}Y = \frac{(X^{1}X)^{-1}(X^{1}Y)}{X^{1}X} \quad (1.9)$$

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 4: 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

$$SSR = \hat{\beta}^{1}X^{1}Y - NY^{2}. \quad (1.10a)$$

$$SSe = Y^{1}Y - \hat{\beta}X^{1}Y \quad (1.10b)$$

$$SST = Y^{1}Y - NY^{2} \quad (1.10c)$$

$$MSR = SSR/K \quad (1.10d)$$

$$MSe = SSe/(N-K-1) \quad (1.10e)$$

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

$$t = \frac{\beta_i}{\sqrt{Se(\beta)}\sqrt{\delta^2 C_{ii}}} = \beta i \quad (4.9)$$

Where

βi = Coefficient of intercept

δ^2 = MSe
C_{ii} = Diagonal element of $(X^{1}X)^{1}$

$$Y = 3.095X_1 + 4.782X_2 + 8.329X_3 + 1.911X_4 \quad (4.10)$$

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

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

Table 5: 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 Ho and accept Ha 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:

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Table 6: 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

CORRELATION

Table 7: Correlation Table

	R^2	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

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 person die 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 0.0909 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 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 [14].

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 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. 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 is a strong positive correlation between Hepatitis and Brain damage.

Coefficient Of Multiple Determination (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, lung diseases, 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.

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 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.

References

[1] **Doll R. and Hilly A., (1950).** *Smoking and carcinoma of the lung. Preliminary report*. British Medical Journal 2 (4682)

[2] **Doll R. and Hilly A., (1954).** *The mortality of doctors in relation to their smoking habits: A preliminary report.* British Medical Journal

[3] **Gay and Freud, (1988).** *A Life for Our Time. New York: W:W. Norton and Company.* Pp. 650-651.

[4] **Geyelin M., (1998).** *Forty-six state agree to accept and 206 billion tobacco settlement.* Wall Street Journal.

[5] **Harris J. R., (1998).** *The nurture assumption: Why children turn out the way they do*. Free Press New York. High Versus Low Fidelity Simulations: *Does the Type of Format Affect Candidates Performance or Perceptions?*

[6] **Hilton M., (2000).** "Smoking in British popular culture, 1800-2000: perfect pleasures". Manchester University Press, p. 229-241.

[7] **Iverson L, (2004).** *why do We smoke?: The physiology of smoking.* in smoke. p 318-320.

[8] **Pollard T, (2006).** *The pleasures and perils of smoking in Early Modern England.* In smoke, p. 38.

[9] **Rock VJ, Malarcher A., Kahende JW, Asman K., Husten C., Caraballo R.**, **et al (2007).** *Cigarette Smoking Among Adults United State, 2006.* United States Centers for Diseases Control and Prevention. http//www.cdc.gov/mmwr/preview/mmwrhtml/htm.

[9] **Timon Screech, (2004).** *Tobacco in Edo Period Japan in smoke.*

[10] **Trugar J, (1994).** *The people's Chronology.*

[11] **West R. and Shiffman S., (2007).** *Fast fact: smoking cessation*.

[12] **World Health Organization Regional Office for the Western Pacific (2007).** *WHO/WPRO-Tobacco Fact sheet.* Retrieved 2009-01-01.

[13] **Yule, G.U and Kendall, M.G. (1950),** "An introduction to the Theory of Statistics", 14th Edition (5th Impression 1968). Charles Griffin & Co. pp258-270