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A Simulation-Based Comparison of Fine-Gray Regression and Bayesian Additive Regression Trees for Prostate Cancer Mortality Prediction Under Competing Risks

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Abstract—Background: *Prediction of prostate cancer-specific mortality is challenging when patients may die from causes unrelated to prostate cancer before the event of interest occurs. In such settings, competing mortality can affect absolute risk estimation and may limit the performance of conventional survival approaches. Fine-Gray regression is widely used for competing risks analysis, whereas Bayesian Additive Regression Trees for competing risks may offer greater flexibility for individualized prediction.* ***Objective***: *This study compared Fine-Gray regression with Bayesian Additive Regression Trees for competing risks in predicting prostate cancer-specific mortality using a simulated cohort calibrated to a Sudanese institutional prostate cancer setting.* ***Methods***: *A virtual cohort of 500 patients was generated under a competing risks framework. The simulated outcome distribution included 102 prostate cancer deaths (20.4%), 96 deaths from other causes (19.2%), and 302 censored observations (60.4%), with a median follow-up of 31 months. Fine-Gray regression and Bayesian Additive Regression Trees for competing risks were evaluated using stratified five-fold cross-validation. Predictive performance was assessed at 12, 24, 36, and 48 months using time-dependent area under the curve, Brier score, and calibration plots. Variable importance was also compared between models.* ***Results***: *Fine-Gray regression showed a slightly higher time-dependent area under the curve at 12 months (0.933 versus 0.925). At later horizons, Bayesian Additive Regression Trees for competing risks demonstrated higher discrimination, with area under the curve values of 0.956, 0.905, and 0.843 at 24, 36, and 48 months, respectively, compared with 0.880, 0.812, and 0.718 for Fine-Gray regression. Bayesian Additive Regression Trees for competing risks also produced lower Brier scores at all time points and showed more favorable visual calibration, particularly at longer prediction horizons. Hormonal therapy, Gleason score, prostate-specific antigen, age, and surgery were among the most frequent splitting variables in the Bayesian tree ensemble.* ***Conclusions***: *In this simulation setting, Bayesian Additive Regression Trees for competing risks provided more favorable overall predictive performance than Fine-Gray regression, especially at medium- and longer-term prediction horizons. Fine-Gray regression remains useful for interpretable competing risks regression, whereas Bayesian tree-based modelling may be a complementary tool for individualized risk prediction. External validation using real-world patient-level data is required before clinical implementation.*

Keywords— *Prostate cancer; Competing risks; Bayesian Regression; Fine-Gray; Calibration.*

I. Introduction

Prostate cancer is a major cause of cancer morbidity and mortality among men worldwide. In many clinical settings, especially among older patients and populations with a high burden of comorbidity, death from causes other than prostate cancer can occur before prostate cancer-specific death. This creates a competing risks problem because the competing event prevents the future observation of the event of interest [1,2].

In competing risks data, treating death from other causes as ordinary censoring can distort estimates of absolute event probability. The cumulative incidence function is therefore preferred for estimating the probability of prostate cancer-specific mortality when competing mortality is present. Regression methods are also needed to examine covariate associations and to generate individualized risk predictions [3,4,5].

The Fine-Gray subdistribution hazard model is one of the most commonly used regression approaches for competing risks. It directly models the subdistribution hazard and allows covariates to be related to the cumulative incidence of the event of interest. This makes the model clinically useful when the objective is absolute risk estimation. However, the Fine-Gray model is still a regression-based approach and may be affected by proportionality assumptions, linear covariate effects, and limited flexibility when complex nonlinear patterns or interactions are present [3,4].

Bayesian Additive Regression Trees are flexible nonparametric models that represent the outcome as a sum of many regression trees. Extensions of this framework to survival and competing risks data allow the model to capture nonlinear relationships and interactions without requiring them to be specified in advance. These features may be useful for prediction tasks, although tree-based Bayesian models are generally less directly interpretable than regression models that provide coefficients or hazard ratios [6,7,8,9].

The present study was designed as a simulation-based comparison of Fine-Gray regression and Bayesian Additive Regression Trees for competing risks. The simulation was calibrated to a Sudanese institutional prostate cancer setting, with prostate cancer-specific death as the event of interest and death from other causes as the competing event. The primary objective was to compare discrimination, prediction error, and

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calibration at 12, 24, 36, and 48 months. A secondary objective was to compare variable importance patterns from the two modelling frameworks.

II. Materials and Methods

Study Design and Data Simulation

This was a simulation study designed to compare a classical competing risks regression model with a flexible Bayesian tree-based prediction model. A virtual cohort of 500 patients with prostate cancer was generated. The simulated outcome distribution was calibrated to aggregate institutional targets from a Sudanese prostate cancer setting: 102 prostate cancer-specific deaths (20.4%), 96 deaths from other causes (19.2%), and 302 censored observations (60.4%). The median follow-up time was 31 months, with an interquartile range of 16 to 46 months.

The competing risks structure included three possible outcome states. The event of interest was prostate cancer-specific death. The competing event was death from causes other than prostate cancer. Patients who did not experience either event before the end of follow-up were treated as censored observations. Follow-up time was measured in months.

Simulated Variables

Baseline covariates were selected to represent demographic, clinical, and treatment-related factors commonly used in prostate cancer prognosis. Demographic variables included age at diagnosis, residence, and family history of prostate cancer. Clinical variables included baseline prostate-specific antigen, Gleason score, cancer stage, and comorbidity burden. Treatment-related variables included radiotherapy, chemotherapy, hormonal therapy, and surgery.

Continuous variables were generated to reflect plausible distributions in the calibration setting, whereas categorical variables were generated according to prespecified marginal proportions. Event times were simulated within a competing risks framework, and the observed time for each patient was defined as the earliest time among prostate cancer-specific death, death from other causes, and administrative censoring.

Statistical Models:

Two models were fitted to the simulated data at baseline time zero.

Fine-Gray Subdistribution Hazard Model FGR:

Fine-Gray regression was used to model the subdistribution hazard of prostate cancer-specific death. This model directly relates baseline covariates to the cumulative incidence of the event of interest while accounting for death from other causes as the competing event. Individuals who experienced the competing event were retained in the modified Fine-Gray risk set, which allows the model to estimate covariate associations with the cumulative incidence function rather than the cause-specific hazard.

Cross-validated performance assessment was conducted using predicted cumulative incidence probabilities for prostate cancer-specific mortality, which were produced at 12, 24, 36, and 48 months, after the Fine-Gray model was fitted using the risk Regression package in R [3,4,10].

Bayesian Additive Regression Trees for Competing Risks BART-CR:

Bayesian Additive Regression Trees for competing risks were used as a flexible Bayesian machine learning approach for individualized risk prediction. The model was implemented within a competing risks prediction framework to estimate event-specific risk patterns for prostate cancer-specific death and death from other causes. This approach allows nonlinear predictor effects and interactions to be captured without requiring these relationships to be specified in advance.

Predicted cumulative incidence probabilities for prostate cancer-specific mortality were generated at 12, 24, 36, and 48 months. These predictions were then evaluated using time-dependent area under the curve, Brier score, calibration assessment, and variable importance analysis [6,7,8,9].

Model Training and Validation

A stratified five-fold cross-validation procedure was used to reduce optimism in predictive performance estimates. The stratification preserved the proportions of prostate cancer-specific death, death from other causes, and censoring across folds. Four folds were used to fit the model in each iteration, while the remaining fold was utilized for testing. This process was repeated until each patient contributed once to the test set.

Model performance was assessed at 12, 24, 36, and 48 months using three complementary criteria. Time-dependent area under the curve was used to assess discrimination, meaning the ability of the model to rank patients according to their predicted risk of prostate cancer-specific death. Brier score was used to assess overall prediction error, with lower values indicating better predictive accuracy. Calibration was assessed graphically by comparing mean predicted cumulative incidence with observed cumulative incidence across risk groups [10,11,12,13,14,15].

Variable Importance Analysis

Variable importance was examined to support interpretation of the prediction models. For the Bayesian tree-based model, variable importance was defined as the mean proportion of posterior splitting rules that used each variable across the tree ensemble. This measure reflects the relative contribution of each predictor to the model prediction structure and should not be interpreted as a regression coefficient or a causal effect.

For the Fine-Gray model, variables were ranked using the absolute value of the Wald statistic, with subdistribution hazard ratios, confidence intervals, and p-values reported. Because the two approaches define importance differently, the comparison was interpreted descriptively rather than as a direct equivalence between regression effects and tree-based splitting frequencies.

Software

All analyses were conducted using R. Fine-Gray regression and prediction performance measures were implemented using the riskRegression package. Bayesian tree-based modelling was implemented using the dbarts package. Cross-validation procedures were implemented using standard resampling routines [9,10].

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The present manuscript used simulated data only. No identifiable patient-level records were analyzed, and no direct contact with human participants occurred. Therefore, formal ethics approval was not required for this simulation analysis. If the model is later validated using individual-level clinical data, institutional ethics approval will be required before analysis.

III. Results

Simulated Cohort Characteristics

The simulated cohort included 500 patients. The median age was 72 years, with an interquartile range of 67 to 78 years. The majority of patients resided in urban areas. Advanced disease was common, with stage III and stage IV disease together representing more than two-thirds of the cohort. During follow-up, 102 patients died from prostate cancer, 96 died from other causes, and 302 were censored. Baseline characteristics and outcome distribution are shown in Table I and II.

TABLE I. Continuous baseline variables and follow-up time in the simulated cohort

Variable	Unit	Median	Q1	Q3
Age	Years	72	67	78
Prostate-specific antigen	ng/mL	16	9	22
Follow-up time	Months	31	16	46

Cross-validated Predictive Performance

Cross-validated model performance is summarized in Table III and Figure 1. At 12 months, Fine-Gray regression showed a slightly higher time-dependent area under the curve than Bayesian Additive Regression Trees for competing risks (0.933 versus 0.925). At 24 months and later, the Bayesian tree-based model showed higher discrimination. The absolute differences in the region under the curve were 0.076 at 24 months, 0.093 at 36 months, and 0.125 at 48 months.

TABLE II. Categorical baseline characteristics and outcome distribution of the simulated cohort

Characteristic	Category	Frequency	Percentage
Residence	Urban	312	62.4
Residence	Rural	150	30.0
Residence	Other	38	7.6
Family history	Yes	47	9.4
Gleason score	6	96	19.2
Gleason score	7	147	29.4
Gleason score	8	100	20.0
Gleason score	9	106	21.2
Gleason score	10	51	10.2
Cancer stage	I	64	12.8
Cancer stage	II	95	19.0
Cancer stage	III	150	30.0
Cancer stage	IV	191	38.2
Comorbidity	0	255	51.0
Comorbidity	1	156	31.2
Comorbidity	>=2	89	17.8
Radiotherapy	Yes	195	39.0
Chemotherapy	Yes	151	30.2
Hormonal therapy	Yes	246	49.2
Surgery	Yes	89	17.8
Outcome status	Censored	302	60.4
Outcome status	Death from other causes	96	19.2
Outcome status	Prostate cancer-specific death	102	20.4

Brier scores are shown in Table III and Figure 2. Bayesian Additive Regression Trees for competing risks produced lower Brier scores than Fine-Gray regression at all evaluated time points. The Brier score was 0.027 versus 0.037 at 12 months, 0.057 versus 0.073 at 24 months, 0.093 versus 0.114 at 36 months, and 0.147 versus 0.173 at 48 months for the Bayesian tree-based and Fine-Gray models, respectively.

Calibration Assessment

Calibration plots are presented in Figure 3. Visual assessment suggested that the Bayesian tree-based model generally aligned more closely with the ideal calibration line across prediction horizons. Fine-Gray regression showed increasing underprediction at longer horizons, particularly among patients in higher predicted-risk ranges. This pattern was most apparent at 36 and 48 months.

TABLE III. Cross-validated performance metrics for BART-CR and Fine-Gray models

Time months	Model	AUC mean	AUC SD	Brier score mean	Brier score SD
12	BART-CR	0.925	0.098	0.027	0.014
12	Fine-Gray	0.933	0.028	0.037	0.010
24	BART-CR	0.956	0.029	0.057	0.011
24	Fine-Gray	0.880	0.072	0.073	0.009
36	BART-CR	0.905	0.028	0.093	0.015
36	Fine-Gray	0.812	0.068	0.114	0.018
48	BART-CR	0.843	0.025	0.147	0.011
48	Fine-Gray	0.718	0.062	0.173	0.010

Variable Importance

The top predictors ranked according to model-specific importance metrics are listed in Table IV. For Bayesian Additive Regression Trees for competing risks, the five most frequently used splitting variables were hormonal therapy, Gleason score, prostate-specific antigen, age, and surgery. Their importance values were close to each other, ranging from 0.069 to 0.070, suggesting distributed predictive contributions across several clinical variables.

TABLE IV. Top five predictors ranked by model-specific importance measures

Rank	BART-CR variable	BART-CR importance	Fine-Gray variable	SHR	95% CI lower	95% CI upper	P value
1	Hormonal therapy	0.070	Hormonal therapy	0.605	0.406	0.901	0.014
2	Gleason score	0.069	Cancer stage III	2.006	0.961	4.185	0.064
3	Prostate-specific antigen	0.069	Surgery	1.447	0.894	2.340	0.130
4	Age	0.069	Family history	1.485	0.830	2.657	0.180
5	Surgery	0.069	Cancer stage IV	1.624	0.786	3.354	0.190

For the Fine-Gray model, hormonal therapy was the strongest predictor and was associated with a lower subdistribution hazard of prostate cancer-specific death (subdistribution hazard ratio 0.605, 95% confidence interval

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0.406 to 0.901, p = 0.014). Cancer stage III, surgery, family history, and cancer stage IV were also among the top ranked Fine-Gray predictors, although their p-values did not meet the conventional 0.05 threshold [16,17,18,19].

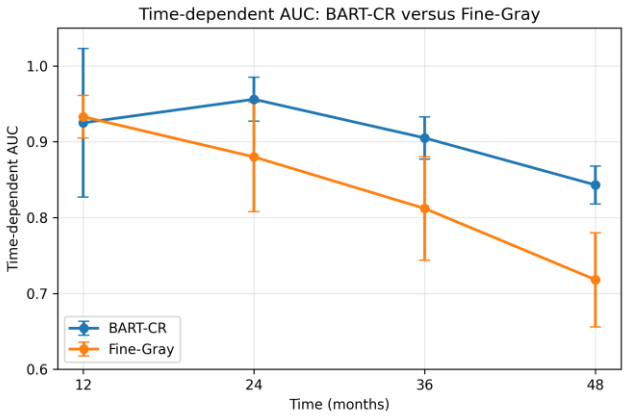

Fig. 1. Time-dependent AUC: BART-CR vs Fine-Gray

Error bars represent the standard deviation across five-fold cross-validation. BART-CR: Bayesian Additive Regression Trees for competing risks; AUC: area under the curve.

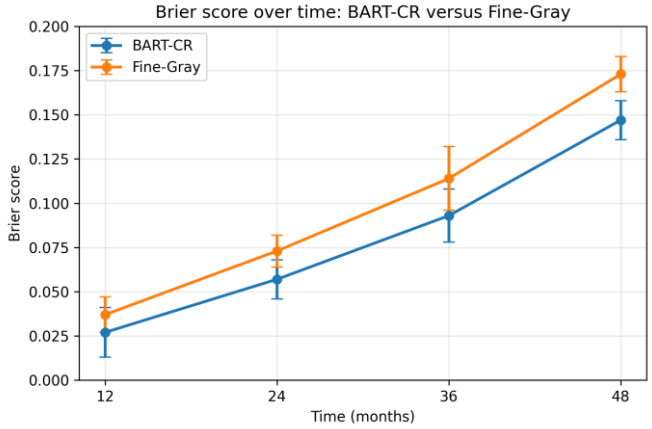

Fig. 2. Brier Score over time: BART-CR vs Fine-Gray

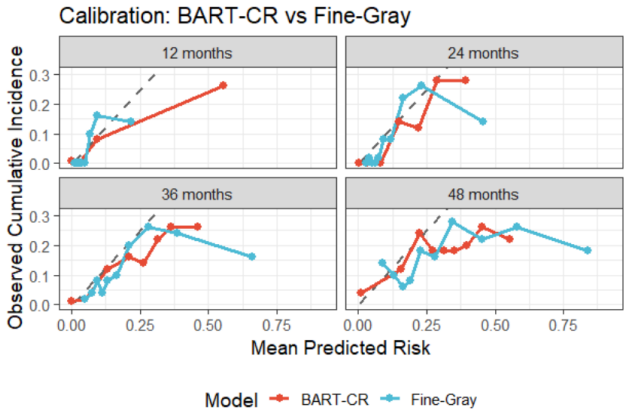

Fig. 3. Calibration plots: BART-CR vs Fine-Gray

Lower Brier scores indicate lower prediction error. Error bars represent the standard deviation across five-fold cross-validation. BART-CR: Bayesian Additive Regression Trees for competing risks.

Points represent mean predicted versus observed cumulative incidence within risk groups. The dashed line indicates perfect calibration. BART-CR: Bayesian Additive Regression Trees for competing risks.

IV. Discussion

Main Findings

This simulation study compared Fine-Gray regression and Bayesian Additive Regression Trees for competing risks for the prediction of prostate cancer-specific mortality in the presence of competing mortality. The Bayesian tree-based model showed more favorable overall predictive performance, particularly at medium- and longer-term prediction horizons. Although Fine-Gray regression performed slightly better in discrimination at 12 months, the Bayesian tree-based model showed higher discrimination at 24, 36, and 48 months, lower Brier scores at all horizons, and more favorable visual calibration.

These findings suggest that flexible Bayesian tree-based modelling may capture nonlinear patterns or interactions that are not easily represented by a standard subdistribution hazard regression model. However, the results should be interpreted within the context of a simulation study. The findings do not imply that Bayesian Additive Regression Trees should replace Fine-Gray regression in all competing risks analyses. Rather, the two approaches answer complementary questions: Fine-Gray regression provides interpretable subdistribution hazard ratios, whereas Bayesian tree-based modelling may provide stronger individualized prediction when complex predictor-outcome relationships are present.

Comparison with Previous Work

The results are consistent with the broader prediction modelling literature, which emphasizes that model performance should be assessed using multiple criteria rather than discrimination alone. A model with strong discrimination may still be poorly calibrated, and a model with good calibration may not rank patients well. In the present simulation, the Bayesian tree-based model showed advantages across both prediction error and visual calibration, especially at later time points [11,12,13,14,15].

Previous work on Bayesian Additive Regression Trees has shown that tree ensembles can flexibly model nonlinear covariate effects and interactions while avoiding the need for the analyst to specify these structures in advance [6,7,8,9]. This flexibility is a plausible explanation for the observed improvement at longer horizons. In contrast, the Fine-Gray model remains valuable because it directly targets the cumulative incidence function and provides regression parameters that can be interpreted in relation to subdistribution hazards [3,4,5].

Clinical Implications

From a clinical prediction perspective, accurate absolute risk estimation is important when clinicians and patients make decisions under competing risks. In prostate cancer, especially among older patients or patients with comorbidity, death from

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non-cancer causes can substantially alter the probability of observing prostate cancer-specific death. A prediction model that accounts for this structure may provide more realistic risk estimates than an approach that ignores competing mortality [3,4,5].

The present findings support the use of a complementary modelling strategy. Fine-Gray regression can be used when the primary objective is interpretable competing risks regression and estimation of covariate associations with cumulative incidence. Bayesian Additive Regression Trees for competing risks may be useful when the primary objective is individualized prediction, particularly if nonlinear effects or interactions are expected. In practice, using both models together may provide a balance between interpretability and predictive flexibility.

Strengths and Limitations

This study has several strengths. The simulation used a clearly defined competing risks structure, included clinically relevant demographic, clinical, and treatment-related variables, and evaluated model performance using discrimination, prediction error, and calibration. The use of stratified five-fold cross-validation reduced optimism compared with evaluating performance on the same data used for model training.

The study also has important limitations. First, the analysis was based on simulated data calibrated to aggregate institutional targets rather than individual-level real-world patient records. Therefore, the data may not fully capture measurement error, treatment selection, missingness, unmeasured confounding, or heterogeneity observed in routine clinical practice. Second, the sample size was modest, and the performance estimates may vary under alternative simulation assumptions. Third, Bayesian tree-based models are less directly interpretable than regression models and may require additional tools, such as partial dependence plots or local explanation methods, before clinical use. Finally, external validation using real patient-level data is required before any clinical implementation is considered [20].

Future Directions

Future studies should validate these findings using real-world prostate cancer cohorts with carefully adjudicated cause-specific mortality and sufficient follow-up. Further work should also compare Bayesian Additive Regression Trees with other flexible competing risks prediction methods, such as random survival forests and penalized regression [21]. Incorporating additional biomarkers, longitudinal covariates, and treatment timing may also improve individualized prediction. Finally, research is needed on explain ability tools that can make flexible prediction models more transparent for clinicians.

V. Conclusion

In this simulation study, Bayesian Additive Regression Trees for competing risks showed more favorable overall predictive performance than Fine-Gray regression for prostate cancer-specific mortality prediction in the presence of competing mortality. The advantage was most evident at medium- and longer-term prediction horizons, where the Bayesian tree-based model showed higher discrimination, lower prediction error, and more favorable visual calibration.

Fine-Gray regression remains an important and interpretable method for modelling cumulative incidence under competing risks. Bayesian tree-based modelling should be viewed as a complementary approach for individualized risk prediction rather than a replacement for classical competing risks regression. These findings support further validation using real-world patient-level data before clinical application.

Additional Information

Data Availability Statement: The simulated data and R analysis code supporting the findings of this study are available from the corresponding author upon reasonable request.

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Conflicts of Interest: The authors declare that they have no conflicts of interest.

Human Subjects and Ethics Statement: This manuscript reports a simulation study and did not involve identifiable patient-level data or direct human participant contact. Formal ethics approval was not required for the present simulated analysis.

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