STOCHASTIC MODEL FOR EXPECTED TIME USING GENERALIZED EXPONENTIAL DISTRIBUTION

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Abstract - ***Alcohol intake is a modifiable lifestyle factor that may affect prostate cancer risk. Alcohol alters the hormonal milieu and contains chemical substances such as flavonoids (red wine), which may alter tumor cell growth.. Alcohol is causally connected to cancers of the esophagus, kidney, larynx, lung, mouth etc. During this study, a non-linear system of differential equations is used to model the dynamics of a population, which incorporates alcoholic. 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 alcoholic associated with the reduction of this average by an education program are determined.***

Keyword: ***Prostate cancer, alcohol, Tumor growth, Expected lifetime, statistical modeling.***

I. INTRODUCTION

Prostate cancer is the most commonly diagnosed malignancy and the second leading cause of cancer deaths after lung cancer in United States men. Although age, ethnic origin and a positive family history of prostate cancer have been established as important risk factors for prostate cancer, its etiology is largely unknown. Migration studies have shown that prostate cancer incidence rates in immigrants moving from lower to higher incidence areas tend to shift toward the rates of the higher incidence country. The shift may be partly due to differences in screening and detection methods in different countries, but it also suggests a prominent role for environmental factors in the etiology of prostate cancer. The worldwide PCa burden is expected to grow to 1.7 million new cases and 499 000 new deaths by 2030 simply due to the growth and aging of the global population.

Environmental factors suspected to be associated with the risk of prostate cancer include diet (intake of fat, vegetables and fruits, dairy products and certain micronutrients and vitamins), occupation, smoking, alcohol consumption, sexual and physical activity, hormonal levels (androgens and estrogens), and body size. These factors are thought to influence the multi-step process of tumor promotion and progression either by acting directly in a causative pathway or indirectly, by acting on genes mediating disease susceptibility. Suggested indirect mechanisms are regulation of hormone level or regulation of metabolism of carcinogenic substances. Considering the slow growing character of most prostate cancers, environmental factors could presumably affect the course of the disease over a long time and in different phases of the disease process.

One environmental factor of interest is alcohol use, because especially heavy use clearly increases the risk of cancers of the oral cavity, larynx, pharynx and possibly other cancers. Inconsistent results have been reported, however, with regard to consumption of various levels and types of alcohol and the subsequent risk of prostate cancer.

Consumption of wine, especially red wine, has recently become a topic of interest due to its relatively high concentration of polyphenols (mostly flavonoids and resveratrol). Polyphenolic compounds have antioxidant activities and are therefore thought to function as anti-carcinogens. For some polyphenols, an anti-androgenic function has also been suggested. Further, basic research has shown that polyphenols inhibit tumor growth in vitro in both the androgen dependent LNCaP prostate cancer cell line and the androgen independent DU145 and PC3 lines.

Considering the widespread use of alcohol, further research focusing on the role of alcohol and red wine in particular in the etiology of prostate cancer is needed. If alcohol consumption is related to the risk of prostate cancer, this may have important public health implications. 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),P. Pandiyan (2018) discussed that any component or device when exposed to shocks which cause damage to the device or system is likely to fail when the total accumulated damage exceeds a level called the threshold.

II. ASSUMPTIONS OF THE MODEL

These assumptions are somewhat artificial, but are made because of the lack of detailed real-world information on one hand and in order to illustrate the proceedings on the other hand.

- Alcoholcontacts are the only source of tumor growth for prostate cancer.
- The threshold of any individual is a random variable. If the total damage crosses a threshold level Y which itself is a random variable, the seroconversion occurs and a person is recognized as an infected.
- The inter-arrival times between alcohol and cancer, the sequence of damage and the threshold are mutually independent.

III. NOTATIONS

X_i: a discrete random variable denoting the amount of contribution to the threshold due to thealcohol conduct in the i^{th} contact, in other words the damage caused to the tumor growth in the i^{th} contact, with p.d.fg(.) and c.d.f G(.).

Y: a discrete random variable denoting the threshold which follows three-parameter generalized Exponential distribution.

U_i : a random variable denoting the inter-arrival times between contact with c.d.f. $F_i(.), i = 1,2, \ldots . k$.

$g(.)$: The probability density functions of X_i.

$g*(.)$: Laplace transform of $g(.)$

$g_k(.)$: The k- fold convolution of $g(.)$i.e,pdf of $\sum_{j=1}^{k} X_i$

$f(.)$:p.d.f. of random variable denoting between alcohol and cancer with the corresponding c.d.f. F(.).

$F_k(.)$: k-fold convolution of F(.),

$V_k(t)$: Probability of exactly k component

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

IV. MODELDESCRIPTION

The three-parameter generalized Exponential distribution has the following cumulative distribution function (CDF) is

$$F(x; \alpha, \lambda, \theta) = \left[1 - e^{-\lambda(x-\theta)}\right]^{\alpha}; \qquad x > \theta, \quad \alpha, \lambda > 0$$

and the corresponding probability density function (PDF) is

$$f(x; \alpha, \lambda, \theta) = \alpha\lambda\left(1 - e^{-\lambda(x-\theta)}\right)^{\alpha-1} e^{-\lambda(x-\theta)}; \qquad x>\theta, \quad \alpha,\lambda>0$$

The corresponding survival function (SF) is

$$\bar{H}(x) = 1 - \left[1 - e^{-\lambda(x-\theta)}\right]$$

$$= e^{-\lambda(x-\theta)} \ldots (1)$$

Assume that shocks occur randomly in time in accordance with a three parameter generalized Exponential distribution. Taking the shape parameter as α = 1.

$$P(X_i < Y) = \int_0^{\infty} g^*(x)\bar{H}(x)dx$$

$$= [g^*\lambda(1-\theta)]^k \ldots (2)$$

The survival function which gives the probability that the cumulative threshold will fail onlyafter time t.

$S(t) = P(T > t)$ = Probability that the total damage beyond t

$$= \sum_{k=0}^{\infty} P\{\text{there are exactly k contacts in } (0,t] * P \text{ (the total cumulative threshold } (0,t]\}$$

It is also known from renewal process that

$P(\text{exactly k contact in } (0,t]) = F_k(t) - F_{K+1}(t)$ with$F_0(t) = 1$

$$P(T > t) = \sum_{k=0}^{\infty} V_k(t)P(X_i < Y)$$

$$= \sum_{k=0}^{\infty} V_k(t)[g^*\lambda(1-\theta)]^k \ldots (3)$$

Now, the lifetime is given by

$P(T < t) = L(t)$ = The distribution function of life time L(T); $L(T) = 1 - S(t)$

Taking Laplace transformation of L(t), we get

$$= 1 - \left\{\sum_{k=0}^{\infty}[F_k(t) - F_{k+1}(t)][g^*(\lambda - \lambda\theta)]^k\right\} \quad \text{on simpification we get}$$

$$L(T) = [1 - g^*(\lambda - \lambda\theta)]\sum_{k=1}^{\infty} F_k(t)[1 - g^*(\lambda - \lambda\theta)]^{k-1} \ldots (4)$$

By taking Laplace-Stieltjes transform, it can be shown that

$$l^*(s) = \frac{[1 - g^*(\lambda - \lambda\theta)]f^*(s)}{[1 - g^*(\lambda - \lambda\theta)f^*(s)]} \ldots (5)$$

Let the random variable U denoting inter arrival time which follows exponential with parameter. Now $f^*(s) = \left(\frac{c}{c+s}\right)$, substituting in the above equation we get

$$= \frac{[1 - g^*(\lambda - \lambda\theta)]\left(\frac{c}{c+s}\right)}{\left[1 - g^*(\lambda - \lambda\theta)\left(\frac{c}{c+s}\right)\right]}$$

$$= \frac{c[1 - g^*(\lambda - \lambda\theta)]}{[c + s - g^*(\lambda - \lambda\theta)c]} \ldots (6)$$

$$E(T) = -\frac{d}{ds}l^*(s) \text{given } s = 0$$

$$E(T) = \frac{1}{c[1 - g^*(\lambda - \lambda\theta)]}$$

$$\text{Now, } g^*(.) \sim \exp(\mu)\ ,\ g^*(\lambda) \sim \exp\left(\frac{\mu}{\mu+\lambda}\right)\ ,\ g^*(\lambda\theta) \sim \exp\left(\frac{\mu}{\mu+\lambda\theta}\right)$$

$$E(T) = \frac{1}{c\left[1 - \left(\frac{\mu}{\mu+\lambda} - \frac{\mu}{\mu+\lambda\theta}\right)\right]}$$

$$= \frac{\mu^2 + \mu\lambda\theta + \mu\lambda + \lambda^2\theta}{c[\mu^2 + 2\mu\lambda + \lambda^2\theta]} \ldots (7)$$

Where,

μ = Gleason

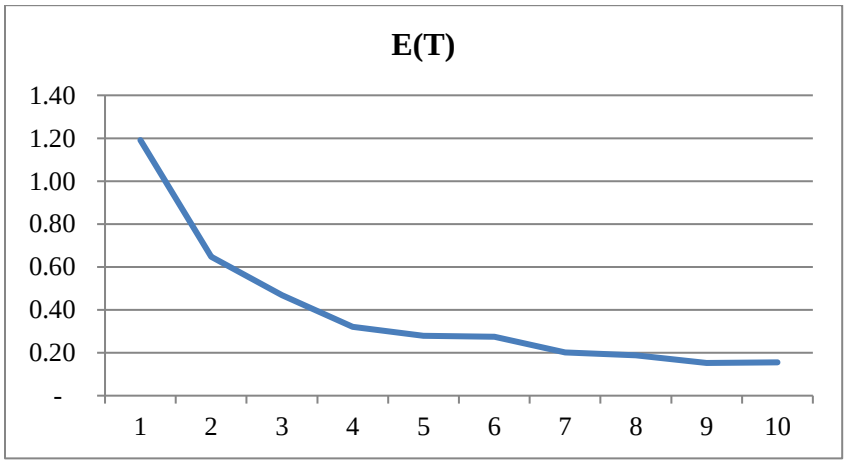
λ =PSA

θ = Prostate Imaging Reporting &Data System (PI-RADS)

TABLE – 1: The infected person's stage wise

C	μ = Gleason	λ =PSA	θ = (PI-RADS)	E(T)
1	5	6	2	1.19
2	6	8	3	0.65
3	8	9	4	0.47
4	6	12	4	0.32
5	8	12	5	0.28
6	11	8	6	0.27
7	12	13	4	0.20
8	14	14	5	0.19
9	12	16	4	0.15
10	16	13	5	0.15

Figure – 1: The Chart for infected person's Expected time



V. RESULTS AND DISCUSSION

In this study showed that the increase in the tumor growth for prostate cancer, the expected lifetime is decreased. This study infers that the person infected with tumor growth for prostate cancer gross the threshold level a lot of quickly. 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 placeable however unobserved cure method in addition to alcohol and transient quitting processes. During this analysis encompass those related to chronic medical conditions, psychological symptoms, alcohol use,

demographic variables, and alcohol history. History of chronic medical conditions was coded as self-reported presence or absence 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. Psychological symptoms were additionally coded as self-reported presence or absence 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). Alcohol history enclosed self-report of taking alcohol (always vs. other), total quantity of alcohol per day (continuous and categorical), and age of alcohol drinking onset, years alcohol-free, and quantity each year.

It has been wide distinguished that the quantity of people infected patients with advanced or pathological process has been increasing in recent years particularly in developing countries. The Gleason Score is that the grading system accustomed to verify the aggressiveness of prostate carcinoma. This grading system may be also opt for applicable treatment choices. The Gleason Score ranges from 1-5 and describes what proportion the cancer from a diagnostic test feels like healthy tissue (lower score) or abnormal tissue (higher score).Most cancers score a grade of three or higher. Since prostate tumors measure typically from cancerous cells that have completely different grades, 2 grades square measure assigned for every patient. A primary grade is given to explain the cells that conjure the most important space of the growth and a secondary grade is given to explain the cells of the next largest area. For instance, if the Gleason Score is written as 4+3=7, it means most of the tumor is grade 3 and the next largest section of the tumor is grade4, along they form up the entire Gleason Score.

If the cancer is nearly entirely form from cells with a similar score, the grade for that space is counted doubly to calculated the entire Gleason Score. Typical Gleason Scores range from 6-10.The higher the Gleason Score, a lot of probably that the cancer can grow and spreads quickly. There is no specific traditional or abnormal level of prostate specific antigen within the blood, and levels might vary over time within the same man. In the past, most doctors thought of prostate specific antigen levels of 4ng/mL and lower as normal. Therefore, if a person had a prostate specific antigen level more than 4 ng/mL, doctors would often recommend a prostate biopsy to determine whether prostate cancer was present. PI-RADS (Prostate Imaging–Reporting and knowledge System) may be a structured investigation for multiparametric prostate MRI within the analysis of suspected carcinoma in treatment naive prostate glands. Each lesion is assigned a score from one to five indicating the chance of clinically vital cancer: PI-RADS 1: terribly low (clinically vital cancer is extremely unlikely to be present) PI-RADS 2: low (clinically vital cancer is unlikely to be present)PI-RADS 3: intermediate (the presence of clinically vital cancer is equivocal)PI-RADS 4: high (clinically vital cancer is probably going to be present) PI-RADS 5: terribly high (clinically vital cancer is extremely probably to be present).

VI. CONCLUSION

In conclude that the person with Gleason score more than 6, PSA more than 4ng/ml and PI- RADS more than 3 is more quickly to cross the threshold level for prostate cancer. The time interval is the drinking of the infected person. The expected lifetime reduces quickly to the threshold level. The time interval duration for the infection depends on the period of alcohol contact of the infected person. The model shows that once the person is infected the breakdown of the immune system starts, which is seen in the above table and figures. We observe that once the cancer, good cells growths in tumor, affects the person and his immune system capacity is decreased. By Proper medical doctor advice and through regular treatment his life span can be extended.

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