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COMPARISON OF BAYESIAN AND FREQUENTIST SURVIVAL ANALYSIS METHODS IN MODELLING OF PROSTATE CANCER SURVIVORSHIP IN OYO STATE, NIGERIA

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ABSTRACT

Background: Survival analysis is a statistical method for modelling the probability that a subset of a given population will survive past a certain time. In the context of cancer, this probability would represent recurrence of tumour, or remission (i.e. being disease-free). This study seeks to compare the traditional frequentist approach and the Bayesian approach to survival analysis in estimating, and the predictors of prostate cancer (CaP) survivorship. Prostate cancer starts when healthy cells in the prostate gland change and grow out of place, forming a mass called a tumour.

Method: A retrospective analytical study design was employed, through extraction of case files patients diagnosed and treated for CaP from January 2010 to December 2017 at UCH, Ibadan. The extracted data was further divided into two cohorts (2010 - 2014) and (2015 - 2017). A proforma was used for extraction which includes the following sections; socio-demographics, clinical/pathological characteristics, date of diagnosis, date last seen, and treatment received. Descriptive statistics was used to describe these characteristics. The survival probability was determined by the KM survival method. Cox proportional hazard (CPH), Weibull AFT and Bayesian Weibull AFT (using normal prior distribution) models was used to determine predictors of survivorship.

Results: The average age of the patients was 72 years, with peak incidence of CaP among those aged 70 – 79 years. Most patients 87.3% were diagnosed at stage IV, with many having metastasis to the spine. Among the patients, 33.6% received chemotherapy and surgery. Patients from North central had the highest median survival (MS) time in the (2015 - 2017) cohort. The overall MS time for the (2010 - 2014) cohort was 2.9 months, and 3.3 months for the (2015 - 2017) cohort while the overall MS time for the study was 3.2 months. Patients treated with chemotherapy and

surgery in both cohorts experienced delayed remission. The Weibull AFT model found that patients with a moderately differentiated Gleason experienced a 50% increase in time ($TR = 0.5$; 95%CI: 0.3 – 0.9) to remission. Patients, with a poorly differentiated Gleason, experienced a 70% decrease in time ($aTR = 1.7$; 95%CI: 1.0 - 2.7) to remission. The Bayesian AFT model also found delay in time to remission for patients with moderately differentiated Gleason ($TR = 0.6$; 95%CrI: 0.3 – 0.9), and those treated with Chemotherapy and Surgery ($aTR = 3.3$; 95%CrI: 2.6 – 4.4). The Bayesian model showed that age, south-south, north-central, no family history of CaP, moderately and poor differentiated Gleason and treatment with Chemotherapy, Radiotherapy and Surgery, Chemotherapy and Surgery to significantly delay time to remission.

Conclusion: This study found that in considering predictors of survivorship a host of factors should be considered, particularly age, location, marital status, occupation, stage, method of diagnosis, Gleason group, site and treatment received. In terms of approaches to survival analysis, greater emphasis should be given to the Bayesian approach, as observed in this study, the Bayesian approach extracted more significant predictors of survivorship than the CPH and Weibull AFT models and besides, it is more suitable for studies with fewer observations.

Keywords: survival analysis, parametric, frequentist, Bayesian, prostate cancer.

Introduction

Survival analysis is a method for modeling time duration to the occurrence or recurrence of events. It is also called reliability theory or reliability analysis in the field of engineering. In general, survival analysis is a statistical modeling approach that involves time to event data. An important aspect of survival analysis data is censoring and non-normality. Non-normality of the data violates the normality assumption of most commonly used statistical method such as multiple linear regressions (Oakes, 1972). The objective of using survival analysis is to answer the question of, what is the probability that subset of a given population will survive past a certain time? In the context of cancer, this probability would represent death, relapse, recurrence of the tumour, or remission (i.e. being disease-free) of a cancer patient.

Background/rationale

Frequentist methodology of survival analysis

Much of the development of frequentist methodology is attributed to the work carried out by R.A. Fisher in the early 20th century. The frequentist approach to statistics evaluates procedures based on imagining repeated sampling from a particular model (the likelihood), which defines the probability distribution of the observed data conditional on unknown parameters. Properties of the procedure are evaluated in this repeated sampling framework for fixed values of unknown parameters; good procedures perform well over a broad range of parameter values (Gandenberg, 2014a, 2014b, 2014c). Survival analysis in the frequentist framework is based on non-parametric methods (Nelson Aalen, Kaplan Meier), Semi-parametric (Cox Proportional Hazard) and the parametric (Weibull, Exponential, Log-logistic etc) models.

Bayesian methodology of survival analysis

The introduction of Bayesian methodology can be attributed to a posthumous paper titled “An essay towards solving the Problem in the Doctrine of Chances” by the Reverend Thomas Bayes in 1763 (Bayes, 1763). Despite its early inception, frequentist methods remain the dominant methodology in modern statistics. In part, this was due to the lack of modern sampling techniques that require substantial computing power (Ibrahim, Chen, & Sinha, 2005). Indeed, for all but the simplest Bayesian models, computation methods were complex and was often impossible to find

any analytical solutions. Whilst advances in theory and computing have made Bayesian methods more accessible, their uses in practice still remain somewhat limited.

In estimating the parameter θ associated with data $\mathbf{x}$, under the Bayesian approach, $P(\mathbf{x}|\theta)$ is evaluated where $P(\cdot)$ represents some probability density (Jackson, 2015). Bayesian methodology considers the data, once observed, to be a fixed quantity and allows the parameters to be the random variables; thus, instead evaluating $P(\mathbf{x}|\theta)$.

Evaluation of $P(\mathbf{x}|\theta)$ is determined via use of Bayes' theorem for conditional probability

$$P(\theta|\mathbf{x}) = \frac{P(\mathbf{x}|\theta)P(\theta)}{P(\mathbf{x})}$$

Under conventional notation, define

$$P(\theta|\mathbf{x}) = \textit{Posterior probability}; P(\mathbf{x}|\theta) = \textit{Likelihood}; P(\theta) = \textit{Prior probability}; \\ P(\mathbf{x}) = \textit{marginal probability}$$

All inferences are made on summaries of the posterior probability density, the posterior is a product of the information obtained from the data and the information taken from prior knowledge. Note that the marginal density $P(\mathbf{x})$ is simply the probability of observing the data which is not dependent upon the parameter θ (Berger, 1985; Box & Tiao, 1992).

Prostate cancer starts when healthy cells in the prostate gland change and grows out of place, forming a mass called a tumour. (ASCO, 2015) The Prostate is a walnut-sized organ located just between the bladder and the male reproductive organ, and it slowly grows larger to an average weight of 40 grams in ageing men. A prostate gland surrounds the urethra that empties urine from the bladder and also secretes prostate fluid that protects sperm. These physiological functions may be compromised during various prostate diseases including prostatitis, benign prostatic hyperplasia or hypertrophy, and cancer (Kgatlé, Kalla, Islam, Sathekge, & Moorad, 2016). The size of the prostate changes with age (ACS, 2016). Pathologic evidence suggests that neoplastic changes of the prostate epithelium develops early in a man's adult life, but do not become clinically evident or problematic until decades later. Some patients live out their lives with prostate cancer that remains stable for decades without treatment. In other cases, cancer grows aggressively, responds poorly to therapy, and causes death within a few years (Sunny, 2005).

Clinically detected prostate carcinomas display a variety of phenotypic and malignant potential. Majority of all prostate carcinomas are typical adenocarcinomas, which can be divided into different tumour grades. The histological differentiation together with tumour stage, determined by tumour size, as well as the presence of lymph node and distal metastases are used to assess the prognosis of the patients (DF, 1992). Epidemiology studies have shown that there are environmental as well as genetic contributions to the development of prostate cancer. Prostate cancer is a heterogeneous disease, and there is large variations from patient to patient even within the same tumour. These differentials in the Prostate cancer architecture and incidence rates may be attributable to genomic instabilities and alterations associated with various Prostate cancer risk factors.

Cancer of the prostate is primarily a disease of the elderly men. About three-quarters of cases worldwide occur in men aged 40 years and above with increasing incidence and morbidity in men of black African ancestry (Fernandez et al., 2012). In 2012, 1.1 million men were diagnosed with CaP and 70% of them (795,000 cases) were in developed countries (Mohammadian-hafshejani, Ghoncheh, Towhidi, Jamehshorani, & Salehiniya, 2016). Available reports indicate that CaP accounts for approximately 20.3% and 4.8% of all male cancers in Sub-Saharan Africa and North Africa, respectively (Cooney, Okuku, & Orem, 2016; Fernandez et al., 2012). CaP is on the upward trajectory among Nigerian men and remains one of the most common cancer among men in this region (T O Akinremi, Adeniyi, Olutunde, Oduniyi, & Ogo, 2014). A prevalence rate of 1046 per 100,000 men of age > 40 screened (Ikuerowo et al., 2013) was reported in Lagos. Compared to African-American men, Nigerian men are 10 times more likely to have prostate cancer and 3.5 times more likely to die from it. Environmental and most importantly, genetic factors have been incriminated as the reason for the geographic differences in incidence. Hence, this study seeks to determine the survivorship rate and to compare and contrast the effectiveness of frequentist and Bayesian approaches in modelling the determinants of survivorship among prostate cancer patients.

Methods

Study design

A retrospective analytical study design was employed. This was done through a review and extraction of hospital records, case files of all patients diagnosed and treated for prostate cancer from January 2010 to December 2017 at the University College Hospital (UCH) Ibadan was included in the study.

Study setting

The study site includes all patients receiving treatment for prostate cancer at the radiation oncology department of UCH, Ibadan. The department offers services in the area of counselling/psychotherapy, group meeting with patients for problem shearing, alleviation of psychological concern, simulation, tumour localization procedure, palliative care, holistic management for cancer sufferers and their families, External beam radiation therapy, radiotherapy treatment for cancer patients using Cobalt 60 therapy machine, gynecology brachytherapy, ward admission, and clinic services.

Study Population

This study consists of all Prostate cancer patients who were managed for prostate cancer at the University College Hospital, Ibadan from January 2010 to December 2017.

Data Collection Procedure

A proforma was used to extract relevant data from patient's records, with the assistance of medical record clerks. The extracted data was divided into two cohorts (2010 - 2014) and (2015 - 2017). A total of 354 patients' records was extracted of which in the (2010 – 2014) cohort, 110 were disease-free while 54 were censored. Also, in the (2015 - 2017) cohort, 115 patients were disease-free while 75 were censored; with a follow up time of 15 months observed in each cohort. To ensure data reliability, the extracted records was cross-checked by a radiotherapist. The sections in the data collection form included patients' socio-demographics, diagnosis, clinical and pathological features, treatment method, and follow-up.

Study Variables

Dependent variable

The dependent variable was the time to being disease-free (remission) after treatment, measured in months from time of presentation to time patient was last seen. A patient or an individual is declared to be disease-free when there is no residual disease on medical examination after completion of treatment. Meanwhile patients still under follow up, lost to follow up, death from CaP or other diseases were censored.

Independent variables

The independent variables for this study include patient's socio-demographics characteristics, clinical characteristics, time of diagnosis, time last seen, time last treated, treatment received and treatment outcome. A description of the coding method is giving in the table below.

Independent variables

Variable	Levels
<i>Socio-demographics</i>	
Age	1=50 – 59, 2= 60 – 69, 3 = 70 – 79, 4= 79 - above
Marital status	1=married 2=never married 3=widower, 4=divorced
Occupation	1=civil servant, 2=farming, 3=factory worker, 4=unemployed, 5=others
Religion	1=Christian 2=Islam, 3=Traditional, 4=Others
Geographical location	1=south west, 2=south south, 3=south east, 4=north west, 5=north east, 6=north central
Co-morbidities	1=coronary artery disease, 2=diabetes, 3=hypertension/stroke, 4=obesity, 5=psychosocial stress, 6=alcohol/tobacco, 7=chemical exposure, 8=others
Family history of CaP	1=yes, 2=no
<i>Clinical/pathological characteristics</i>	
Method of diagnosis	1=PSA test, 2=DRE, 3=Transrectal ultrasound 4=biopsy, 5=DRE and biopsy
PSA level at diagnosis	ng/ML
Stage at diagnosis	1=stage I, 2=stage II, 3=stage III, 4=stage IV 5=others
Site of metastasis	1=lymph node, 2=rectum (bladder neck), 3=bones, 4=spine, 5=multiple metastasis
Gleason group	1=well differentiated, 2=moderately differentiated, 3=poorly differentiated
<i>Treatment</i>	

Treatment received	1=surgery, 2=radiotherapy, 3=chemotherapy, 4=radiotherapy and surgery, 5=chemotherapy and radiotherapy, 6=chemotherapy and surgery, 7=chemotherapy + surgery + radiotherapy
Treatment outcome	1=disease-free, 2=death from CaP or other disease, 3=still under follow up, 4=loss to follow
<i>Follow up time</i>	
Date of diagnosis	dd/mm/yy
Date treatment started	dd/mm/yy
Date last treated	dd/mm/yy
If loss to follow up, date last seen	dd/mm/yy

Data Management

Descriptive statistics was used to describe patient's socio-demographic characteristics, clinical characteristics and treatment modality, (frequency, proportions,). Survival probability was determined by Kaplan-Meier survival method. Semi-parametric (Cox proportional hazard) and Bayesian and Frequentist parametric (Weibull) models was used to determine factors affecting survivorship. STATA 15 was employed in the analysis. All analysis was carried out at 5% significance level.

Determination of survival probabilities

Due to the presence of right censored data, the Kaplan-Meier method was used in this study.

Suppose we have p distinct survival times arranged in increasing order, say $t_1 < t_2 < \dots < t_p$. At time t_i there are n_i subjects who are said to be at risk i.e. they survived up to this time and were not censored. The number of subjects who failed at time t_i is denoted by d_i . Consider the recursive equation

$$\begin{aligned} S(t_i) &= P(T > t_i) = P(T > t_i | T > t_{i-1}) * P(T > t_{i-1}) \\ &= P(T > t_i | T > t_{i-1}) * S(t_{i-1}), i = 1, \dots, p \end{aligned}$$

Where $t_0 = 0$ and $S_0 = 1$

Therefore, the survival function is given as

$$s(t) = S'(t) = \frac{d}{dt} S(t) = \frac{d}{dt} \int_t^{\infty} f(u) du = \frac{d}{dt} [1 - F(t)] = -f(t) \quad 1.0$$

The survival time t is the time period (months) to being disease-free from time of diagnosis to time last treated/seen, since all patients are at risk of this event. The censoring index for patients who were disease-free is “1” whereas patients who are still undergoing treatment, died from CaP or a disease other than CaP, patients who can no longer be observed (Loss-to-follow-up), are declared censored and coded as “0”. While the log-rank test was used to test the equality of survival curves.

Cox Proportional hazard model

As a nod to the proportional hazard assumption, consider two subjects for which the values of the covariates are $x_{i1}, \dots, x_{ip}$ and $x_{j1}, \dots, x_{jp}$, respectively. The ratio of the hazard function is

$$\frac{h(t, x_{i1}, \dots, x_{ip}, \beta_1, \dots, \beta_p)}{h(t, x_{j1}, \dots, x_{jp}, \beta_1, \dots, \beta_p)} = \exp \{ \beta_1(x_{i1} - x_{j1}) + \dots + \beta_p(x_{ip} - x_{jp}) \} \quad 1.1$$

Given the risk of death at a particular time, depends on the values $x_1, x_2, \dots, x_p$ of p explanatory variables, $X_1, X_2, \dots, X_p$. These values will be assumed to have been recorded at the time origin of the study; assuming the explanatory variables do not depend on time, the hazard function has the following form:

$$h(t, x_1, \dots, x_p, \beta_1, \dots, \beta_p) = h_0(t) \exp \{ \beta_1 x_1 + \dots + \beta_p x_p \}$$

The function $h_0(t)$ is called the baseline hazard function

For this study, the Proportion hazard model was fitted for the following independent variables (age, occupation, marital status, methods of diagnosis, Gleason group, PSA level, Stage at diagnosis, site of metastasis, treatment type). The coefficients of the independent variables were estimated using partial likelihood method, using the “stcox” package in STATA 15.

$$\text{i.e. } \log \left(\frac{h_i(t)}{h_0(t)} \right) = B_1 X_1 + B_2 X_{2i} + B_3 X_{3i} + B_4 X_{4i} + B_5 X_{5i} + B_6 X_{6i} + B_7 X_{7i} + B_8 X_{8i} + B_9 X_{9i}$$

Where

$X_1 = \text{Age}, X_{2i} = \text{Occupation}, X_{3i} = \text{Marital Status}, X_{4i} = \text{Method of diagnosis}$
 $X_{5i} = \text{Grade of disease}, X_{6i} = \text{PSA level at diagnosis}, X_{7i} = \text{Stage at diagnosis}$
 $X_{8i} = \text{Site of metastasis}, X_{9i} = \text{Treatment modality}$

The Weibull Accelerated Failure time (AFT) Model (Frequentist)

Accelerated failure time models, also known as accelerated-time models or $\ln(\text{time})$ models follow the parameterization

1.2

$$\ln(t_j) = x_j\beta_X + \epsilon_j$$

The word “accelerated” is used in describing these models because rather than assuming that failure time t_j is exponential, Weibull, or some other form, a distribution is instead assumed for

$$\tau_j = \exp(-x_j\beta_X)t_j$$

and $\exp(-x_j\beta_X)$ is called the acceleration parameter

Assuming that the survival time follows a Weibull distribution with scale parameter β_0 and shape parameter p , written as

$$\tau_j \sim Weibull(\beta_0, p)$$

τ_j is distributed as Weibull with parameters (β_0, p) .

The Weibull hazard and survivor functions are

$$h_0(t) = \beta_0 p t^{p-1}$$

and

$$S_i(t) = \exp(-\beta_0 t^p)$$

respectively,

Since the effect of the covariates is to accelerate time by a factor of $\exp(-x_j\beta_X)$, thus for the AFT model

$$\begin{aligned} S(t_j|x_j) &= S_0\{\exp(-x_j\beta_X)t_j\} \\ &= \exp[-\{\exp(-\beta_0)\exp(-x_j\beta_X)t_j\}^p] \\ &= \exp[-\{\exp(-\beta_0 - x_j\beta_X)t_j\}^p] \end{aligned} \tag{1.3}$$

The “*streg*” package in STATA 15 was used to estimate the maximum likelihood estimate for the Weibull AFT model by specifying the “*tr(time ratio)*” option. Essentially, the accelerated failure time implies a deceleration of time or, in other terms, an increment in the expected waiting time for an event of interest to occur. Time Ratio (TR) > 1 indicates that an individual experience being disease-free at an earlier timing, TR = 1 implies no difference in survival time, while TR < 1 suggests that an individual experienced a delay or decreased in time to being disease-free.

Bayesian Survival Analysis

Bayesian analysis generates conclusions based on the synthesis of new information from the observed data and previous knowledge or external evidence. The Weibull Model is one of the

accelerated failure time models that can be used fit survival data under the Bayesian framework. Let t_i denote the survival time of the i^{th} patient. Assuming that t_i has a Weibull distribution with parameters $\alpha > 0$ and $r > 0$, with a density function of the form:

$$f(t_i|\alpha, r) = \alpha t_i^{\alpha-1} \exp(-[rt_i]^\alpha), \quad 0 < t_i < \infty \quad 1.4$$

The survival function of t_i is given by

$$S(t_i|\alpha, r) = \exp(-\exp(rt_i)^\alpha t_i^\alpha) \quad 1.5$$

The hazard function is given by

$$h(t_i|\alpha, r) = r \alpha t_i^{\alpha-1} \exp(-[rt_i]^\alpha) \quad 1.6$$

The likelihood function of the unknown parameters (α, r) given the data can be written as:

$$L(\alpha, r|D) = \prod_{i=1}^n f(t_i|\alpha, r) S(t_i|\alpha, r)^{(1-\delta_i)}$$

$$= \alpha \sum \delta_i \exp\{r \sum_{i=1}^n \delta_i + \sum_{i=1}^n \delta_i (\alpha - 1) \log(t_i) - \exp(r) t_i^\alpha\}$$

Where δ_i is an indicator variable taking value 1 if t_i is the failure time (i.e patients who are disease-free) and 0 if t_i is right censored (loss-follow-up, death, still under follow up).

To incorporate covariates we, therefore, write $r = X'_i \beta$, where X'_i and β are $p \times 1$ vector of covariates and regression coefficients respectively.

Assuming a normal prior for β , $N_p(\mu_0, \Sigma_0)$ then the joint posterior is given by

$$\prod (\beta, \alpha|D)^\alpha \alpha^{\alpha_0+d+1} \exp \sum_{i=1}^n (\delta_i + X'_i \beta + \delta_i (\alpha - 1) \log(t_i)) - t_i^\alpha \exp(X'_i \beta) - k_0^\alpha - \frac{1}{2} (\beta - \mu_0)' \Sigma_0^{-1} (\beta - \mu_0)$$

Where $D = (n, t, X, \delta)$ denote the observed data for the regression model and X is an $n \times p$ matrix of covariates with the i -th row as X'_i and $\delta = (\delta_1, \dots, \delta_n)'$.

The “bayes, streg, tr” package in STATA 15 was used to fit the Bayesian AFT model using the random-walk Metropolis-Hastings Monte Carlo Markov Chain (MCMC) sampling method for simulating the results of the parameters. The Metropolis-Hastings algorithm is given as follows:

Let $q(\theta, \vartheta)$ be a proposal density which is also termed as a candidate generating density such that

$$\int q(\theta, \vartheta) d\vartheta = 1$$

Also, let $\mu(0,1)$ denote the uniform distribution over $(0,1)$. Then, a general version of the Metropolis-Hastings algorithm for sampling from the posterior distribution $P(\boldsymbol{\theta}|\mathbf{x})$ can be described as follows:

Step 0. Choose an arbitrary starting point θ_0 and set $i = 0$.

Step 1. Generate a candidate point θ^* from $q(\theta_i, \cdot)$ and u from $\mu(0,1)$

Step 2. Set $\theta_{i+1} = \theta^*$ if $u \leq a(\theta_i, \theta^*)$ and $\theta_{i+1} = \theta_i$ otherwise, where the acceptance probability is given by

$$a(\theta, \vartheta) = \min \left\{ \frac{\pi(\vartheta|D)q(\theta, \vartheta)}{\pi(\theta|D)q(\theta, \vartheta)}, 1 \right\}$$

Step 3. Set $i = i + 1$, and go to Step 1.

The iteration steps from 0 through 3 is referred to as an MH update. By design, any Markov chain simulated using this MH algorithm is guaranteed to have the posterior distribution as its stationary distribution.

Criteria's for measuring the efficiency of MCMC are the acceptance rate of the chain and the degree of autocorrelation in the generated sample. When the acceptance rate is close to "0" then most of the proposals are rejected, which means that the chain failed to explore regions of appreciable posterior probability. The cut-off for acceptance rate is 0.234 for a multivariate posterior and 0.45 for a univariate posterior.

Model Evaluation Methods

The model was evaluated using -2LogL, Akaike Information Criterion (AIC) Bayesian Information Criterion (BIC) Deviance Information Criterion (DIC). Among the (2010 - 2014), (2015 - 2017) cohorts model and overall group, any of Cox Proportional Hazard (CPH), parametric regression model with consistent low values of -2LL, AIC and BIC was selected as the best fitting model while for Bayesian regression a model with consistently low values of -2LL, AIC, BIC, DIC and an acceptance rate of 0.2 for the multivariate model and 0.4 for the univariate model was selected as the best fitting model.

Ethical consideration

Ethical approval was obtained from UI/UCH Ethics Committee of the University of Ibadan approval reference: UI/EC/18/0492

Results

Distribution of prostate cancer (CaP) patients by sociodemographic characteristics

Three hundred and fifty-four patients were observed age 50 – 104 years, with an average age of 72years and a standard deviation of 9.52. Patients 50 – 59 years were 33(9.3%), 60 – 69 years 90(25.4%) The peak incidence of CaP was observed among those age 70 – 79 years 141(39.8%) while those 79 years above were 90(25.4%). Those married were 347(97.7%) while 8(2.3%) were widowers. 181(51.1%) civil servants, 4(1.1%) unemployed, 9(2.5%) farmers and 16(4.5%) factory workers and others (taxi drivers, artisans, labourers, business men) 144(40.7%).

Of the 354 cases, 257(72.6%) were Christians, 94(26.6%) Islam while 3(0.8%) practised other religion, 278(78.5%) were from south-western Nigeria 38(10.7%) south-south, 30(8.5%) south-east while 8(2.3%) from north central. Those with a family history of prostate cancer were 77(21.8%) while 167(33.0%) smoked tobacco or drank alcohol, 101(20.0%) had coronary heart disease, 118(23.3%) diabetes, and 120(23.7%) were hypertensive (table 1).

Table 1: Distribution of prostate cancer patients by sociodemographic characteristics

Characteristics	n	%
<i>Age (years)</i>		
50 – 59	33	9.3
60 – 69	90	25.4
70 – 79	141	39.8
79 – above	90	25.4
<i>Marital Status</i>		
Currently married	346	97.7
Widower	8	2.3
<i>Occupation</i>		
Civil Servant	181	51.1
Farming	9	2.5
Factory Worker	16	4.5
Unemployed	4	1.1
Others	144	40.7
<i>Religion</i>		
Christian	257	72.6
Islam	94	26.6
Others	3	0.8
<i>Geographical Location</i>		
South-west	278	78.5

South-south	38	10.7
South-east	30	8.5
North-central	8	2.3
<i>Family History of Prostate Cancer</i>		
Yes	77	21.8
No	277	78.2
<i>Year of Diagnosis</i>		
2010	45	12.7
2011	18	5.1
2012	4	1.1
2013	26	7.3
2014	71	20.1
2015	64	18.1
2016	59	16.7
2017	67	18.9
<i>Co-morbidities</i>		
Alcohol and Tobacco	167	33.0
Coronary Artery	101	20.0
Diabetes	118	23.3
Hypertension/Stroke	120	23.7
Total	354	100.0

Others include: taxi drivers, artisans, labourers, business men

Distribution of CaP patients by clinical characteristics

Of the 354 Patients enrolled into this study, 193(54.5%) were diagnosed via Digital Rectal Examination (DRE), Biopsy 98(27.7%), Prostate Specific Antigen test 20(5.6%), while 34(9.6%) using both DRE and Biopsy. Patients with a poorly differentiated Gleason score were 220(62.1%), 62(17.5%) were moderately differentiated while 72(20.3%) were well differentiated; 309(87.3%) were diagnosed at stage IV while 11(3.1%) were diagnosed at stage I, 18(5.1%) had metastasis to the lymph node, 55(15.5%) rectum, 84(23.7%) bones, 116(32.8%) spine, while about a quarter had multiple metastases 81(22.9%).

Surgery was carried out on 67(18.9%) patients, while 42(11.9%) and 62(17.5%) received radiotherapy and chemotherapy respectively, combination therapy was applied to 7(2.0%), radiotherapy and surgery 48(13.6%), 48(13.6%) chemotherapy and radiotherapy, 119(33.6%) Chemotherapy and surgery while 9(2.5%) received all the three combinations of chemotherapy, surgery and radiotherapy. Of the total patients record extracted during this period, 225(55.6%)

were disease-free after treatment, 17(10.2%) died from prostate cancer, 75(25.0%) were still under follow-up while 37(9.2%) were lost to follow-up (table 2).

Table 2: Distribution of prostate cancer patients by clinical characteristics

Characteristics	n	%
<i>Method of Diagnosis</i>		
PSA test	20	5.6
DRE test	193	54.5
Transrectal Ultrasound	9	2.5
Biopsy	98	27.7
DRE & Biopsy	34	9.6
<i>Gleason group</i>		
Well-differentiated	72	20.3
Moderately differentiated	62	17.5
Poorly differentiated	220	62.1
<i>Stage at Diagnosis</i>		
Stage I	11	3.1
Stage II	12	3.4
Stage III	12	3.4
Stage IV	309	87.3
Unknown	10	2.8
<i>Site of Metastasis</i>		
Lymph node	18	5.1
Rectum (bladder neck)	55	15.5
Bones	84	23.7
Spine	116	32.8
Multiple Metastasis	81	22.9
<i>Treatment Modality</i>		
Surgery alone	67	18.9
Radiotherapy alone	42	11.9
Chemotherapy alone	62	17.5
Radiotherapy & Surgery	7	2.0
Chemotherapy & Radiotherapy	48	13.6
Chemotherapy & Surgery	119	33.6
Chemotherapy & Surgery & Radiotherapy	9	2.5
<i>Treatment Outcome</i>		
Disease-free	225	55.6
Death from Prostate cancer	17	10.2
Still under follow-up	75	25.0

Loss-to-follow up	37	9.2
Total	354	100.0

Figure 1: Distribution of CaP patients by stage at diagnosis and site of metastasis

Median survival time of socio-demographics and clinical characteristics among CaP patients

The result showed that a total of 354 patients were diagnosed and treated for CaP between 2010 – 2017, with a follow up time of 15 months observed for each patient, of which for the 164 patients in the 2010 – 2014 cohort, 110 were disease-free while 54 were censored. Among the 190 patients in the 2015 – 2017 cohort; 115 were disease-free while 75 were censored. It was observed that patients aged 50 – 59 years in the 2010 – 2014 cohort had a MS time of 3.5 months while those in the 2015 – 2017 cohort had a MS time of 3.8 months, patients 60 – 69 years in the 2010 – 2014 cohort had a MS time of 4.2 months; meanwhile patients age 79 years and above had a median survival time of 2.2 months in the 2010 – 2014 cohort (table 3).

Those who were married, had a MS time of 2.8 months among the 2010 – 2014 cohort and 3.3 months among the 2015 – 2017 cohort respectively and an overall median survival time of 3.2 months, while widowers had a median survival time of less than one month among patients in the 2010 – 2014 cohort and 0.9 months among the 2015 – 2017 cohort. Patients from the south-western

part of the country had a MS time of almost three months for the 2010 – 2014 cohort and more than three months for the 2015 – 2017 cohort of patients, while patients from the south-south region had a MS time of 1.5 months for the 2010 – 2014 cohort of patients and more than four and a half months among the 2015 – 2017 cohort; overall MS time was 2.6 months. The highest MS time was recorded amongst those from the north central region 14.2 months in the 2015 – 2017 cohort (table 3).

The MS time of patients with a family history of CaP among the 2010 – 2014 cohort of patients was three and a half months, and an overall MS time of 2.8 months while those with no family of CaP had a MS time of 2.9 months for the 2010 – 2014 cohort of patients and 3.5 months among the 2015 – 2017 cohort. Overall MS time was 3.1 months for those with no family history of prostate cancer (table 3).

MS time among patients diagnosed via Prostate Specific Antigen (PSA) test was 3.1 months among the 2010 – 2014 cohort and 2.8 months for the 2015 – 2017 cohort while those diagnosed using Digital Rectal Examination (DRE) had a MS time of 2.9 months for the 2010 – 2014 cohort of patients and more than three and a half month for the 2015 – 2017 cohort with an overall MS time of three and a half month, similarly patients diagnosed with a combination of Biopsy and DRE had a MS time of a little above two and half months for the 2010 – 2014 cohort of patients and more than four months for the 2015 – 2017 cohort. Patients whose Gleason group were categorized as well differentiated had a median survival time of 1.9 months for the 2010 – 2014 cohort of patients and 4.7 months for the 2015 – 2017 cohort (table 3); while those poorly differentiated had a MS time of 3.5 months for the 2010 – 2014 cohort and 3.3 months for the 2015 – 2017 cohort of patients. Probability of being disease-free was highest among those diagnosed via DRE and Biopsy in the 2015 – 2017 cohort, while patients with a well differentiated Gleason score also had a high probability of being disease-free (*figure 3*).

Of the 164 patients; patients in the 2010 – 2014 cohort, those diagnosed at stage I had a MS time of 2.4 months, while among the 2015 – 2017 cohort of patients was 3.0 months; those diagnosed at stage IV had a MS time of 3.1 months for the 2010 – 2014 cohort and 3.4 months for the 2015 – 2017 cohort of patients. The lowest MS time was recorded among those diagnosed at stage III,

1.4 months among the 2010 – 2014 cohort of patients. Meanwhile among the 190 patients in the 2015 – 2017 cohort, those diagnosed at stage II had a MS time of three and a half months; patients with lymph node metastasis had a MS time of four months with an overall MS time of 3.5 months, as those with metastasis to the rectum. Patients with metastasis to the bones had a MS time of 2.4 months among the 2010 – 2014 cohort of patients and 2.9 months for patients in the 2015 – 2017 cohort. Those with multiple metastases in the 2010 – 2014 cohort survived for a period of 4.2 months while those in the 2015 – 2017 cohort survived for three months; overall survival time for multiple metastasis was 3.4 months. Probability of being disease-free was lowest among those diagnosed at stage IV in the 2010 – 2014 cohort while it was observed to be highest in the 2015 – 2017 cohort (figure 3).

Patients who underwent surgery among the 2010 – 2014 cohort survived for a period of 1.6 months and 2.4 for those in 2015 – 2017 cohort. Overall MS time was 2.2 months, while those who received radiotherapy alone had a MS time of one and a half month. Those treated with combination of radiotherapy and surgery had a MS time of 4.6 months while those treated with chemotherapy and surgery had a MS time of more than five months in the 2010 - 2014 cohort. Among patients in the 2015 – 2017 cohort, those treated with radiotherapy and surgery survived for 6.9 months and 4.6 months for those in the 2010 – 2014 cohort, with an overall MS time of 6.9 months, those treated with chemotherapy and radiotherapy survived for 4.0 months, with an overall MS time of 3.7 months. Patients treated with chemotherapy and surgery survived for almost four and a half months in the 2015 – 2017 cohort, and 5.9 months among those in the 2010 – 2014 cohort, with an overall survival time of 4.6 months.

Probability of being disease-free was highest among those who were treated with radiotherapy and surgery in the 2015 – 2017 cohort, while it was lowest for those treated with radiotherapy alone in the 2010 – 2014 cohort of patients (Figure 3) The overall MS time for the 2015 – 2017 cohort of patients was 3.3 months meanwhile for the 2010 – 2014 cohort the MS time was 2.9 months (table 3). As shown in Figure 2, patients in the 2015 – 2017 cohort had the highest probability of being disease-free compared to patients in the 2010 – 2014 cohort. The overall MS time for the study was 3.2 months (table 3).

Table 3: Median survival time of prostate cancer patients

Characteristic	2010 – 2014		2015 - 2017		Overall	
	n	MST	n	MST	n	MST
Age						
50 – 59	15	3.5	18	3.8	33	3.5
60 – 69	44	4.2	46	2.9	90	3.6
70 – 79	69	2.7	72	3.4	141	2.9
79 – above	36	2.2	54	3.4	90	2.8
Marital Status						
Currently married	160	2.8	186	3.3	346	3.2
Widower	4	0.7	4	0.9	8	0.9
Occupation						
Civil Servant	92	2.6	89	3.0	181	2.9
Farming	3	1.8	6	2.8	9	2.8
Factory Worker	9	5.2	7	4.5	16	5.1
Unemployed	3	1.1	1	CBC	4	1.2
Others	57	3.5	87	3.3	144	3.3
Religion						
Christian	121	2.9	136	3.3	257	3.2
Islam	41	2.5	53	3.4	94	2.7
Others	2	1.8	1	CBC	3	3.7
Geographical Location						
South-west	132	2.9	146	3.3	278	3.1
South-south	18	1.5	20	4.9	38	2.6
South-east	10	3.5	20	3.3	30	3.3
North-central	4	2.5	4	14.2	8	3.7
Family History of Prostate Cancer						
Yes	35	3.5	42	2.4	77	2.8
No	129	2.9	148	3.5	277	3.1
Method of diagnosis						
PSA test	10	3.1	10	2.8	20	2.9
DRE test	90	2.9	103	3.8	193	3.5
Transrectal Ultrasound	5	2.3	4	0.6	9	1.4
Biopsy	44	2.9	54	2.9	98	2.9
DRE & Biopsy	15	2.6	19	4.2	34	2.7
Gleason group						
Well differentiated	32	1.9	40	4.7	72	2.9
Moderately differentiated	32	3.2	30	3.0	62	3.0
Poorly differentiated	100	3.5	120	3.3	220	3.3

<i>Stage at Diagnosis</i>						
Stage I	6	2.4	5	3.0	11	2.3
Stage II	6	2.1	6	3.5	12	3.3
Stage III	8	1.4	4	1.6	12	1.6
Stage IV	137	3.1	172	3.4	309	3.3
Unknown	7	3.5	3	1.5	10	3.5
<i>Site of Metastasis</i>						
Lymph node	6	2.3	12	4.3	18	3.5
Rectum (bladder neck)	22	3.1	33	4.0	55	3.5
Bones	41	2.4	43	2.9	84	2.7
Spine	56	2.6	60	3.3	116	2.9
Multiple Metastasis	39	4.2	42	3.0	81	3.4
<i>Treatment Modality</i>						
Surgery alone	29	1.6	38	2.4	67	2.2
Radiotherapy alone	22	1.5	20	3.2	42	2.6
Chemotherapy alone	28	2.9	34	2.7	62	2.9
Radiotherapy & Surgery	3	4.6	4	6.9	7	6.9
Chemotherapy & Radiotherapy	24	1.7	24	4.0	48	3.7
Chemotherapy & Surgery	56	5.9	63	4.4	119	4.6
Chemotherapy & Surgery & Radiotherapy	2	1.9	7	CBC	9	CBC
Overall	164	2.9	190	3.3	354	3.2

NB: CBC = can't be computed; Overall = 2010 – 2017

Figure 2: Survivorship curves showing the probability of survival for cohort 2010 – 2014 and 2015 – 2017

Comparison of survivor function across the selected Sociodemographic and clinical characteristics of the CaP patients

The comparison of survivor curves showed that the probability of survival was significantly different, across the different patients' age, for those in the 2010 – 2014 and 2015 – 2017 cohorts respectively. There were no differences in survival experiences among patients with characteristics such as occupation, family history, methods of diagnosis, Gleason group, stage at diagnosis, site of metastasis across 2010 - 2014 and 2015 – 2017 cohort.

Treatment received showed a significant difference in survivorship experience for patients in the 2010 – 2014 and 2015 – 2017 cohort. Overall treatment regimen received showed significant difference in survival experience for all patients (Table 4).

Table 4: Comparison of survivor function across selected demographic and clinical characteristics of CaP Patients

	2010 - 2014	2015 - 2017	Overall
Characteristics	χ^2, <i>P-value</i>*	χ^2, <i>P-value</i>*	χ^2, <i>P-value</i>*
Age	61.55, < 0.05	55.28, < 0.05	0.56,0.75
Occupation	1.72,0.78	4.59,0.33	4.37,0.36
Family History	0.92,0.33	3.23,0.07	0.22,0.64
Method of diagnosis	2.39,0.66	3.13,0.53	3.41,0.49
Gleason group	2.58,0.27	4.79,0.09	1.13,0.57
Stage at Diagnosis	3.07,0.54	3.02,0.55	5.58,0.23
Site of Metastasis	0.66,0.96	0.19,0.99	0.39,0.98
Treatment Modality	22.57,< 0.05	14.22,< 0.03	24.87,< 0.05

Overall = 2010 – 2017 *from Log rank test

Figure 3: Kaplan Meier survival curves for selected clinical characteristics

Cox proportional regression

The outcome of the bivariate regression models showed that widowers in the 2015 – 2017 cohort had a 30% increase in risk (HR = 3.3; 95%CI: 1.0 – 10.5) of being disease-free compared to patients who were currently married (Table 4).

Patients treated using a combination of chemotherapy and surgery experienced a 60%, (HR = 0.6; 95%CI: 0.4 – 1.0) and 40% (HR = 0.4; 95%CI: 0.2 – 0.7) delay in risk of being disease-free for the 2015 - 2017, and 2010 – 2014 cohorts respectively. Meanwhile, patients who received a combination of chemotherapy, surgery and radiotherapy in the overall group had a 10% delay (HR = 0.1; 95%CI: 0.1 – 0.9) in risk of being disease-free compared to patients who underwent surgery alone.

Table 4: Unadjusted determinants of prostate cancer survivorship using Cox proportional hazard regression model

Characteristic	2010 - 2014	2015 - 2017	Overall
	HR(95%CI)	HR(95%CI)	HR(95%CI)
Age			
50 - 59	1.0	1.0	1.0
60 - 69	1.1(0.5 – 2.3)	0.9(0.5 – 2.1)	1.0(0.6 – 1.7)
70 - 79	1.1(0.5 – 2.2)	1.1(0.5 – 2.1)	1.0(0.6 – 1.7)
79 - above	1.4(0.7 – 3.1)	1.0(0.5 – 2.0)	1.1(0.7 – 1.9)
Marital Status			
Currently married	1.0	1.0	1.0
Widower	1.9(0.6 – 6.1)	3.3(1.0 – 10.5)*	2.5(1.0 – 5.6)*
Occupation			
Civil Servant	1.0	1.0	1.0
Farming	1.2(0.3 – 5.2)	1.3(0.5 – 3.1)	1.3(0.6 – 2.7)
Factory Worker	0.6(0.3 – 1.4)	0.3(0.1 – 1.4)	0.5(0.2 – 1.0)
Unemployed	1.2(0.3 – 5.0)	0.6(0.1 – 4.3)	0.9(0.3 – 2.8)
Others	1.0(0.7 – 1.5)	0.8(0.5 – 1.1)	0.9(0.7 – 1.2)
Religion			
Christian	1.0	1.0	1.0
Islam	1.5(1.0 – 2.4)	0.9(0.6 – 1.3)	1.2(0.9 – 1.5)
Others	1.7(0.4 – 7.1)	CBC	0.9(0.2 – 3.8)
Geographical Location			
South-west	1.0	1.0	1.0

South-south	1.3(0.8 – 2.4)	0.8(0.4 – 1.3)	1.0(0.7 – 1.5)
South-east	0.7(0.3 – 1.7)	1.2(0.6 – 2.2)	0.9(0.6 – 1.6)
North-central	0.9(0.3 – 3.1)	0.1(0.0 – 1.0)	0.5(0.2 – 1.3)

Family History of Prostate Cancer

Yes	1.0	1.0	1.0
No	1.3(0.8 – 2.0)	0.7(0.4 – 1.0)	0.9(0.8 – 1.3)

Method of diagnosis

PSA test	1.0	1.0	1.0
DRE test	0.8(0.4 – 1.7)	0.8(0.3 – 1.9)	0.8(0.4 – 1.4)
Transrectal Ultrasound	1.2(0.3 – 4.8)	1.4(0.3 – 5.9)	1.3(0.5 – 3.5)
Biopsy	0.8(0.4 – 1.9)	1.0(0.4 – 2.6)	0.9(0.5 – 1.7)
DRE & Biopsy	1.2(0.5 – 3.0)	0.7(0.2 – 2.2)	0.9(0.4 – 1.9)

Gleason group

Well differentiated	1.0	1.0	1.0
Moderately differentiated	0.7(0.4 – 1.2)	1.9(1.1 – 3.4)	1.2(0.8 – 1.8)
Poorly differentiated	0.7(0.4 – 1.1)	1.3(0.8 – 2.1)	0.9(0.7 – 1.4)

PSA level	1.0(0.9 – 1.0)	1.0(0.1 – 1.0)	1.0(0.9 – 1.0)
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Stage at Diagnosis

Stage I	1.0	1.0	1.0
Stage II	0.9(0.3 – 3.5)	0.7(0.1 – 3.9)	0.9(0.3 – 2.6)
Stage III	2.1(0.6 – 6.9)	1.3(0.2 – 7.8)	1.9(0.7 – 5.2)
Stage IV	1.0(0.4 – 2.6)	0.6(1.5 – 2.5)	0.9(0.4 – 1.9)
Unknown	1.3(0.4 – 4.2)	1.0(0.2 – 6.2)	1.2(0.5 – 3.3)

Site of Metastasis

Lymph node	1.0	1.0	1.0
Rectum (bladder neck)	0.8(0.3 – 2.4)	1.2(0.5 – 2.9)	1.0(0.5 – 2.0)
Bones	0.9(0.4 – 2.5)	1.2(0.5 – 2.8)	1.1(0.6 – 2.1)
Spine	1.1(0.4 – 2.8)	1.1(0.5 – 2.6)	1.1(0.6 – 2.1)
Multiple Metastasis	0.9(0.4 – 2.5)	1.2(0.5 – 2.9)	1.1(0.7 – 2.1)

Treatment Modality

Surgery alone	1.0	1.0	1.0
Radiotherapy alone	1.3(0.7 – 2.2)	0.7(0.3 – 1.3)	0.9(0.6 – 1.4)
Chemotherapy alone	0.8(0.4 – 1.2)	1.1(0.7 – 1.9)	0.9(0.6 – 1.3)
Radiotherapy & Surgery	0.4(0.1 – 1.7)	0.4(0.1 – 1.6)	0.4(0.2 – 1.1)
Chemotherapy & Radiotherapy	0.5(0.3 – 1.1)	0.7(0.3 – 1.2)	0.6(0.4 – 0.9)*
Chemotherapy & Surgery	0.4(0.2 – 0.7)*	0.6(0.4 – 1.0)*	0.5(0.3 – 0.7)*

Chemotherapy & Surgery & Radiotherapy	1.4(0.2 – 10.5)	3.9(0 - CBC)	0.1(0.1 – 0.9)*
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CBC = can't be computed; Overall = 2010 - 2017 CI Confidence Interval

While controlling for other variables patients who were widowers in the 2015 – 2017 cohort experienced being disease-free earlier (aHR = 5.7; 95%CI: 1.4 – 23.1) compared to patients who were currently married. Meanwhile, patients with no family history of CaP in the same cohort experienced a 40% delay in risk of being disease-free (aHR = 0.6; 95%CI: 0.3 – 1.0) compared to patients with a positive family history of CaP. Patients with a poorly differentiated Gleason score in the 2010 – 2014 cohort were disease-free later (aHR = 0.5; 95%CI: 0.3 – 0.9) compared to patients with a well-differentiated Gleason score. Patients treated using a combination of chemotherapy and surgery had a 80% delay in risk of being disease-free (aHR = 0.2; 95%CI: 0.1 – 0.4) (table 5).

Table 5: Adjusted determinants of prostate cancer survivorship using Cox proportional hazard regression model

Characteristic	2010 – 2014 aHR(95%CI)	2015 - 2017 aHR(95%CI)	Overall aHR(95%CI)
Age			
50 – 59	1.0	1.0	1.0
60 – 69	0.7(0.3 – 1.9)	1.2(0.5 – 2.9)	1.2(0.7 – 2.3)
70 – 79	0.8(0.4 – 2.1)	0.9(0.4 – 1.7)	1.1(0.7 – 1.9)
79 – above	0.7(0.2 – 1.8)	0.7(0.3 – 1.7)	1.2(0.7 – 2.2)
Marital Status			
Currently married	1.0	1.0	1.0
Widower	4.4(1.1 – 16.5)*	5.7(1.4 – 23.1)*	3.7(1.4 – 8.1)*
Occupation			
Civil Servant	1.0	1.0	1.0
Farming	1.3(0.3 – 7.1)	1.1(0.3 – 3.6)	1.1(0.5 – 2.7)
Factory Worker	0.4(0.1 – 1.2)	0.3(0.1 – 1.4)	0.5(0.2 – 1.1)
Unemployed	4.1(0.8 – 21.2)	1.0(0.1 – 9.2)	1.2(0.3 – 3.9)
Others	1.4(0.8 – 2.5)	0.8(0.5 – 1.2)	0.9(0.7 – 1.4)
Religion			
Christian	1.0	1.0	1.0
Islam	1.4(0.8 – 2.3)	1.1(0.7 – 1.9)	1.2(0.8 – 1.6)
Others	2.9(0.5 – 16.9)	CBC	1.2(0.3 – 5.2)

Geographical Location

South-west	1.0	1.0	1.0
South-south	1.7(0.9 – 3.4)	0.6(0.3 – 1.2)	1.0(0.7 – 1.6)
South-east	0.7(0.2 – 1.9)	0.9(0.4 – 2.4)	1.1(0.6 – 1.9)
North-central	1.5(0.3 – 7.7)	0.1(0.0 – 1.2)	0.4(0.1 – 1.3)

Family History of Prostate Cancer

Yes	1.0	1.0	1.0
No	1.3(0.7 – 2.3)	0.6(0.3 – 1.0)*	0.9(0.7 – 1.4)

Method of diagnosis

PSA test	1.0	1.0	1.0
DRE test	1.1(0.4 – 3.1)	0.7(0.2 – 2.2)	0.7(0.4 – 1.3)
Transrectal Ultrasound	2.2(0.4 – 11.3)	0.9(0.1 – 6.9)	1.2(0.4 – 3.5)
Biopsy	1.6(0.5 – 4.9)	0.9(0.3 – 3.4)	0.9(0.5 – 1.7)
DRE & Biopsy	1.8(0.5 – 6.2)	0.5(0.1 – 2.0)	0.8(0.3 – 1.7)

Gleason group

Well differentiated	1.0	1.0	1.0
Moderately differentiated	0.6(0.3 – 1.2)	1.9(0.9 – 3.9)	1.3(0.8 – 1.9)
Poorly differentiated	0.5(0.3 – 0.9)*	1.4(0.8 – 2.6)	0.9(0.7 – 1.4)

PSA level	1.0(0.9 – 1.0)	0.9(0.9 – 1.0)	1.0(0.9 – 1.0)
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Stage at Diagnosis

Stage I	1.0	1.0	1.0
Stage II	2.4(0.5 – 12.9)	1.1(0.1 – 8.4)	1.5(0.5 – 4.6)
Stage III	3.6(0.9 – 13.6)	2.1(0.2 – 21.2)	2.8(0.9 – 8.3)
Stage IV	2.1(0.7 – 6.3)	0.8(0.1 – 4.9)	1.2(0.5 – 2.7)
Unknown	2.9(0.6 – 13.1)	1.2(0.1 – 12.6)	1.6(0.5 – 4.8)

Site of Metastasis

Lymph node	1.0	1.0	1.0
Rectum (bladder neck)	0.9(0.3 – 3.7)	0.9(0.3 – 2.8)	1.2(0.6 – 2.4)
Bones	1.0(0.3 – 3.5)	0.6(0.2 – 1.7)	1.1(0.6 – 2.3)
Spine	1.4(0.4 – 4.6)	0.6(0.2 – 1.9)	1.2(0.6 – 2.3)
Multiple Metastasis	0.8(0.3 – 2.9)	0.7(0.2 – 2.2)	1.1(0.5 – 2.2)

Treatment Modality

Surgery	1.0	1.0	1.0
Radiotherapy	1.5(0.7 – 3.0)	0.9(0.4 – 2.2)	1.1(0.7 – 1.8)
Chemotherapy	0.5(0.2 – 1.0)	1.1(0.5 – 2.1)	0.8(0.6 – 1.3)
Radiotherapy & Surgery	0.2(0.0 – 1.9)	0.7(0.1 – 4.5)	0.4(0.1 – 1.4)

Chemotherapy & Radiotherapy	0.6(0.2 – 1.4)	0.6(0.3 – 1.4)	0.5(0.3 – 0.9)
Chemotherapy & Surgery	0.2(0.1 – 0.4)*	0.9(0.3 – 1.6)	0.5(0.3 – 0.7)*
Chemotherapy & Surgery & Radiotherapy	2.2(0.2 – 25.8)	CBC	0.1(0.0 – 1.0)

NB: CBC = can't be computed; Overall = 2010 – 2017

Parametric survival analysis (Weibull distribution)

Result from the bivariate Weibull regression model showed that patients who were widowers in the 2015 – 2017 cohort experienced a significant increase of 70% in time to being disease-free (TR = 0.3; 95% CI: 0.1 – 0.9) compared to patients currently married (Table 6).

Patients with a moderately differentiated Gleason score experienced a significant increase of 50% in time to being disease-free (TR = 0.5; 95%CI: 0.3 – 0.9) for those in the 2015 – 2017 cohort.

Being treated by chemotherapy and radiotherapy significantly decreased time to being disease-free by 60% (TR = 1.6; 95%CI: 1.0 – 2.6) for the overall group, compared to patients who underwent surgery. In like manner patients who received chemotherapy and surgery had a significant delay in time to being disease-free (TR = 2.4; 95%CI: 1.4 – 3.9) for the 2010 – 2014 cohort, also for the overall group, time to being disease-free was delayed (TR = 1.9; 95%CI: 1.4 – 2.9) compared to patients who underwent surgery. Patients who received a combination of chemotherapy, surgery and radiotherapy also experienced a delay in time to being disease-free (TR = 7.0; 95%CI: 1.0 – 49.2) compared to patients who underwent surgery (Table 6).

Table 6: Unadjusted determinants of prostate cancer survivorship using Weibull Parametric Regression

Characteristic	2010 - 2014	2015 - 2017	Overall
	TR(95%CI)	TR(95%CI)	TR(95%CI)
<i>Age</i>			
50 – 59	1.0	1.0	1.0
60 – 69	0.9(0.4 – 1.9)	1.1(0.5 – 2.3)	1.0(0.6 – 1.7)
70 – 79	0.9(0.5 – 1.8)	1.0(0.5 – 1.9)	0.9(0.6 – 1.6)
79 – above	0.7(0.3 – 1.5)	1.1(0.6 – 2.2)	0.9(0.6 – 1.5)
<i>Marital Status</i>			
Currently married	1.0	1.0	1.0
Widower	0.5(0.2 – 1.6)	0.3(0.1 – 0.9)*	0.4(0.2 – 0.9)*

<i>Occupation</i>			
Civil Servant	1.0	1.0	1.0
Farming	0.8(0.2 – 2.9)	0.8(0.3 – 2.0)	0.8(0.3 – 1.7)
Factory Worker	1.6(0.7 – 3.7)	2.8(0.7 – 11.5)	1.9(0.9 – 3.9)
Unemployed	0.9(0.2 – 3.3)	1.7(0.2 – 11.9)	1.1(0.4 – 3.5)
Others	0.9(0.7 – 1.4)	1.3(0.9 – 1.8)	1.1(0.9 – 1.5)
<i>Religion</i>			
Christian	1.0	1.0	1.0
Islam	0.6(0.4 – 0.9)	1.1(0.7 – 1.7)	0.9(0.6 – 1.1)
Others	0.6(0.1 – 2.1)	CBC	1.1(0.3 – 4.2)
<i>Geographical Location</i>			
South-west	1.0	1.0	1.0
South-south	0.7(0.4 – 1.3)	1.3(0.7 – 2.2)	0.9(0.7 – 1.5)
South-east	1.4(0.6 – 3.5)	0.9(0.5 – 1.5)	1.0(0.6 – 1.7)
North-central	1.0(0.3 – 3.2)	2.9(0.7 – 11.6)	1.8(0.8 – 4.3)
<i>Family History of Prostate Cancer</i>			
Yes	1.0	1.0	1.0
No	0.8(0.5 – 1.2)	1.4(0.9 – 2.2)	1.1(0.8 – 1.4)
<i>Method of diagnosis</i>			
PSA test	1.0	1.0	1.0
DRE test	1.3(0.6 – 2.7)	1.3(0.5 – 3.2)	1.3(0.7 – 2.3)
Transrectal Ultrasound	0.8(0.2 – 2.9)	0.7(0.2 – 2.9)	0.8(0.3 – 1.9)
Biopsy	1.2(0.5 – 2.6)	0.9(0.4 – 2.5)	1.1(0.6 – 1.9)
DRE & Biopsy	0.8(0.3 – 2.0)	1.5(0.5 – 4.5)	1.1(0.5 – 2.1)
<i>Gleason group</i>			
Well differentiated	1.0	1.0	1.0
Moderately differentiated	1.4(0.8 – 2.5)	0.5(0.3 – 0.9)*	0.8(0.6 – 1.3)
Poorly differentiated	1.4(0.8 – 2.2)	0.8(0.5 – 1.2)	1.0(0.7 – 1.4)
PSA level	0.9(0.9 – 1.0)	1.0(0.9 – 1.0)	0.9(0.9 – 1.0)
<i>Stage at Diagnosis</i>			
Stage I	1.0	1.0	1.0
Stage II	1.1(0.3 – 3.9)	1.4(0.3 – 7.5)	1.1(0.4 – 3.1)
Stage III	0.5(0.2 – 1.6)	0.8(0.1 – 4.4)	0.5(0.2 – 1.4)
Stage IV	0.9(0.4 – 2.3)	1.6(0.4 – 6.2)	1.1(0.5 – 2.3)
Unknown	0.8(0.3 – 2.5)	0.8(0.2 – 5.2)	0.8(0.3 – 2.2)
<i>Site of Metastasis</i>			

Lymph node	1.0	1.0	1.0
Rectum (bladder neck)	1.1(0.4 – 3.0)	0.9(0.3 – 2.1)	0.9(0.5 – 1.9)
Bones	1.0(0.4 – 2.5)	0.9(0.4 – 2.1)	0.9(0.5 – 1.7)
Spine	0.9(0.4 – 2.2)	0.9(0.4 – 2.1)	0.9(0.5 – 1.6)
Multiple Metastasis	1.0(0.4 – 2.6)	0.8(0.3 – 2.1)	0.9(0.5 – 1.8)
<i>Treatment Modality</i>			
Surgery	1.0	1.0	1.0
Radiotherapy	0.8(0.5 – 1.4)	1.5(0.8 – 2.7)	1.1(0.7 – 1.6)
Chemotherapy	1.4(0.9 – 2.5)	0.9(0.6 – 1.6)	1.2(0.8 – 1.7)
Radiotherapy & Surgery	2.4(0.6 – 8.9)	2.3(0.6 – 9.3)	2.3(0.9 – 6.2)
Chemotherapy & Radiotherapy	1.7(0.9 – 3.6)	1.5(0.8 – 2.8)	1.6(1.0 – 2.6)*
Chemotherapy & Surgery	2.4(1.4 – 3.9)*	1.6(0.9 – 2.7)	1.9(1.4 – 2.9)*
Chemotherapy & Surgery & Radiotherapy	0.8(0.1 – 4.3)	0.4(0 - CBC)	7.0(1.0 – 49.2)*

CBC = can't be computed; Overall = 2010 – 2017; TR Time Ratio

Result from the multivariate analysis showed that patients who were widowers experienced a 80% increase in time to being disease-free in the 2015 – 2017 cohort (aTR = 0.2; 95% CI: 0.1 – 0.7) and also a 70% increase in the 2010 – 2014 cohort and overall group (aTR = 0.3; 95% CI: 0.1 – 0.8) and (aTR = 0.3; 95% CI: 0.1 – 0.7) compared to patients who are currently married (Table 7).

Time to being disease-free significantly decreased by 70% for patients with a poorly differentiated Gleason score in the 2010 – 2014 cohort (aTR = 1.7; 95% CI: 1.0 – 2.7) compared to patients with a well-differentiated gleason score. Patients diagnosed at stage III also in the same cohort experienced a significant increase of 60% in time to being disease-free (aTR = 0.4; 95% CI: 0.1 – 1.0) compared to patients diagnosed at stage I. Patients in the 2010 – 2014 cohort who were treated using a combination of chemotherapy and surgery experienced a 220% delay in time to being disease-free (aTR = 3.2; 95% CI: 1.9 – 5.3). Those treated using a combination chemotherapy and radiotherapy in the overall group also had a significant delay of 80% in time to being disease-free (aTR = 1.8; 95% CI: 1.1 – 3.1). Likewise, time to being disease-free significantly decreased by 90% for patients treated using a combination of chemotherapy and surgery in the overall group respectively (aTR = 1.9; 95% CI: 1.4 – 2.8) compared to patients who underwent surgery alone (Table 4.7).

Table 7: Adjusted determinants of prostate cancer survivorship using Weibull Parametric Regression model

Characteristic	2010 – 2014	2015 - 2017	Overall
	aTR(95%CI)	aTR(95%CI)	aTR(95%CI)
<i>Age</i>			
50 – 59	1.0	1.0	1.0
60 – 69	1.2(0.6 – 2.6)	0.9(0.4 – 2.0)	0.8(0.5 – 1.5)
70 – 79	1.0(0.5 – 2.1)	1.1(0.6 – 2.2)	0.9(0.5 – 1.4)
79 – above	1.3(0.6 – 3.1)	1.3(0.7 – 2.8)	0.8(0.5 – 1.4)
<i>Marital Status</i>			
Currently married	1.0	1.0	1.0
Widower	0.3(0.1 – 0.8)*	0.2(0.1 – 0.7)*	0.3(0.1 – 0.7)*
<i>Occupation</i>			
Civil Servant	1.0	1.0	1.0
Farming	0.8(0.2 – 2.9)	1.0(0.3 – 2.9)	0.9(0.4 – 2.0)
Factory Worker	2.1(0.9 – 4.8)	2.7(0.7 – 10.4)	1.9(0.9 – 3.8)
Unemployed	0.4(0.1 – 1.4)	0.9(0.1 – 7.1)	0.9(0.3 – 2.9)
Others	0.8(0.5 – 1.2)	1.2(0.8 – 1.9)	1.0(0.8 – 1.4)
<i>Religion</i>			
Christian	1.0	1.0	1.0
Islam	0.8(0.5 – 1.2)	0.9(0.5 – 1.4)	0.9(0.6 – 1.1)
Others	0.4(0.1 – 1.7)	CBC	0.9(0.2 – 3.4)
<i>Geographical Location</i>			
South-west	1.0	1.0	1.0
South-south	0.7(0.4 – 1.1)	1.4(0.8 – 2.6)	0.9(0.7 – 1.5)
South-east	1.3(0.6 – 2.9)	1.0(0.4 – 2.3)	0.9(0.5 – 1.6)
North-central	0.7(0.2 – 2.7)	3.3(0.7 – 14.9)	2.2(0.8 – 5.8)
<i>Family History of Prostate Cancer</i>			
Yes	1.0	1.0	1.0
No	0.8(0.5 – 1.3)	1.5(0.9 – 2.4)	1.0(0.7 – 1.4)
<i>Method of diagnosis</i>			
PSA test	1.0	1.0	1.0
DRE test	0.9(0.4 – 2.2)	1.4(0.5 – 3.9)	1.4(0.8 – 2.6)
Transrectal Ultrasound	0.5(0.1 – 1.9)	0.9(0.2 – 5.3))	0.8(0.3 – 2.3)
Biopsy	0.7(0.3 – 1.6)	0.9(0.3 – 2.8)	1.1(0.5 – 2.1)
DRE & Biopsy	0.6(0.2 – 1.7)	1.9(0.6 – 7.2)	1.3(0.6 – 2.8)
<i>Gleason group</i>			
Well differentiated	1.0	1.0	1.0

Moderately differentiated	1.4(0.8 – 2.5)	0.6(0.3 – 1.1)	0.8(0.5 – 1.2)
Poorly differentiated	1.7(1.0 – 2.7)*	0.7(0.4 – 1.2)	1.0(0.7 – 1.4)
PSA level	0.9(0.9 – 1.0)	1.0(0.9 – 1.0)	0.9(0.9 – 1.0)
<i>Stage at Diagnosis</i>			
Stage I	1.0	1.0	1.0
Stage II	0.5(0.1 – 1.9)	0.9(0.1 – 5.6)	0.7(0.3 – 1.9)
Stage III	0.4(0.1 – 1.0)*	0.5(0.1 – 3.7)	0.4(0.1 – 1.1)
Stage IV	0.5(0.2 – 1.3)	1.2(0.2 – 5.9)	0.8(0.4 – 1.8)
Unknown	0.5(0.1 – 1.5)	1.3(0.5 – 3.6)	0.7(0.2 – 1.9)
<i>Site of Metastasis</i>			
Lymph node	1.0	1.0	1.0
Rectum (bladder neck)	0.9(0.3 – 2.6)	1.1(0.4 – 3.0)	0.9(0.4 – 1.7)
Bones	0.9(0.4 – 2.5)	1.5(0.6 – 4.1)	0.9(0.5 – 1.7)
Spine	0.7(0.3 – 1.9)	1.3(0.5 – 3.5)	0.9(0.5 – 1.6)
Multiple Metastasis	1.1(0.4 – 2.9)	1.3(0.5 – 3.6)	0.9(0.5 – 1.8)
<i>Treatment Modality</i>			
Surgery	1.0	1.0	1.0
Radiotherapy	0.7(0.4 – 1.3)	0.9(0.5 – 2.1)	0.9(0.6 – 1.4)
Chemotherapy	1.7(0.9 – 3.0)	0.9(0.5 – 1.8)	1.2(0.8 – 1.7)
Radiotherapy & Surgery	3.4(0.7 – 17.2)	1.3(0.2 – 7.5)	2.2(0.8 – 6.7)
Chemotherapy & Radiotherapy	1.6(0.7 – 3.2)	1.6(0.7 – 3.4)	1.8(1.1 – 3.1)*
Chemotherapy & Surgery	3.2(1.9 – 5.3)*	1.2(0.7 – 2.0)	1.9(1.4 – 2.8)*
Chemotherapy & Surgery & Radiotherapy	0.5(0.1 – 3.6)	CBC	6.2(0.9 – 40.4)
_cons	6.3(1.5 – 27.4)	1.7(0.2 – 17.9)	4.2(1.2 – 14.4)
/ln_p	0.2(0.1 – 0.4)	0.1(-0.1 – 0.2)	0.1(-0.0 – 0.2)
P	1.2(1.1 – 1.5)	1.1(0.9 – 1.3)	1.1(0.9 – 1.2)
1/p	0.8(0.7 – 0.9)	0.9(0.8 – 1.1)	0.9(0.8 – 1.0)

CBC = can't be computed; Overall = 2010 – 2017; aTR = adjusted Time Ratio

Bayesian survival analysis (Weibull distribution)

Results from the bivariate Bayesian parametric (Weibull) regression model showed that men who were factory workers experienced a 20% delay in time to being disease-free (TR = 2.2; 95% Credible Interval (CrI: 1.1 – 4.3)) compared to individuals who are civil servants (Table 8).

Patients with a moderately differentiated Gleason score in the 2015 – 2017 cohort experienced a significant increase in time to being disease-free by 60% (TR =0.6; 95% CrI: 0.3 – 0.9) compared to patients with a well-differentiated Gleason score (table 8).

Patients treated using a combination of radiotherapy and surgery in the overall group experienced a significant delay of 90% in time to being disease-free (TR = 2.9; 95% CrI: 1.1 – 7.5) compared to patients who underwent surgery alone. Meanwhile, treatment via chemotherapy and surgery for both the 2010 – 2014 cohort significantly delayed time to being disease-free (TR = 2.0; 95% CrI: 1.4 – 4.2); compared to patients who underwent surgery alone. Patients who received all three combinations of chemotherapy, surgery and radiotherapy experienced delay in time to being disease-free for the 2015 – 2017 cohort and the overall group, compared to patients who underwent surgery alone (Table 8).

Table 8: Unadjusted determinants of prostate cancer survivorship using Bayesian Parametric (Weibull distribution) Regression model

Characteristic	2010 – 2014	2015 - 2017	Overall
	TR(95%CrI)	TR(95%CrI)	TR(95%CrI)
<i>Age</i>			
50 – 59	1.0	1.0	1.0
60 – 69	0.9(0.4 – 1.7)	1.1(0.5 – 2.2)	0.9(0.5 – 1.5)
70 - 79	0.9(0.4 – 1.7)	1.0(0.5 – 1.9)	0.9(0.6 – 1.4)
79 - above	0.7(0.3 – 1.4)	1.1(0.5 – 2.1)	0.9(0.5 – 1.4)
<i>Marital Status</i>			
Married	1.0	1.0	1.0
Widower	0.8(0.2 – 2.5)	0.5(0.1 – 1.5)	0.5(0.2 – 1.1)
<i>Occupation</i>			
Civil Servant	1.0	1.0	1.0
Farming	1.5(0.3 – 6.9)	1.1(0.4 – 3.0)	0.9(0.4 – 2.1)
Factory Worker	1.9(0.8 – 4.8)	6.7(0.9 – 28.6)	2.2(1.1 – 4.3)*
Unemployed	1.7(0.3 – 6.9)	13.9(0.5 – 103.1)	1.7(0.4 – 5.8)
Others	0.9(0.7 – 1.5)	1.3(0.8 – 1.8)	1.1(0.8 – 1.5)
<i>Religion</i>			
Christian	1.0	1.0	1.0
Islam	0.7(0.4 – 1.0)	1.4(0.7 – 1.8)	0.9(0.6 – 1.2)
Others	1.2(0.1 – 4.9)	1.2(4.6 – 3.7)*	2.0(0.4 – 8.9)
<i>Geographical Location</i>			

South-west	1.0	1.0	1.0
South-south	0.8(0.5 – 1.5)	1.3(0.7 – 2.4)	1.0(0.7 – 1.5)
South-east	1.8(0.7 – 4.6)	0.9(0.5 – 1.7)	1.1(0.7 – 1.8)
North-central	1.6(0.4 – 4.9)	5.3(1.0 – 21.2)	2.2(0.9 – 5.6)
<i>Family History of Prostate Cancer</i>			
Yes	1.0	1.0	1.0
No	0.8(0.5 – 1.2)	1.4(0.8 – 2.1)	1.0(0.7 – 1.4)
<i>Method of diagnosis</i>			
PSA test	1.0	1.0	1.0
DRE test	1.3(0.5 – 2.4)	1.3(0.4 – 2.8)	1.3(0.6 – 2.2)
Transrectal Ultrasound	1.2(0.3 – 4.2)	1.1(0.2 – 4.0)	0.9(0.3 – 2.2)
Biopsy	1.2(0.5 – 2.4)	0.9(0.3 – 2.2)	1.1(0.5 – 1.9)
DRE & Biopsy	0.9(0.3 – 1.9)	1.7(0.4 – 4.8)	1.1(0.5 – 2.1)
<i>Gleason group</i>			
Well differentiated	1.0	1.0	1.0
Moderately differentiated	1.5(0.8 – 2.6)	0.6(0.3 – 0.9)*	0.9(0.7 – 1.3)
Poorly differentiated	1.4(0.9 – 2.2)	0.8(0.5 – 1.2)	1.0(0.7 – 1.4)
PSA level	1.0(0.9 – 1.0)	1.0(0.9 – 1.0)	1.0(0.9 – 1.0)
<i>Stage at Diagnosis</i>			
Stage I	1.0	1.0	1.0
Stage II	0.6(0.1 – 1.6)	2.0(0.1 – 5.6)	1.3(0.4 – 3.1)
Stage III	0.9(0.3 – 2.1)	1.6(0.1 – 4.7)	0.6(0.2 – 1.3)
Stage IV	0.9(0.2 – 2.6)	1.4(0.1 – 6.1)	1.1(0.5 – 2.1)
Unknown	CBC	CBC	0.9(0.3 – 2.3)
<i>Site of Metastasis</i>			
Lymph node	1.0	1.0	1.0
Rectum (bladder neck)	1.2(0.3 – 2.9)	0.9(0.3 – 1.9)	1.0(0.5 – 1.8)
Bones	1.0(0.3 – 2.3)	0.9(0.3 – 1.9)	0.9(0.4 – 1.7)
Spine	0.9(0.3 – 2.0)	0.9(0.3 – 1.9)	0.9(0.4 – 1.6)
Multiple Metastasis	1.1(0.3 – 2.4)	0.9(0.3 – 1.9)	0.9(0.4 – 1.7)
<i>Treatment Modality</i>			
Surgery	1.0	1.0	1.0
Radiotherapy	0.8(0.5 – 1.3)	1.6(0.8 – 3.2)	1.1(0.7 – 1.7)
Chemotherapy	1.5(0.9 – 2.5)	0.9(0.5 – 1.7)	1.2(0.8 – 1.7)
Radiotherapy & Surgery	3.5(0.8 – 11.1)	5.7(0.8 – 26.9)	2.9(1.1 – 7.5)*
Chemotherapy & Radiotherapy	2.0(0.9 – 3.8)	1.8(0.8 – 3.8)	1.7(1.0 – 2.8)
Chemotherapy & Surgery	2.0(1.4 – 4.2)*	1.8(0.9 – 3.2)	2.0(1.4 – 2.9)*

Chemotherapy & Surgery & Radiotherapy	4.5(0.2 – 20.4)	4.2(18.3 – 1.6)*	40.4(1.7 – 291.6)*
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NB: CBC = can't be computed; Overall = 2010 - 2017; CrI = Credible Interval

While adjusting for other variables, patients age 60 – 69 years and 79 years and above in the 2010 – 2014 cohort experienced a significant delay of 30% and 40% respectively in time to being disease-free (aTR = 1.3; 95%CrI: 1.1 – 1.5); (aTR = 1.4; 95%CrI: 1.1 – 1.8). Widowers experienced a significant increase of 80%, 70% and 80% in time to being disease-free for the 2010 - 2014, and 2015 – 2017 cohorts and overall group (aTR = 0.3; 95%CrI: 0.2 – 0.3), (aTR = 0.2; 95%CrI: 0.1 – 0.3), and (aTR = 0.3; 95%CrI: 0.2 – 0.4) compared to patients who were currently married. Time to being disease-free more than delayed for patients who were factory workers for the 2015 – 2017 cohort (aTR = 2.9; 95%CrI: 1.7 – 4.7) meanwhile patients who were unemployed experienced a 40% increase in time to being disease-free for the 2010 – 2014 cohort (TR = 0.4; 95%CrI: 0.4 – 0.4) compared to patients who were civil servants. Patients from the south-south and north-central experienced 40% and 230% delay in time to being disease-free respectively for the 2015 – 2017 cohort (TR = 1.4; 95% CrI: 1.1 – 1.7), (TR = 3.3; 95% CrI: 2.1 – 4.9) compared to patients from the south-west. However, for the 2010 – 2014 cohort, patients from north-central experienced a significant increase of 30% in time to being disease-free (aTR = 0.7; 95% CrI: 0.6 – 0.8) compared to patients from the south-west (Table 9).

Patients with no family history of CaP experienced a significant delay of 50% in time to being disease-free for the 2015 – 2017 cohort, compared to patients with a positive family history of CaP (aTR = 1.5; 95% CrI: 1.2 – 2.0). Patients diagnosed by a combination of DRE and biopsy experienced a delay of 10% in time to being disease-free (aTR = 2.1; 95% CrI: 1.5 – 2.7) for the 2015 – 2017 cohort compared to patients diagnosed via PSA test. Meanwhile, for the 2010 – 2014 cohort, patients diagnosed via transrectal ultrasound, biopsy and a combination of DRE and Biopsy experienced 50%, 70% and 60% experienced being disease-free earlier, compared to patients who were diagnosed via PSA test.

Patients with a moderately differentiated Gleason score for the 2010 – 2014 cohort experienced a 40% decrease in time to being disease-free (aTR = 1.4; 95% CrI: 1.3 – 1.6), similarly patients with a poorly differentiated Gleason score experienced 70% delay in time to being disease-free (aTR =

1.7; 95% CrI: 1.3 – 2.2) compared to patients with a well-differentiated Gleason score. Patients diagnosed at stage III for the 2015 – 2017 cohort time to being disease-free increased by 50% (aTR = 0.5; 95% CrI: 0.3 – 0.8) compared to patients diagnosed at stage I. Meanwhile patients diagnosed at stage II, III and IV experienced 50%, 40%, and 50% increase in time to being disease-free compared to patients diagnosed at stage I. patients with metastasis to the bones and spine in the 2015 – 2017 cohort experienced 50% and 30% delay in their time to being disease-free (aTR = 1.5; 95%CrI: 1.1 – 2.1) and (aTR = 1.3; 95%CrI: 1.1 – 1.6) while patients in the 2010 – 2014 cohort and overall group experienced 30% increase in time to being disease-free respectively (aTR = 0.7; 95%CrI: 0.6 – 0.9) compared to patients with metastasis to the lymph node.

Patients who were treated using a combination of chemotherapy and radiotherapy experienced a 70% delay in time to being disease-free compared to patients who underwent surgery for the 2015 – 2017 cohort (aTR = 1.7; 95%CrI: 1.2 – 2.3). Patients who received radiotherapy alone experienced a 70% increase in time to being disease-free (aTR = 0.7; 95%CrI: 0.7 – 0.8) for the 2010 – 2014 cohort compared to patients who underwent surgery. However, individuals who were treated using chemotherapy, radiotherapy and surgery, chemotherapy and radiotherapy, chemotherapy and surgery experienced 20%, 20%, 60% and 30% delay in time to being disease-free for the 2010 – 2014 cohort compared to patients who underwent surgery alone (table 9).

Table 9: Adjusted determinants of prostate cancer survivorship using Bayesian Parametric (Weibull Distribution) Regression model

Characteristic	2010 – 2014	2015 - 2017	Overall
	aTR(95%CrI)	aTR(95%CrI)	aTR(95%CrI)
Age			
50 - 59	1.0	1.0	1.0
60 - 69	*1.3(1.1 – 1.5)	1.3(0.4 – 2.8)	0.9(0.8 – 1.2)
70 – 79	1.1(0.8 – 1.4)	1.3(0.5 – 2.2)	0.9(0.8 – 1.2)
79 - above	*1.4(1.1 – 1.8)	1.9(0.7 – 3.5)	0.9(0.7 – 1.3)
Marital Status			
Married	1.0	1.0	1.0
Widower	0.3(0.2 – 0.3)*	0.2(0.1 – 0.3)*	0.3(0.2 – 0.4)*
Occupation			
Civil Servant	1.0	1.0	1.0
Farming	0.8(0.6 – 1.1)	1.0(0.8 – 1.4)	1.1(0.5 – 1.8)
Factory Worker	2.1(1.3 – 3.2)*	2.9(1.7 – 4.7)*	1.9(1.0 – 3.1)

Unemployed	0.4(0.4 – 0.4)*	1.0(0.7 – 1.4)	0.6(0.4 – 1.6)
Others	0.8(0.7 – 0.8)*	1.2(0.9 – 1.5)	0.9(0.7 – 1.3)
<i>Religion</i>			
Christian	1.0	1.0	1.0
Islam	0.8(0.7 – 0.9)*	0.8(0.7 – 1.2)	0.9(0.7 – 1.3)
Others	0.4(0.3 – 0.6)	CBC	1.2(0.8 – 1.7)
<i>Geographical Location</i>			
South-west	1.0	1.0	1.0
South-south	0.7(0.5 – 1.1)	1.4(1.1 – 1.7)*	0.9(0.7 – 1.3)
South-east	1.3(0.9 – 1.7)	1.0(0.9 – 1.3)	1.0(0.7 – 1.6)
North-central	0.7(0.6 – 0.8)*	3.3(2.1 – 4.9)*	2.6(1.8 – 3.5)
<i>Family History of Prostate Cancer</i>			
Yes	1.0	1.0	1.0
No	0.8(0.7 – 0.9)*	1.5(1.2 – 2.0)*	1.1(0.7 – 1.4)
<i>Method of diagnosis</i>			
PSA test	1.0	1.0	1.0
DRE test	0.9(0.9 – 1.0)	1.4(0.9 – 1.9)	1.4(1.0 – 1.9)
Transrectal Ultrasound	0.5(0.4 – 0.7)*	0.9(0.6 – 1.4)	0.8(0.4 – 1.4)
Biopsy	0.7(0.6 – 0.8)*	0.9(0.7 – 1.2)	1.1(0.8 – 1.4)
DRE & Biopsy	0.6(0.5 – 0.8)*	2.1(1.5 – 2.7)*	1.4(0.9 – 1.9)
<i>Gleason group</i>			
Well differentiated	1.0	1.0	1.0
Moderately differentiated	1.4(1.3 – 1.6)*	0.6(0.4 – 0.8)*	0.9(0.6 – 1.3)
Poorly differentiated	1.7(1.3 – 2.2)*	0.8(0.6 – 0.9)*	1.1(0.6 – 1.7)
PSA level	0.9(0.9 – 1.0)	1.0(0.9 – 1.0)	1.1(0.9 – 1.0)
<i>Stage at Diagnosis</i>			
Stage I	1.0	1.0	1.0
Stage II	0.5(0.4 – 0.6)*	0.9(0.7 – 1.2)	1.2(0.4 – 2.5)
Stage III	0.4(0.3 – 0.4)*	0.5(0.3 – 0.8)*	0.3(0.2 – 0.5)*
Stage IV	0.5(0.4 – 0.7)*	1.2(1.0 – 1.4)	0.9(0.6 – 1.2)
Unknown	0.5(0.4 – 0.6)*	0.9(0.6 – 1.4)	0.5(0.4 – 0.7)*
<i>Site of Metastasis</i>			
Lymph node	1.0	1.0	1.0
Rectum (bladder neck)	0.9(0.6 – 1.4)	1.1(0.8 – 1.4)	0.7(0.5 – 0.9)*
Bones	0.9(0.7 – 1.2)	1.5(1.1 – 2.1)*	0.9(0.7 – 1.1)
Spine	0.7(0.6 – 0.9)*	1.3(1.1 – 1.6)*	0.8(0.5 – 1.1)
Multiple Metastasis	1.1(0.9 – 1.3)	1.3(0.9 – 1.9)	0.9(0.6 – 1.2)

<i>Treatment Modality</i>			
Surgery	1.0	1.0	1.0
Radiotherapy	0.7(0.7 – 0.8)*	1.0(0.8 – 1.4)	0.9(0.6 – 1.3)
Chemotherapy	1.2(1.5 – 2.1)*	0.9(0.8 – 1.2)	1.1(0.7 – 1.7)
Radiotherapy & Surgery	3.2(2.3 – 4.4)*	1.4(0.9 – 2.0)	2.4(1.3 – 4.5)*
Chemotherapy & Radiotherapy	1.6(1.3 – 1.9)*	1.7(1.2 – 2.3)*	2.1(1.3 – 2.9)*
Chemotherapy & Surgery	3.3(2.6 – 4.4)*	1.2(0.9 – 1.6)	1.9(1.4 – 2.7)*
Chemotherapy & Surgery & Radiotherapy	0.5(0.4 – 0.8)*	CBC	10.4(4.7 – 16.7)*
baseline time	6.2(5.2 – 7.3)	1.8(1.3 – 2.2)	5.5(2.3 – 11.9)
In_p	0.2(0.0 – 0.3)	0.0(-0.1 – 0.2)	-0.0 (-0.1 – 0.1)

NB: CBC = can't be computed; Overall = 2010 - 2017; CrI = Credible Interval

Model adequacy checks

The fitness of the models considered in the multivariate analysis was determined to use *-2LL*, *AIC*, *BIC* and *DIC*. In addition, convergence for the Bayesian model was determined by acceptance rate and graphical diagnostics using trace, histogram, autocorrelation and kernel density plots.

Model check for the Cox proportional hazard regression it was observed that the model for the 2010 – 2014 cohort of patients was the best fitting model with consistently lesser values of *-2LL*, *AIC* and *DIC*. For the parametric Accelerated (Weibull distribution) Failure Time regression, the model for the 2010 – 2014 cohort of patients was observed as the best fitting model. Adequacy check for the Bayesian AFT regression showed that the best fitting model for the Bayesian analysis is the model of the 2010 – 2014 cohort of patients (Table 10).

While checking for the significant parameters in the Bayesian AFT regression, it was observed that most parameters in the 2010 – 2014 cohort model were significant. Observing their MCMC convergence, the result showed that stage I and II levels under the parameter stage at diagnosis converged more as can be shown in the trace plot. A trace plot, plots the values of the simulated parameters against the iteration number and connect consecutive values with a line. For a convergent parameter, the drawn line looks almost vertical and dense, while sparseness and trends suggest convergence problem. The histogram is plotted in relation to the selected prior distribution, in this study a normally distributed prior was selected as such the histogram plot should be normally distributed as deviation from normality indicates convergence issues. The kernel density

plot or smoothed histogram shows three density plots first, second and overall of an MCMC sample, a closely matched plots indicates convergence, while large discrepancies between the halves' suggests convergence problem (*figure 4*). Convergence was best among patients who received chemotherapy for the 2010 – 2014 cohort (*figure 5*).

Table 10: Model Adequacy Check

Criteria	2010 - 2014	2015 - 2017	Overall
Cox Proportional Hazard model (Adjusted)			
-2LL	863.8	927.9	2147.2
AIC	931.8	991.9	2215.1
BIC	1037.2	1095.8	2347.1
Parametric Accelerated Failure Time model (Adjusted)			
-2LL	376.3	435.4	858.1
AIC	448.3	507.4	930.1
BIC	559.9	624.3	1069.4
Bayesian Parametric Accelerated Failure Time model (Adjusted)			
-2LL	959.3	900.8	1281.2
DIC	394.7	456.0	908.7
BIC	932.7	928.2	1311.8
AR	0.4	0.3	0.3
Efficiency	0.2	0.1	0.1

NB: AR = Acceptance Rate; Overall = 2010 - 2017

Figure 4: MCMC Convergence diagnostics for selected characteristics (2010 - 2014) cohort

Figure 5: MCMC Convergence diagnostics for selected characteristics (2010 - 2014) cohort

DISCUSSION

This study accessed the rate of survivorship and determinants of survivorship among CaP patients. The incidence of CaP was highest among patients older than seventy-two years. This is consistent with studies of (Ifere, Abebe, & Ananaba, 2012). CaP is known to be a disease of the aged with very few incidences observed among those below forty years, and very recurring as men progress in age. Advanced age is associated with an aggressive form of this disease (Ji et al., 2017) and a predictor for decreased survival rate as also shown in the Bayesian analysis of this study.

It was observed that patients currently married had longer survival after treatment compared to widowers, this could be due to support received from spouse and family members. Particularly in an African setting where cancer is seen as a death sentence, support from family members and a feeling of not being left alone could encourage quick recovery (Odedina et al., 2009). Further analysis of the data using Cox Proportional Hazard (CPH), Weibull parametric and Bayesian parametric regression however showed that widowers were more likely to be disease-free, with the Bayesian result presenting greater precision as shown in its narrower credible interval. The highest median survival time observed in this study was among the CaP patients from the north central region, this could be attributable to distance patients had to travel to receive treatment, making them better prepared and financially prepared to cover all medical bills and as such receive better care and longer survival. This opinion was also shared by (Zhang & Lawson, 2012) Most patients either drinks alcohol or smokes tobacco, these are known determinants for lung cancer, but it is not yet known if it could be defining in CaP survivorship (Peisch et al., 2017).

Diagnosis via DRE was popular among attending physician, closely followed by biopsy, this is because a DRE can indicate whether a prostate biopsy is recommended for a patient, especially in more aggressive cases (James, 2014) . DRE diagnosis increased survivorship time, this could be due to its ability to quickly detect hardness of the prostate indicating the presence of CaP. DRE is known to be an independent marker for better prognosis (Migowski, 2010), however this was not consistent with this study. It was observed under the Bayesian analysis that diagnosis via transrectal ultrasound, biopsy and a combination of DRE and biopsy were markers for better survival prognosis.

The absence of a parent, or first-degree male relative with a known case of CaP did impact the likelihood of experiencing remission, given the genetics and hereditary factors that come to play from one generation to another it further highlight that there is every likelihood of this disease not recurring in another generation and in agreement with an earlier finding (P A Kupelian, V A Kupelian, J S Witte, R Macklis, 1997).

A well differentiated Gleason group was associated with better prognosis however moderately and poorly differentiated Gleason group was associated with poor disease prognosis as found in the all three analysis, with the Bayesian analysis appearing more precise as can be shown in its narrower credible interval. It was also observed that a large proportion of patients presented with either a moderately or poorly differentiated Gleason score, this could be due to late presentation and lack of adequate awareness on the importance of frequent medical check-ups and early presentation (Afriansyah, Rizal, Hamid, Mochtar, & Umbas, 2018; Khan, 2011). As also reported in studies by (Titilola O Akinremi, Ogo, & Olutunde, 2011) most patients presented at an advanced stage which is a recurring theme among Nigerian patients. This may be a case of negligence and lack of regular awareness on the part of policy-makers on the part of patient's, distance and cost of receiving care may be responsible. As shown in this study late presentation was also a contributing factor for poor survival prognosis and patients in this category often experience recurrence (Migowski, 2010) this is consistent with findings in Ghana (Diaz, Lally, & Pollack, 2010). This shows that early presentation particular at stage I, could increase a patient's chance of being disease-free.

Most patients presented with metastasis especially to the spine and others with multiple metastasis (Titilola O Akinremi et al., 2011; Jedy-agba, Paula, Ogunbiyi, Oga, & Fabowale, 2012). It has been observed that before diagnosis, patients usually go through a series of assumptive diagnosis such that before a disease is detected it has metastasized beyond the prostate region. Much more effort is needed particularly by policy makers to ensure quick diagnosis and frequent screening practices to detect this disease early. Paramount to survivorship is the management of CaP as most patients still experience residual and possible recurrence given the spread of this disease to advance areas, especially in the case of multiple metastasis.

An elevated PSA level at diagnosis is usually associated with poor disease prognosis, although due to differences in technical performance and reference standards, PSA measurements from different labs are not necessarily comparable, however approximately 98% of patients with metastatic CaP will have elevated PSA (Yang & Armstrong, 2010). In this study though, PSA level did not affect treatment outcome across the CPH, parametric and Bayesian models. Observed among patients in this facility was the inconsistency of PSA levels during treatment. This could be due to lack of proper care and lack of finance for consistent treatment on the part of patients, as some patients may miss a certain number of treatments before resuming treatment.

As observed in the CPH model patients who received chemotherapy and surgery were less likely to experience remission, this was also observed in the Weibull and Bayesian parametric regression. Patients who received radiotherapy alone as observed in the Bayesian analysis experienced being disease-free earlier. However only a few patients received this treatment regimen, this could be due to unavailability of a radiotherapy machine, coupled with frequent breakdown and workers industrial action (Gillesen et al., 2018; Robinson et al., 2018; Wolff et al., 2015).

In comparing the CPH, parametric Weibull and Bayesian Parametric models, the Bayesian approach performed better than other models by extracting the most significant predictors particular in the 2010 – 2014 cohort. Some of significant factors were in agreement with those found in (Balogun, Role, & Dawodu, 2014; Migowski, 2010); it was observed that the Bayesian approach is a more appealing alternative to analyzing and reporting time-to-event studies (Zhang & Lawson, 2012) as compared to traditional frequentist approach, due to providing a flexible framework for smoothing rates and representing statistical uncertainty in rate ratios (Chen et al., 2013). Although still used sparingly, the Bayesian approach is fast gaining recognition due to advancement in computing power and software as found in this study.

Limitation of the study

In an effort to carry out this research, the researcher was faced with the challenge of extracting the required data for the study as it involves review of patient medical records; of great concern was the limitation of proper record keeping and lack of follow-up as the true state of most patient could

not be determined. An important limitation is lack of records on patient's com-morbid status, lifestyle and behavioural variables.

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