

## SHOCK MODEL APPROACH WITH TIME FOR CANCER

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### Abstract:

Smoking is the root of the disease and a risk factor. Small cell carcinoma and non-small cell carcinoma are the two classifications of lung cancer that are used to assess the illness prognosis and available treatments. Depending on the type of tumour and the number of metastases, several signs and symptoms can sometimes be present. The diagnostic strategy for a lung cancer examination involves tissue diagnosis, including cancer evaluation, and histologic diagnosis. Treatment and prognosis are based on the type and stages of the cancer. There are currently no early diagnosis or screening treatments for lung cancer. Therefore, quitting smoking continues to be an important part of primary care prevention.

### INTRODUCTION

The most prevalent cancer in the world, with roughly 13% of all newly diagnosed cases in 2008 being lung cancer. Although the illness has frequently been seen as a burden on the industrialised world, nearly half of all malignancies are found in developing nations. The incidence of cancer varies greatly across different geographical regions. Around 70% of all new instances of carcinoma in the globe are found in wealthy nations. Although incidence rates are often lower in women, lung cancer is currently the fourth most common cancer in women internationally (516 000 cases, or 8.5% of all cancers), as well as the second leading cause of cancer death (427 000 deaths, or 12.8% of all cancer fatalities). Middle Africa has the lowest incidence rate, whereas Northern America has the highest, with lung cancer currently being the second most common cancer in women (15th most frequent cancer). In affluent nations, female incidence and mortality from carcinoma are increasing while male incidence and mortality are dropping.

Lung cancer remains the most important killer among all cancers within the world. It kills more people of both genders than the cancers of breast, colon and prostate combined, and more women than carcinoma. An awesome majority of cases is said to exposure to Polycyclic Aromatic Hydrocarbons (PAH), like benzo[a]pyrene, first of all, within the tobacco smoke, but genetic predisposition also plays a serious role. A comprehensive stochastic model of carcinoma should involve genetic and behavioral determinants of susceptibility, the progression of the disease from precursor lesions through early localized tumors to disseminated disease, detection

by various modalities, and medical intervention. The model should be ready to predict mortality reduction caused by early detection programs, under different scenarios, in presence of competing death causes. It'll be important to utilize the genetic indicators of susceptibility to carcinoma to define the highest-risk subgroups of the high-risk behavior population (smokers). One might consult Willcox, R.R. (1976), Purushottam A Giri, et al. (2013), Wodarz, D and M.A. Nowak (2002), Jeevaet al. (2004), and Sathiyamoorthi (1980) for further information about the anticipated time to pass the threshold level of the organisation.

### ASSUMPTIONS OF THE MODEL

The maximum that the human immune system can withstand is represented by the sum of the random variables. The projected time to cross the threshold level of the seroconversion phase is explored in greater detail by Esary et al. (1973) and Pandiyan et al. (2014). In this paper, a stochastic model is presented while accounting for an individual's risk.

Smoking and alcohol usage are the two main factors contributing to the development of lung cancer tumours. The threshold for each individual is a random variable. If the cumulative harm is greater than a threshold level  $Y$ , which is a random variable in and of itself, the person is said to be infected. The same random variables govern the space between subsequent haplite arrivals.

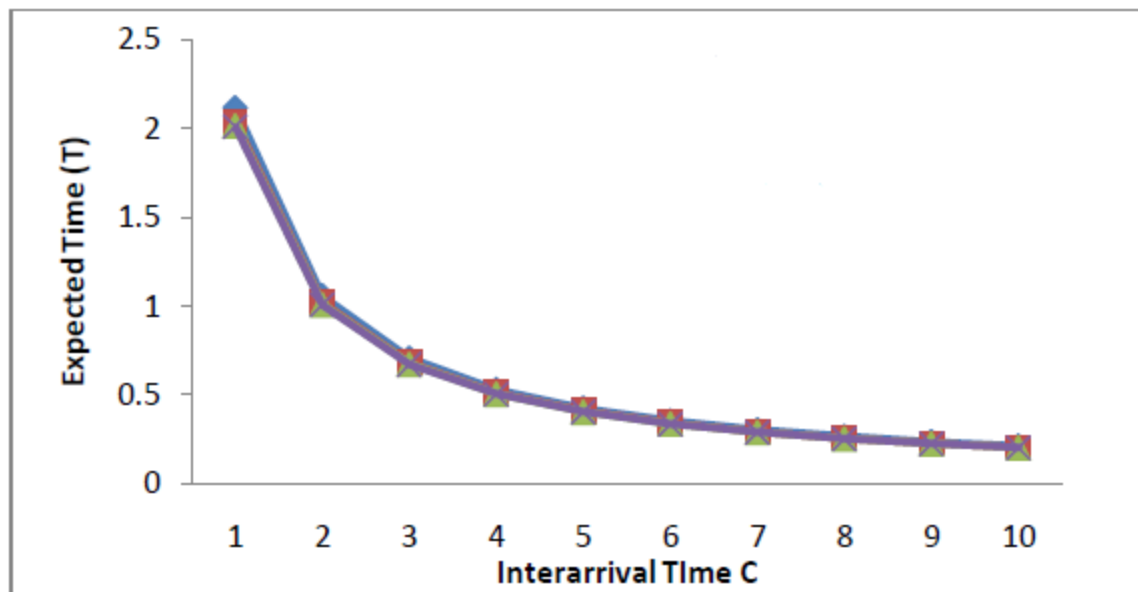
### MODEL DESCRIPTION

The Cumulative density function (CDF) of the Distribution function Exponential with parameter is

$$F(x; \alpha) = 1 - e^{-\alpha x}; \quad x > 0,$$

It can be seen that when the threshold level is denoted and the life time is transformed using the exponential and erlang distributions, we obtain By substituting the random variable  $U$ , which represents the inter-arrival time and has an exponential relationship with the parameter, into the previous equation, we obtain

$$E(T) = \frac{1}{c} \left[ \left\{ -\sum_{i=1}^4 \left( \frac{\mu + \alpha_i}{\alpha_i} \right) + \sum_{i=3}^3 \left( \frac{\mu + \alpha_i + \alpha_{i+1}}{\alpha_i + \alpha_{i+1}} \right) + \left( \frac{\mu + \alpha_2 + \alpha_4}{\alpha_2 + \alpha_4} \right) + \left( \frac{\mu + \alpha_1 + \alpha_3}{\alpha_1 + \alpha_3} \right) + \left( \frac{\mu + \alpha_1 + \alpha_2}{\alpha_1 + \alpha_2} \right) + \left( \frac{\mu + \alpha_1 + \alpha_2 + \alpha_3}{\alpha_1 + \alpha_2 + \alpha_3} \right) + \left( \frac{\mu + \alpha_1 + \alpha_2 + \alpha_4}{\alpha_1 + \alpha_2 + \alpha_4} \right) + \left( \frac{\mu + \alpha_2 + \alpha_3 + \alpha_4}{\alpha_2 + \alpha_3 + \alpha_4} \right) + \left( \frac{\mu + \alpha_1 + \alpha_3 + \alpha_4}{\alpha_1 + \alpha_3 + \alpha_4} \right) + \left( \frac{\mu + \alpha_1 + \alpha_2 + \alpha_3 + \alpha_4}{\alpha_1 + \alpha_2 + \alpha_3 + \alpha_4} \right) \right\} + \left\{ \left( \frac{\mu + \alpha_4}{\alpha_4} \right) - \left( \frac{\mu + \alpha_3 - \alpha_4}{\alpha_3 - \alpha_4} \right) - \left( \frac{\mu + \alpha_2 - \alpha_4}{\alpha_2 - \alpha_4} \right) + \left( \frac{\mu + \lambda_2 + \lambda_3 - \lambda_4}{\lambda_2 + \lambda_3 - \lambda_4} \right) - \left( \frac{\mu + \alpha_1 - \alpha_4}{\alpha_1 - \alpha_4} \right) + \left( \frac{\mu + \alpha_1 + \alpha_3 - \alpha_4}{\alpha_1 + \alpha_3 - \alpha_4} \right) + \left( \frac{\mu + \alpha_1 - \alpha_4}{\alpha_1 - \alpha_4} \right) - \left( \frac{\mu + \alpha_1 + \alpha_2 + \alpha_3 - \alpha_4}{\alpha_1 + \alpha_2 + \alpha_3 - \alpha_4} \right) \right\} \alpha_4 \right] \dots\dots\dots$$



## CONCLUSION

The estimated time is the infected person's drinking. The patient quality of life duration rapidly approaches the threshold level. The infection's time interval duration is determined by the affected person's smoking contact. The model proposes that after a person is infected, the immune system begins to deteriorate, as seen in the table and figures above. We see that when a person is impacted by cancer, beneficial cells increase in the tumour and cells are harmed, and his or her immune system capacity decreases. His/her long life can be increased with proper medical practitioner advice and regular treatments.

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