Expected Time Using Alpha–Poisson Distribution Under Stochastic Model

S. Jothimanickam & P. Pandiyan

ABSTRACT - Today, cigarette smoking causes 85 percent of lung cancer deaths. Cigarette smoking is causally connected to cancers of the cervix, bladder, oesophagus, lung, mouth, kidney, abdomen and pancreas. During this study, a non-linear system of differential equations is used to model the dynamics of a population, which incorporates smokers. The parameters of the model are obtained from data revealed by cancer institute’s, health and government organizations. The typical variety of people United Nations agency become smokers associated with the reduction of this average by an education program are determined.

Index Terms: Lung cancer, Smoking, Tumour growth, Expected lifetime, statistical modelling.

1. INTRODUCTION

Cancers remaining that most important killer among all cancers within the world. It kills additional individuals of each gender than the cancers of breast, colon and prostate combined, and more women than breast cancer. An amazing majority of cases is expounded to exposure to Polycyclic Aromatic Hydrocarbons (PAH), like benzo pyrene, within the tobacco smoke; however, genetic predisposition also plays a major role. A comprehensive random model of lung cancer ought to involve genetic and behavioral determinants of susceptibility, the progression of the disease from precursor lesions through early-localized tumors to disseminated disease, detection by varied modalities, and medical intervention. The model ought to be ready to predict mortality reduction caused by early detection programs, below completely different situations, in presence of competitive death causes. It will be necessary to utilize the genetic indicators of condition to lung cancer to define the high-risk behavior population (smokers). Cigarette smoking is that the single greatest preventable risk issue for mortality and morbidity. According to a 2004 surgeon General report, cigarette smoking is causally linked to cancers of the bladder, cervix, esophagus, kidney, larynx, lung, mouth, pancreas, and stomach. Furthermore, there exists a causative relationship between smoking and coronary heart disease, cerebrovascular disease, coronary artery disease, various respiratory diseases and a number of other reproductive maladies. 440,000 deaths are attributed to smoking within the United States annually. Health problem from smoking is calculable to feature $157 billion annually to national health expenditures. In short, a 2004 united states Surgeon General Report on smoking concludes by stating: Smoking harms nearly each organ of the body, inflicting several diseases and reducing the health of smokers generally. Smoking is that the leading preventative cause of death within the United States. In the United States alone, 44.5 million adults, or 20.9% of the adult population, were smokers in 2004. The foremost tragic consequence is that the 440,000 annual premature deaths because of smoking. Different consequences include, however do not seem to be restricted to, $75.5 billion smoking related medical expenditures and $92 billion in mortality related annual productivity losses. Worldwide, smoking related mortality is about to rise from 4.9 million annually to ten million by 2030. Smoking may be a major explanation for an outsized variety of diseases, together with cancers of the respiratory organ, larynx, mouth, pharynx, esophagus, pancreas, and bladder as coronary cardiovascular disease, stroke and chronic obstructive pulmonary disease (COPD). Although the prevalence of adult smoking within the United States born from 42.4% in 1965 to 25.5% in 1990, progress has been low since the 1990s (26.5% in 1992, 24.7% in 1995, and 23.3% in 2000). This is often part due to high rates of relapse following quit tries among smokers. This is often revealed by the actual fact that the prevalence of bring to an end within the united states accumulated from 24.3% in 1965 to 49.6% in 1993 and then flattened to 48.8% in 2000. Surveys show that top smoking prevalence is a minimum of partly because of high rates of relapse among smokers World Health Organization attempt quitting. A major problem once learning addiction behavior is that participants generally build many quit attempts before they with success quit. Thus, for economic development, targeting, and analysis of interventions, it is necessary to differentiate transient cessation (temporarily smoking-free however relapse later) from permanent cessation (lifelong smoking-free) and establish the chance factors related to permanent cessation. Our objectives are to identify and quantify baseline factors related to success of permanent smoking cessation and describe the complete stochastic nature of the smoking addiction pattern. With in the remainder of this section, we have a tendency to describe the dataset, covariates, and modeling strategy to attain these objectives. There has been a recent progress in lung cancer detection techniques. Computed tomography (CT) allows the visualization of terriblythin nodules within the lungs and thus it is the potential to detect malignant tumors after they still are in associate early stage. It detects in the main peripheral tumors, an oversized proportion of that is

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adenocarcinomas that recently became the foremost common style of lung cancer, and Broncho alveolar carcinomas. The results of preliminary studies within the United States, Japan associated Western Europe is printed and that they purpose at a hyperbolic detection rate of potential early malignancies. The chance that these can transform progressing respiratory organ cancers is not far-famed, notably that of a number of the lesions which regularly lack well-defined nodule components. Another space of progress considerations the genetic factors predisposing people to developing lung cancer. There exists loads of information, regarding largely the families of lung cancer patients. Molecular epidemiological studies have shown a poor DNA repair capability, as measured by assays that provide its overall estimate, to be a risk factor for developing lung cancer. Genetic factors concerned within the metabolism of carcinogens have conjointly been suggested to contribute to lung cancer susceptibility. These associations will facilitate to identify people (smokers) at a high risk to develop lung cancer. The purpose of this paper is to spot the issues associated with early detection and treatment of lung cancer and to explain a random model that makes it potential to deal with these problems, i.e., to reconcile the results of screening trials with alternative existing statistics of lung cancer. The model is given on the background of previous works on the subject. Arguably, its additional complete and higher tested compared with these previous attempts. In its gift version, the model includes neither express mechanisms of growth growth, nor personal genetic susceptibleness.

Poisson distribution was first published in 1837. Pillai (1990) and Anil (2001) introduced the alpha-Poisson distribution as a generalization of Poisson process. Esary, Marshall and Prochan (1973)

### 2. ASSUMPTIONS OF THE MODEL

These assumptions square measure somewhat artificial however square measure created as a result of the shortage of elaborate real-world data on one hand and so as for example the proceedings on the opposite hand.

- Smoking contacts are the only source of tumor growth for lungs cancer.
- The threshold of anyone could be a variable quantity. If the overall harm crosses an intensity, Y that itself could be a variable quantity, the seroconversion happens associate degree an individual is recognized as an infected.
- The inter-arrival times between serial contacts, the sequence of injury and the threshold area unit reciprocally freelance.

### 3. NOTATIONS

**X_i:** A discrete random variable denoting the amount of contribution to the threshold due to the smoking conduct in the i^{th} contact, in other words the damage caused to the tumor growth in the i^{th} contact, with p.d.f \( f_{X_i} \) and c.d.f \( F_{X_i} \). 
**Y:** A discrete random variable denoting the threshold which follows alpha – Poisson distribution.

**U_i:** A random variable denoting the inter-arrival times between contact with c.d.d. \( F_{U_i}, i = 1, 2, ..., k \).

**g(\cdot):** The probability density functions of \( X_i \).

**g_{k}(\cdot):** The k-fold convolution of \( g(\cdot) \) i.e., pdf of \( \sum_{i=1}^{k} \), \( \sum_{i=1}^{k} X_i \)

**f_{C}(\cdot):** p.d.f of random variable denoting between successive contacts with the corresponding c.d.f \( F_{C}(\cdot) \).

**V_{k}(t):** Probability of exactly k component

**S(t):** Survival function. i.e \( P[T > t]; L(t) = 1 - S(t) \)

## 4. MODEL DESCRIPTION

The Alpha-Poisson distribution with parameter a and \( \alpha \) is given by

The probability generating function

\[
P(x) = \sum_{n=0}^{\infty} (-1)^n \frac{(k + n)^{\alpha+1}}{\Gamma(\alpha+n+1)} \frac{\alpha^\alpha}{\alpha!} \]

When the shape parameter \( \alpha = 1 \); it represents the Alpha-Poisson distribution.

The survival function becomes

\[
S(t) = P(X_i < Y) = \int_0^t g_k(x)H(x) \, dx
\]

The survival function which is the probability that an individual survives for a time \( t \)

\[
S(t) = P(T > t) = \text{Probability that the system survives beyond } t = \sum_{k=0}^{\infty} P(\text{there are exactly } k \text{ instants of exists in } (0, t]) \]

\[
S(t) = \sum_{k=0}^{\infty} V_k(t) P(X_i < Y)
\]

\[
S(t) = \sum_{k=0}^{\infty} [F_k(t) - F_{k+1}(t)] [g^*(a)]^k
\]

Using convolution theorem for Laplace transforms, \( F_0(t) = 1 \) and on simplification, it can be shown that Lifetime \( L(T) = 1 - S(t) \)

Taking Laplace transform of \( L(T) \), we get,

\[
1 - \sum_{k=0}^{\infty} [F_k(t) - F_{k+1}(t)] [g^*(a)]^k
\]

\[
= 1 - \left\{ \sum_{k=0}^{\infty} [F_k(t) - F_{k+1}(t)] [g^*(a)]^k \right\}
\]

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\[ g^*(a) = \frac{1}{1 + a^s} \]

\[ E(T) = \frac{1 + a}{c (a)} + \frac{1 + (ar)}{c (ar)} \]

Table: 1

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<th>r=(FVC)</th>
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Figure - 1: The chart for FEV1, FVC infected person's stage wise

Figure - 2: The Chart for infected person’s Expected time

5. RESULTS AND DISCUSSION

In this study showed that the person increase the tumor growth for lung cancer contact the expected lifetime is decreased. This study infers that the person infected with tumor growth for lung cancer gross the threshold level a lot of quickly. The tumor cells are growth once someone is infected and he/she is probably going to be affected once they are infected with the lungs. Life expectancy is ‘the average range of years a personal is anticipated to measure, with neoplasm cell infection. We advise a technique for modeling participant-level random addiction behavior. We have a tendency to model one by one transient and permanent surcease and permit for risk factors to possess completely different impacts on these two cessation states. Moreover, we have a tendency to introduce a statistically place able however unobserved cure method in addition to smoking and transient quitting processes and enclosed liver disease of the liver, chronic joint disease, autoimmune disease, alternative inflammatory disease, chronic bronchitis, myocardial infarction, coronary heart disease, and heart failure, diabetes, debilitating back pain, knee pain, joint ache, muscle ache, hip pain, leg cramps, and headache, and enclosed anxiety, depression, poor memory, issue concentrating, fatigue, poor appetency, and sleep disorder. Alcohol use was coded as mean grams per day as each a continuous and a categorical variable, as was body mass index (BMI). Demographic variables include age at enrollment (continuous), marital status (categorical), education (categorical), employment status (categorical), and physical activity (categorical). It has been widely distinguished that the quantity of people infected patients with advanced or pathological process FEV1, FVC -negative cells are growths thus damaged in others smart cells mouth cancer has been increasing in recent years particularly in developing countries. The threshold level of FEV1, - negative infected
cells is been calculated through statistical model of the infected person. Several standard of medical care are supported the demonstrated effects of varied treatment ways or method. When the FVC negative infected cells are affected in human body, shock with very different infected variable is that the one to appear. When the immune system doesn’t accumulated the increase in shock that is that the inter-arrival time, the expected life time of the human system can reach the edge. The total cumulative damage found with shock model approach exploitation renewal method. The expected lifetime is has been derived through distribution. The first data within the model derived for the expected time.

6. CONCLUSION
In conclude that the person infected with mouth cancer FEV1, FVC cell infected is more quickly to cross the threshold level. Once the person is infected, the FEV1, FVC Cells growth gets damaged both men and women are likely to affect, when infected with human tumor cells. The time interval is the drinking of the infected person. The expected lifetime decreases quickly to the threshold level. The interval period for the infection depends on the amount of smoking contact of the infected person. The model shows that the person is infected the immune system starts that is observed in the above table and figures. We observe that once the person gets affected by the cancer, good cells growths in tumor and FEV1, FVC cells are damaged mouth cancer his/her system capability gets diminished. By correct medical doctor recommendation and thru regular treatment his/her lifetime will be extended.

7. REFERENCES