**Title**

Development and Validation of the Other-Cause Comorbidity-Adjusted Mortality (OCCAM) Model for Clinical Use in Men with Prostate Cancer

**Authors**

Elizabeth C. Chase, MS (1)

Alex K. Bryant, MD (2)

Yilun Sun, PhD (2)

William C. Jackson, MD (2)

Daniel E. Spratt, MD (2)

\*Robert T. Dess, MD (2)

\*Matthew J. Schipper, PhD (1, 2)

\*contributed equally as senior author

**Affiliations**

1. Department of Biostatistics, University of Michigan, Ann Arbor
2. Department of Radiation Oncology, University of Michigan, Ann Arbor

**Acknowledgements**

We thank the National Science Foundation for their support of this research (DGE-1256260) and the Cancer Data Access System staff for their assistance in obtaining our validation data.

**Corresponding Author**

Elizabeth C. Chase

Department of Biostatistics

University of Michigan

1415 Washington Heights, Ann Arbor, MI 48109

Email: ecchase@umich.edu

Tel: (919)370-1073

**Running Head**: Development of the OCCAM Model in Men with Prostate Cancer

**Presented Elsewhere**

* Poster presentation at ASTRO, Miami, FL (virtual due to COVID-19), Oct. 28, 2020

**Disclaimers**: None

**Date of Submission**: 1/7/2021

**Manuscript Word Count**: 3246

**Abstract Word Count**: 276

**ABSTRACT**

**PURPOSE**: STAR-CAP recently released the first AJCC compliant prostate-cancer specific mortality (PCSM) model for men with non-metastatic prostate cancer (www.star-cap.org). However, other-cause mortality (OCM) is the dominant form of death in this patient population. Few clinically usable tools exist to predict OCM risk in these men. Therefore, we aimed to develop and validate an OCM model for men with non-metastatic prostate cancer using two prospective national cohorts.

**METHODS**:Model training was performed using the National Health and Nutrition Examination Survey (NHANES) including men ages > 40 from 1999-2010 with mortality follow-up through 2014 (n = 2,420). The model was validated in the Prostate, Lung, Colon, and Ovarian (PLCO) Cancer Screening Trial, which enrolled men from 1993-2001 with mortality follow-up through 2015 (n = 8,220). Model discrimination (time-dependent AUC) and calibration were assessed in the validation cohort.

**RESULTS**: The median age in the training cohort was 59, with median follow-up of 9.1 years and 459 deaths during follow-up. The final other-cause comorbidity-adjusted mortality (OCCAM) model included 8 predictors: age, education level, marital status, diabetes, hypertension, stroke, BMI, and smoking status. The validation cohort had a median age of 69, with median follow-up of 12.6 years and 2,415 OCM deaths during follow-up. OCCAM had a time-dependent AUC of 0.78 at 15 years for predicting OCM. OCCAM’s performance was superior to an age-only model and actuarial estimates.

**CONCLUSION**: Using eight readily obtained clinical variables, we have developed and validated OCCAM, a model of OCM for men with prostate cancer that performs similarly to or better than other, more complex models. OCCAM will aid in clinical decision-making and can be integrated with PCSM models like STAR-CAP for improved outcome prediction.

**Introduction**

In 2019, there were 174,650 new cases of prostate cancer in the U.S. However, most men diagnosed with prostate cancer will not die of their cancer, instead dying of old age or other comorbidities [1]. Because competing risks of other-cause mortality (OCM) are substantial, they may sometimes outweigh the benefits of prostate cancer treatment or treatment intensification. As a result, prostate cancer treatment guidelines recommend that clinicians take the patient's risk of OCM and life expectancy into account when making treatment decisions.

Little guidance on how to estimate other-cause life expectancy in prostate cancer patients has been provided. The National Comprehensive Cancer Network (NCCN) recommends that clinicians use Social Security Administration (SSA) actuarial tables to assess patient life expectancy [2]. However, research suggests that the SSA tables may overestimate the life expectancy of patients with distant disease [3], and they do not adjust for patient comorbidities, which can make a major difference in patient life expectancy [4]. To address these problems, the NCCN advised that clinicians combine SSA estimates with their clinical assessment of the patient's comorbidity burden. Unfortunately, research suggests that clinical intuition for estimating comorbidity burden is poor [3, 5, 6], and incorrect estimates of patient life expectancy may have major ramifications on treatment plan, with high rates of overtreatment in patients with low-risk prostate cancer and high comorbidity burden [7].

There are few good tools to estimate comorbidity-adjusted life expectancy in men with prostate cancer. Based on a literature review, we identified four OCM prediction models specifically for men with prostate cancer that provided survival predictions and incorporated comorbidity information in some capacity [8, 9, 10, 11]. However, three of the models required so many predictors (twenty or more) that they seemed like they might be burdensome to use in a clinical setting [9, 10, 11], while the fourth incorporated comorbidity information only through self-reported health status [8].

We seek here to develop a clinically usable, parsimonious, validated prediction model for OCM in men with prostate cancer to help personalize treatment decision making. We aim to accurately predict OCM and comorbidity-adjusted life expectancy in men with prostate cancer, and to improve upon the SSA actuarial estimates and existing OCM prediction models.

**Methods**

*Patients*

Our training data come from the National Health and Nutrition Examination Survey (NHANES), a cross-sectional survey conducted every two years in the U.S. since 1999. NHANES is nationally representative of the U.S. non-institutionalized civilian population and contains information on patient demographics and health. More information on NHANES' sample construction and data collection protocols can be found at the survey's website [12]. Mortality follow-up on all NHANES participants is provided through a linkage with the National Death Index, with mortality follow-up through Dec. 31, 2014. Critically, NHANES is not limited to prostate cancer patients, but instead represents the entire U.S. population. We used NHANES data from 1999-2010 and restricted to men older than 40 free of non-prostate cancer with complete data for all covariates used in model-building: age, race, educational attainment, marital status, veteran status, insurance status; diagnosis of anemia, angina, arthritis, asthma, chronic bronchitis, coronary heart disease, congestive heart failure, diabetes, emphysema, high cholesterol, hypertension, kidney issues, liver disease, mental health concern, or myocardial infarction; and alcohol use, regular access to healthcare, whether the patient had been hospitalized in the past year, BMI, and smoking status.

Our validation data come from the Prostate, Lung, Colon, and Ovarian Cancer Screening Trial (PLCO) prostate cohort. PLCO enrolled 155,000 participants between November 1993 and July 2001, with mortality follow-up through Dec. 31, 2009. In order to enroll in the trial, participants had to be ages 55-74, with no history of prostate, lung, colon, or ovarian cancer, and with prostate, lungs, colon, and ovaries intact. Additionally, participants could not be receiving treatment for any cancer, and they could not have received screening for prostate, lung, colon, or ovarian cancer in the past year. Patients who met trial inclusion criteria completed a medical history questionnaire at baseline and were randomized to receive screenings for prostate, lung, and colon cancers if male. If a screening indicated a potential cancer, patients received diagnostic workup, which was included in the PLCO data along with their primary treatment information. For more information on the PLCO protocol and data, please see the trial's website [13]. For validation, we restricted the PLCO sample to men who had been diagnosed with prostate cancer and had complete data for the covariates in our candidate model.

*Variable Definition*

For model training, we used all-cause mortality (ACM) as our outcome (as opposed to OCM) because most patients in the NHANES training population did not have prostate cancer, and the rate of prostate-cancer specific mortality (PCSM) in the general population is low (19.1 per 100,000 annually [14]). Because PCSM comprises only a small portion of ACM in men with localized prostate cancer, we thought that OCM could be approximated by ACM. We did sensitivity analyses, explained further below, to further support this choice.

However, for validation we used OCM as our endpoint, rather than ACM, in order to demonstrate the performance of our model for OCM. To obtain OCM-specific performance estimates, we censored men in the validation data who died of prostate cancer at their time of death. Cause-specific performance was our primary interest, because we wanted to build a cause-specific model, but we also considered performance in the competing risks setting, by setting the censoring time of men who died of prostate cancer at 100,000 months and using that as our endpoint for performance calculation.

More information on variable definition is provided in the supplementary materials; the training data can be reconstructed using the code provided at https://github.com/blindedforreview/PCOtherCause.

*Statistical Analysis*

We considered three classes of models in initial model building: Cox proportional hazards models [15], survival random forests [16], and parametric cubic spline hazard models [17]. We used the NHANES survey weights when performing variable selection for the Cox models, but otherwise did not weight our analysis. Including the survey weights complicated model performance assessment and is beyond the scope of this paper. All analyses were performed in R 3.6.2 [18].

Initial checks suggested that model performance would not suffer substantially from restricting to variables that also appeared in our validation data, so we focused on age, race, educational attainment, marital status, arthritis, chronic bronchitis, coronary heart disease, diabetes, emphysema, hypertension, BMI, and smoking status. We considered clinically relevant interactions and flexible functional forms for the effect of age. Using a combination of clinical knowledge, statistical significance, and assessment of cross-validated C-index, we built separate models using each of our three modeling strategies in the NHANES data, ultimately settling on a Cox proportional hazards model as our leading candidate model.

We conducted sensitivity analyses to assess whether our OCM prediction model developed in a non-prostate cancer population would translate to a prostate cancer patient population. In addition to considering prostate cancer diagnosis, years since diagnosis, and relevant interactions with these covariates as predictors in all models, we examined relationships between the linear predictor of our final models and prostate cancer diagnosis and years since diagnosis (all in NHANES data). These analyses all suggested that having prostate cancer had little effect on predictions for ACM. Despite its minimal impact on model performance, we included prostate cancer as a predictor in all models in order to adjust for its effect.

After completing model-building but before advancing to external validation, we re-estimated model effects and the baseline hazard in the training data using a larger sample of patients with complete data for the final group of predictors. Using our candidate model and the baseline hazard estimated from NHANES, we obtained survival predictions for all men in PLCO and compared these predictions to their true mortality outcomes. We calculated the inverse probability of censoring weighted (IPCW) time-dependent AUC to assess discrimination and obtained bootstrapped confidence intervals for it. We also calculated the IPCW C-index; however, we use the time-dependent AUC as our primary metric of discrimination because of its superior properties in time-horizon settings [19]. We calculated the median survival (truncated at 15 years) as an estimate of life expectancy and made calibration plots to assess the calibration of these life expectancy predictions to observed patient life expectancy. Additionally, we obtain OCM predictions from the 2001 SSA actuarial life tables and the National Vital Statistics System’s (NVSS) life expectancy estimates. We compare the IPCW time-dependent AUC, bootstrapped confidence intervals, and life expectancy calibration of these models to our final candidate model in order to better understand the importance of comorbidity/demographic information for OC mortality predictions. Validation code is provided on GitHub; the validation data are not publicly accessible.

**Results**

*Cohort Features*

After restricting the NHANES data to patients who met our inclusion criteria (Figure 1), we were left with a training sample of 2,420 men, of whom 459 died of all causes over a median of 9.1 years of follow-up. Of these deaths, 111 (24.1%) were from malignant neoplasms. Characteristics of the sample are given in Table 1. Mean age was 59.4 years. 127 patients (5.2%) had been diagnosed with prostate cancer at the time of survey collection; of these patients, 18 had died of malignant neoplasms and 35 had died of other causes by the end of follow-up. Almost two-thirds of the sample were current or former smokers, and more than 75% of the sample was overweight or obese. The PLCO external validation data consisted of 8,220 patients with complete data for all predictors (Figure 1). PLCO patients were markedly different from NHANES patients (Table 1). PLCO patients were older, more likely to be white, more educated, more likely to be married, and generally healthier than NHANES patients. The only characteristic on which NHANES and PLCO patients were not significantly different was previous heart attack or diagnosis of coronary heart disease; roughly 12% of patients had this diagnosis in both samples.

*Model Building and Validation*

Our final other-cause comorbidity-adjusted mortality (OCCAM [20]) model included eight predictors (age, diabetes, education, hypertension, marital status, smoking status, previous stroke, and BMI) and interactions between age and diabetes, age and education, age and hypertension, and age and previous stroke. Model effect estimates for OCCAM are given in Figure 2. Increased age, diabetes, hypertension, current smoking, previous stroke, and non-normal BMI all had harmful effects on OCM, while increased education and being married were protective. All model main effects were significant. Age was the most important predictor, explaining 76.3% of the likelihood, followed by smoking status (5.4%) and marital status (4.0%), then stroke, education, diabetes, hypertension, and BMI. The least important predictor was prostate cancer (0.1%). We also present the survey-weighted version of OCCAM in Supplementary Figure 1, for reference.

In external validation, OCCAM had a cause-specific IPCW AUC of 0.75 at 10 years and 0.78 at 15 years (Figure 3). The IPCW C-index was 0.70. For comparison, the SSA life tables and NVSS life expectancy estimates produced IPCW AUCs of 0.71 and 0.75 at 10 and 15 years, respectively. The IPCW AUCs of OCCAM and the age-only methods were significantly different at 10 years, suggesting that including comorbidity information does improve OCM predictions compared to relying only on age. Estimates of the IPCW AUC for competing risks are given in Supplementary Table 1; performance was largely similar to performance in the cause-specific setting. Model performance was also similar across prostate cancer treatment groups (Supplementary Table 2).

Calibration of OCCAM with the baseline hazard estimated in NHANES (NHANES-Calibrated OCCAM), OCCAM with the baseline hazard re-estimated in PLCO (PLCO-Calibrated OCCAM), and the SSA actuarial table predictions is given in Figure 4. We see that the linear predictor of our model performs very well; calibration of the PLCO-Calibrated OCCAM is excellent. However, we see evidence that the NHANES population is poorly calibrated to PLCO. NHANES patients are at increased risk of OCM compared to the PLCO population; despite having the same linear predictor, the NHANES-Calibrated OCCAM shows pessimism for predicting patient life expectancy in PLCO by about 1.5 years. This pessimism is even more pronounced in the SSA life tables, which generally underestimate life expectancy in PLCO by about 4 years.

*Other Cause Mortality Risk Drivers*

Age is the primary driver of OCM. No men in PLCO ages 55-64 had an OCCAM-predicted OC life expectancy of less than 5 years. However, there is still substantial variation in life expectancy within age bracket (Table 2). For example, in patients ages 75-84, 39.8% have predicted life expectancy less than 10 years, while 60.2% have predicted life expectancy greater than 10 years. Even among younger patients ages 65-74, 3.8% of patients have predicted life expectancy less than 10 years. In the oldest age bracket (85+), approximately 20% of men ages 85+ have predicted life expectancy less than 5 years.

More patient characteristics, split by OCCAM-predicted OC life expectancy, can be found in Supplementary Table 2. We see that patients with reduced OC life expectancy are more likely to be black and other race. Graduating from college is highly protective for OCM, as is being married. Current smokers and former smokers are overrepresented in the high OCM risk group, as are underweight and obese patients. Despite not being considered in modeling, prostate cancer characteristics were also linked with OCCAM predictions. Patients with more aggressive cancers were overrepresented in the high OCM risk group, while patients with less aggressive cancers generally had reduced OCM risk. Patients in the high OCM risk group were also more likely to die of their prostate cancer. Sample sizes for aggressive cancers are quite low, though, given the nature of the PLCO trial, so conclusions about these patients must be made with care. OCCAM predictions can be further explored using the app given at https://blindedforreview.shinyapps.io/ocm\_app/.

In fact, when we use the linear predictor from OCCAM as a predictor for PCSM in PLCO, it still has a time-dependent cause-specific AUC of 0.72 at 15 years, despite not including any prostate cancer specific factors other than age.

*OC Mortality Risk and Treatment Assignment*

We see evidence that both OCM and prostate cancer risk were taken into account in treatment assignment (Figure 5),as patients with lower predicted life expectancy are overall less likely to receive aggressive treatment. Patients with increased predicted life expectancy and increased prostate cancer risk are much more likely to receive more aggressive treatment.

**Discussion**

We present OCCAM, a parsimonious and accurate prediction model for OCM in US prostate cancer patients, built and tested in two diverse, prospective national cohorts. Our model shows excellent discrimination and calibration despite being validated in a population that is very different from its training population. It requires only eight easily obtained predictors and can be accessed at https://blindedforreview.shinyapps.io/ocm\_app/. OCCAM performs comparably to a growing list of OCM prediction models for men with prostate cancer [8, 9, 10, 11]; however, OCCAM stands out for its simplicity, usability and impressive predictive performance in diverse patient populations. OCCAM estimates OC life expectancy, which allows clinicians to follow NCCN guidelines, and because of its simplicity and accessibility, it may facilitate future research on OCM in prostate cancer patients.

When building OCCAM, we considered many predictors and several modeling strategies. The statistical methods used to produce the model are rigorous and do not rely on intermediate risk scores. We present both life expectancy and risk predictions over time, rather than just point estimates. These features make it attractive for future statistical work, and by providing all training data and code used to produce the model, OCCAM can be combined with other prediction tools and treatment effect estimates to build new models. For example, following the approach of Pfeiffer and Gail [21], we can combine OCCAM estimates with cause-specific estimates of PCSM from STAR-CAP [22] to provide personalized estimates of cumulative incidence for both PCSM and OCM, potentially under different treatment paradigms.

We have also made it easy for future researchers to reproduce our analysis and perform similar analyses for new disease sites and applications. This is particularly important because the approach we use here—building an OCM model for cancer patients in a non-cancer patient study—may be generalizable to other cancer patient populations where the cancer-specific mortality is low, and using cohorts likes NHANES vastly increases the number and quality of the OCM predictors that can be considered as candidates. Further research is needed, but this idea may be useful to researchers working on breast cancer, head and neck cancer, and other disease sites where a substantial proportion of patients die of OCM.

We also provide evidence that incorporating comorbidity information is beneficial to predicting OCM, although these gains were modest on the AUC metric. OCCAM significantly outperformed the SSA life tables and NVSS life expectancy estimates. However, in terms of absolute performance the age-only predictors do still perform fairly well and provide a good benchmark for future models. For research purposes, two of our predictors—educational attainment and marital status—may pose problems, as they are not usually collected in prostate cancer cohorts. In exploratory checks, we found that our model refit in NHANES without education and marital status still performed quite well in external validation, with a time-dependent AUC of 0.74 and 0.77 at 10 and 15 years, respectively.

One surprising finding of our model was that race was not included as one of our final eight predictors, although we included it as a candidate predictor during model building. Although race is associated with both OCM and PCSM when unadjusted for other confounders, the inclusion of other health and socioeconomic indicators appears to play a larger role in predicting OCM than race itself.

Our study is subject to several limitations. Although it has its strengths, building a cancer OCM prediction model in a non-cancer patient population could introduce bias. We addressed this limitation through sensitivity analyses of the effect of prostate cancer within our model, and we validated our model in a prostate cancer population, so we believe the risk of bias is low. However, this approach limits our ability to obtain predictions of OC cumulative incidence without refitting the model within PLCO or combining our model with other PCSM prediction tools. Some may argue that our use of a cause-specific endpoint instead of a more traditional competing risks endpoint is a weakness, necessitated by NHANES not being a prostate cancer patient population. However, we argue that the cause-specific endpoint is more reflective of clinical use (i.e. when the NCCN refers to OC life expectancy, they refer to life expectancy *in the absence* of prostate cancer), and we wanted a cause-specific model in order to provide integrated cumulative incidence predictions in future work. Despite being a cause-specific model, our model still appears to perform well on the competing risks metrics presented in the Supplement. As with all cause-specific research, ascertainment of cause of death is a challenge. We sidestep this issue somewhat during model-building by using an overall mortality endpoint, but in PLCO we validate for an OCM endpoint. PLCO assessed cause of death using annual patient surveys and a linkage with the National Death Index. Uncertain deaths were assessed by an adjudication board. Despite this, some PLCO participants may have been incorrectly assigned to OCM or PCSM endpoints.

*Conclusions*

We have built and validated OCCAM, an accurate and succinct model for OCM in prostate cancer patients that is ready for clinical use. OCCAM will improve quality of care when patient life expectancy is a factor in treatment decision-making. The generalizability of our approach and transparency of our documentation will aid future research on cancer OCM across disease sites.

**References**

1. *American Cancer Society*. 2019. Key Statistics for Prostate Cancer. https://www.cancer.org/cancer/prostate-cancer/about/key-statistics.html.
2. National Comprehensive Cancer Network. 2020. NCCN Guidelines Version 2.2020: Prostate Cancer.
3. JD Sammon, F Abdollah, A D'Amico, M Gettman, A Haese, N Suardi, A Vickers, QD Trinh. 2015. Predicting life expectancy in men diagnosed with prostate cancer. *European Association of Urology* 68: 756-65.
4. C Jeldres. 2012. Life expectancy estimation in prostate cancer patients. *Canadian Urological Association Journal* 6.5: 374-5.
5. KMYB Leung, WM Hopman, J Kawakami. 2012. Challenging the 10-year rule: the accuracy of patient life expectancy predictions by physicians in relation to prostate cancer management. *Canadian Urological Association Journal* 6.5: 367-73.
6. J Walz, A Gallina, P Perrotte, C Jeldres, QD Trinh, GC Hutterer, M Traumann, A Ramirez, SF Shariat, M McCormack, JP Perreault, F Bénard, L Valiquette, F Saad, PI Karakiewicz. 2007. Clinicians are poor raters of life-expectancy before radical prostatectomy or definitive radiotherapy for localized prostate cancer. *BJU International* 100.6.
7. TJ Daskivich, K Chamie, L Kwan, J Labo, R Palvolgyi, A Dash, S Greenfield, MS Litwin. 2010. Overtreatment of men with low-risk prostate cancer and significant comorbidity. *Cancer* 117.10: 2058-66.
8. RM Hoffman, T Koyama, PC Albertsen, MJ Barry, TJ Daskivich, M Goodman, AS Hamilton, JL Stanford, AM Stroup, AL Potosky, DF Penson. 2015. Self-reported health status predicts other-cause mortality in men with localized prostate cancer: results from the prostate cancer outcomes study. *Journal of General Internal Medicine* 30.7: 924-34.
9. TJ Daskivich, L Kwan, A Dash, C Saigal, MS Litwin. 2015. An age adjusted comorbidity index to predict long-term, other cause mortality in men with prostate cancer. *Journal of Urology* 194: 73-78.
10. M Kent, DF Penson, PC Albertsen, M Goodman, AS Hamilton, JL Stanford, AM Stroup, B Ehdaie, PT Scardino, AJ Vickers. 2016. Successful external validation of a model to predict other cause mortality in localized prostate cancer. *BMC Medicine* 14.25.
11. P Riviere, C Tokeshi, J Hou, V Nalawande, R Sarkar, AJ Paravat, M Schiaffino, B Rose, R Xu, JD Murphy. 2019. Claims-Based Approach to Predict Cause-Specific Survival in Men With Prostate Cancer. *JCO Clinical Cancer Informatics* 3: 1-7.
12. National Center for Health Statistics, Centers for Disease Control and Prevention. 2019. National Health and Nutrition Examination Survey Data, 1999-2010. https://wwwn.cdc.gov/nchs/nhanes/ContinuousNhanes/Default.aspx
13. Division of Cancer Prevention, National Cancer Institute. 2019. Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial (PLCO). https://prevention.cancer.gov/ major-programs/prostate-lung-colorectal-and-ovarian-cancer-screening-trial
14. U.S. Cancer Statistics Working Group. U.S. Cancer Statistics Data Visualizations Tool, based on 2019 submission data (1999-2017): U.S. Department of Health and Human Services, Centers for Disease Control and Prevention and National Cancer Institute; [www.cdc.gov/cancer/dataviz](https://www.cdc.gov/cancer/dataviz), released in June 2020.
15. DR Cox. 1972. Regression models and life tables. *Journal of the Royal Statistical Society Series B (Methodological)* 34.2: 187-220.
16. H Ishwaran, UB Kogalur, EH Blackstone, MS Lauer. 2008. Random survival forests. *Annals of Applied Statistics* 2.3: 841-60.
17. P Royston, MKB Parmar. 2002. Flexible parametric proportional-hazards and proportional-odds models for censored survival data, with application to prognostic modelling and estimation of treatment effects. *Statistics in Medicine* 21: 2175-97.
18. R Core Team, R Foundation for Statistical Computing. 2019. *R: A Language and Environment for Statistical Computing*. Vienna, Austria. https://www.R-project.org/
19. Blanche P, Kattan MW, Gerds TA. 2019. The c-index is not proper for the evaluation of t-year predicted risks. *Biostatistics* 20.2: 347-357.
20. “Occam’s razor.” *Merriam-Webster’s Unabridged Dictionary.* 2021. https://www.merriam-webster.com/dictionary/Occam%27s%20razor#:~:text=%3A%20a%20scientific%20and%20philosophical%20rule,in%20terms%20of%20known%20quantities
21. Pfeiffer RM and MH Gail. *Absolute Risk: Methods and Applications in Clinical Management and Public Health.* London: Chapman & Hall/CRC Monographs on Statistics and Applied Probability, 2017.
22. Dess RT, Suresh K, Zelefsky MJ, Freedland SJ, Mahal BA, Cooperberg MR, Davis BJ, Horwitz EM, Terris MK, Amling CL, Aronson WJ, Kane CJ, Jackson WC, Hearn JWD, DeVille C, DeWeese TL, Greco S, McNutt TR, Song DY, Sun Y, Mehra R, Kaffenberger SD, Morgan TM, Nguyen PL, Feng FY, Sharma V, Tran PT, Stish BJ, Pisansky TM, Zaorsky NG, Moraes FY, Berlin A, Finelli A, Fossati N, Gandaglia G, Briganti A, Carroll PR, Karnes RJ, Kattan MW, Schipper MJ and Spratt DE. 2020. Development and Validation of a Clinical Prognostic Stage Group System for Non-Metastatic Prostate Cancer: Disease Specific Mortality Results from the International Staging Collaboration for Cancer of the Prostate (STAR-CAP). *JAMA Oncology* 6.12: 1912-1920*.*