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Association Structures in Bayesian Joint Models of Longitudinal Markers and Survival Outcomes: Application to Cardiac Data

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Abstract

In clinical research, longitudinal biomarkers and time-to-event outcomes are often correlated. Standard separate modeling of these processes leads to biased estimates due to the failure to account for endogeneity. This study investigates the application of the Bayesian joint modeling framework to quantify the association between pulse rate trajectories and mortality risk in cardiac patients. A primary focus is placed on comparing various association structures to determine the most effective parameterization for medical inference. Data from cardiac patients at an Ethiopian cardiac center were analyzed using a linear mixed model for pulse rate and a Cox PH model for time-to-death. Three distinct association structures current value, current value plus slope, and shared random effects were estimated via Markov chain Monte Carlo (MCMC) algorithms. The current value plus slope association structure provided the best model fit, demonstrating that both the current level of pulse rate and its instantaneous rate of change are significant predictors of mortality. Clinical findings also indicated that underweight status and pulmonary complications significantly increase the hazard of death, while corrective surgery serves as a protective factor. Joint modeling offers a robust unified approach for medical research, yielding more efficient and less biased estimates than separate analyses. The choice of association structure is critical; specifically, incorporating the trajectory's slope alongside its current value enhances the predictive performance and interpretability of clinical biomarkers in survival analysis.

Keywords

Joint Modeling, Association Structures, Bayesian Inference, Longitudinal Data, Survival Analysis, Cardiac Data

1. Introduction

In many clinical researches, longitudinal measurements on continuous outcomes alongside time-to-event data are collected [1, 2]. Such data structures are particularly prevalent in biomedical and behavioral sciences [3]. For instance, in clinical studies, repeated assessments of biomarkers such as blood pressure, antibody affinity, or cholesterol levels are often obtained to monitor disease progression and are intrinsically linked to events, including mortality, disease onset, or recovery [4]. Substantial interest lies in quantifying the association between biomarker trajectories and the event of interest [5, 6].

From a methodological standpoint, classical approaches treat these outcomes separately: linear mixed effects models are used to analyze longitudinal measurements, whereas sur-

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vival models are used for time-to-event outcomes [7-9]. However, such a framework is often inadequate due to the presence of endogeneity between the two processes [8]. Specifically, the longitudinal measurements are informative of the underlying event process, implying that the observed biomarker history at a given time point carries predictive information about the future occurrence of the event [10, 11]. Ignoring this intrinsic dependence can result in biased parameter estimates, loss of efficiency, and invalid inference [12].

To address these limitations, joint modeling frameworks have been developed, enabling the simultaneous estimation of longitudinal and time-to-event processes within a unified probabilistic approach [2, 3]. These models explicitly account for the dependence between the two outcomes, typically through functional association structures, and provide direct inference on the relationship between the longitudinal marker and the hazard of the event [12, 13]. Consequently, joint models yield more efficient and less biased estimates while offering improved predictive performance [15].

A key strength of joint modeling lies in its flexibility in specifying the association structure between the longitudinal and survival submodels [16, 17]. The longitudinal process and its relationship with the hazard function can be parameterized in multiple ways, including dependence on the current value, rate of change, or latent random effects [15]. Each specification carries distinct theoretical and practical implications, underscoring the importance of carefully selecting an appropriate association structure [5, 8].

Despite methodological advantages and increasing adoption in medical statistics, joint modeling approaches remain relatively underutilized in clinical and psychological research [7, 14, 15]. This gap is notable given the growing availability of rich longitudinal datasets in these fields and the increasing importance of understanding how long-term behavioral and cognitive trajectories relate to health outcomes and survival [11, 13].

Motivated by these considerations, the present study focuses on association structures within the Bayesian joint modeling framework for longitudinal and time-to-event data. Specifically, it aims to investigate how alternative parameterizations of the association between the longitudinal process and the hazard function influence statistical inference, model fit, and interpretability. By providing a rigorous comparative analysis, this work seeks to contribute both to methodological development and to the prasctical application of joint models in complex data settings.

2. Statistical Framework

This section outlines the components underlying joint modeling of longitudinal and time-to-event data. For completeness, formulations of both the mixed-effects submodel for longitudinal outcomes and the survival submodel for event-time data are present, which together define the joint modeling framework.

Joint modeling, formally introduced by Wulfsohn and Tsiatis [18] and further extended in subsequent work [3, 5, 8, 10, 14], comprises two interconnected submodels: (i) a longitudinal submodel for repeated measurements, and (ii) a time-to-event submodel that describes the hazard of an event occurrence. The defining feature of joint models is the simultaneous specification of a joint likelihood, whereby the longitudinal and survival processes are linked through shared latent structures or functional relationships, rather than being modeled independently through separate marginal likelihoods [11, 12].

More specifically, the two submodels are combined by allowing the hazard function in the survival component to depend on certain features of the longitudinal process [10]. These features may include the current value, slope, or subject-specific random effects [5, 8]. This formulation explicitly accounts for the endogenous relationship between the repeated measurements and the event process.

2.1. Linear Mixed Effects Submodel

The longitudinal component consists of repeated measurements of a continuous outcome collected for each individual over time. Due to the within-individual association, observations from the same individual are dependent [19]. Each individual is assumed to follow an individual specific trajectory, reflecting both population level trends and individual level deviations [20].

To handle this hierarchical structure, the longitudinal outcome is typically modeled using a linear mixed effects model, which decomposes variability into fixed and random effects [21, 22]. The fixed effects capture the average population trajectory, while the random effects account for between-individual variability by allowing individual-specific deviations from the population mean.

Let Y_{ij} be a measurement on longitudinal outcome Y for the $i-th$ individuals $(i = 1,2,\dots,n)$ at time t_{ij} of visit j $(j = 1,2,\dots,n_i)$. The linear mixed effect model can be given by [21, 22]:

$$Y_{ij} = X_{ij}\beta + Z_{ij}b_i + \epsilon_i, \quad (1)$$

$$Y_{ij} = \eta_i(t) + \epsilon_i, \quad (2)$$

$$b_i \sim N(0, \Sigma_b), \epsilon_i \sim N(0, \Sigma),$$

where β is denote fixed effect parameters and b_i denotes the vector of random effects for individual i with $b_i \sim N(0, \Sigma)$, *and* Σ is the variance-covariance matrix of the random effects (typically a 2 X 2 unstructured matrix with random intercept and random slope effects). X_{ij} and Z_{ij} represent the design matrix containing the predictors for the fixed effects coefficients β and for the random effects regression coefficients b_i, respectively (typically b_{oi} and b_{1i} for random intercept and slope effects).

2.2. Time-to-Event Submodel

Time-to-event analysis is designed to analyze the distribution of time until the occurrence of a well-defined event of interest [23]. The event time is defined as the elapsed time from a specified origin to the occurrence of an event, such as death, disease onset, or recovery [24].

Let T_i^* denote the failure time, C_i as the censoring time and $T_i = min(T_i^*, C_i)$ as the observed time for ith subject. The Cox PH model can be written as [23, 24]:

$$h_i(t) = \lambda_o(t)exp(\alpha^T W_i), t > 0 \quad (3)$$

where $\lambda_o(t)$ is the baseline hazard function at time t and W_i is a vector of baseline predictors with corresponding regression coefficients α. The submodel has two distinct parts. First, $\lambda_o(t)$ describes the hazard when all covariates W_i have value zero. Second, the effect parameters $\alpha^T W_i$ describe how the hazard varies as a function of the explanatory covariates W_i.

2.3. Joint Model

Joint models provide a framework for simultaneously analyzing longitudinal and time-to-event data by linking them through shared parameters [15]. The dependence between the two processes is captured by allowing the hazard of the event to depend on individual specific features of the longitudinal trajectory [14]. This is achieved through association parameters that quantify the strength and types of association between the longitudinal and survival outcome [16].

This approach models the hazard of an event as dependent on individual specific characteristic of longitudinal trajectory and assumes that the risk of an event is a function of the linear predictor $\eta_{ik}(t)$ and/or the random effects [8, 10, 15]. More specifically:

$$h_i(t|H_i(t), W_i) = \frac{\lim_{\Delta t \to 0} P(t \le T_i^* < t + \Delta t | T_i^* \ge t, H_i(t), W_i)}{\Delta t}, t > 0 \quad (4)$$

$$h_i(t|H_i(t), W_i) = \lambda_o(t)exp\{\alpha^T W_i + \sum_{l=1}^{L} f(H_l(t), b_i, \gamma_l)\}, t \quad (5)$$

where $H_i(t) = \{\eta_i, 0 \le s < t\}$ denotes the history of the underlying longitudinal process up to t, $\lambda_o(t)$ denotes the baseline hazard function, and W_i is a vector of covariates with corresponding regression coefficients α. Functions $f(.)$ parameterized by vector γ_l, specify which components/features of each longitudinal outcome are included in the linear predictor of the survival submodel.

2.4. Association Structures

The linking function $f(\cdot)$ in equation (5) have various options which lead to different forms of association between the longitudinal and time to event data. One is called current value [2, 25] association $(\gamma_l \eta_i(t))$ in which longitudinal measure $\eta_i(t)$ is predictive of the risk of event at the same time t. Second one is called current value plus slope [2, 25] association$(\gamma_1 \ \eta_i(t) + \gamma_2 \eta_i'(t))$. This extends the first structure by adding the slope term at time t. The third one is called shared random effects s association$(\gamma_l b_i)$, which includes only the random effects from the longitudinal submodel.

2.4.1. Current Value Association

The first association structure between the longitudinal and the time-to-event submodels is known as the current value [2, 25], and assumes that for individual i, the true value $\eta_i(t)$ of the longitudinal measure at time t is predictive of the risk $\lambda_i(t)$ of experiencing the event at that same time t.

Association structure takes into account current value association structure [2, 25] is defined as

$$f(H_i(t), b_i, \gamma) = \gamma_l \ \eta_i(t), \quad (6)$$

So, The Cox PH submodel with this association structure is written as

$$h_i(t|H_i(t), W_i, b, \gamma) = \lambda_o(t)exp\{\alpha^T W_i + \gamma_l \ \eta_i(t)\}, t > 0 \quad (7)$$

The instantaneous risk of the event at time t depends on the subject-specific true value of the longitudinal outcome at that same time, with $exp(\alpha)$ representing the relative change in hazard associated with a one-unit increase in the current biomarker level.

2.4.2. Current Value Plus Slope Association

The second association structure between the time-to-event and the longitudinal submodels is known as the current value plus slope parameterization [2, 25]. This extends the previous structure by adding the rate of change of the measurement at time t, estimated as the derivative of $\eta_i(t)$ w. r. t. the time metric.

Association structure takes into account current value and slope association structure [2, 25] is defined as

$$f(H_i(t), b_i, \gamma) = \gamma_1 \ \eta_i(t) + \gamma_2 \eta_i'(t), \quad (8)$$

where $\eta_i'(t) = \frac{d\eta_i(t)}{dt}$ $(\eta_i'(t))$ is the derivative of $\eta_i(t)$with respect to the time variable t.

So, the time-to-event sub-model is rewritten as

$$h_i(t|H_i(t), W_i, b, \gamma) = \lambda_o(t)exp\{\alpha^T W_i + \gamma_1 \ \eta_i(t) + \gamma_2 \eta_i'(t)\}, t > 0 \quad (9)$$

where the hazard of experiencing the event at time t is now assumed to be associated with both the value of $\eta_i(t)$ and its rate of change (slope) $\eta_i'(t)$ at time t. The hazard of the event at time t is jointly determined by the current level of the longitudinal marker and its instantaneous rate of change, where γ_1 captures the effect of the level and γ_2 captures the

effect of the trajectory's. For individuals having the same level of $\eta_i(t)$, the hazard ratio for a one-unit increase of the current rate of change $\eta_i'(t)$ (trajectory) is $\exp(\gamma_2)$. Identically, for individuals having the same rate of change $\eta_i'(t)$ one-unit increase of the current value $\eta_i(t)$ is $\exp(\gamma_1)$.

2.4.3. Shared Random Effects Association

The shared random effects association structure links the longitudinal and survival processes through the subject-specific random effects estimated from the longitudinal submodel [16, 17]. In this approach, the random effects capture the unobserved individual-specific characteristics that jointly influence both the repeated measurements and the risk of the event.

The association structure is defined as

$$f(H_i(t), W_i(t), b_i, \gamma) = \gamma_1 b_{0i} + \gamma_2 b_{1i} \quad (10)$$

So, the time-to-event sub-model is rewritten as

$$h_i(t|H_i(t), W_i, b, \gamma) = \lambda_0(t) exp(\alpha^T X_i + \gamma_1 b_{0i} + \gamma_2 b_{1i}) (11)$$

where γ are the coefficients linking the longitudinal and survival parts.

The hazard of the event is determined by subject-specific latent characteristics of the longitudinal trajectory, where the random intercept captures baseline disease level and the random slope captures progression rate, and their effects on risk are quantified by γ_1 and γ_2, respectively. For a random effects structure (random intercept and slope effects), the random effects represent the individual deviations from the sample average intercept and slope values, and the association parameters reflect the change in the log hazard for a one-unit change in these deviations.

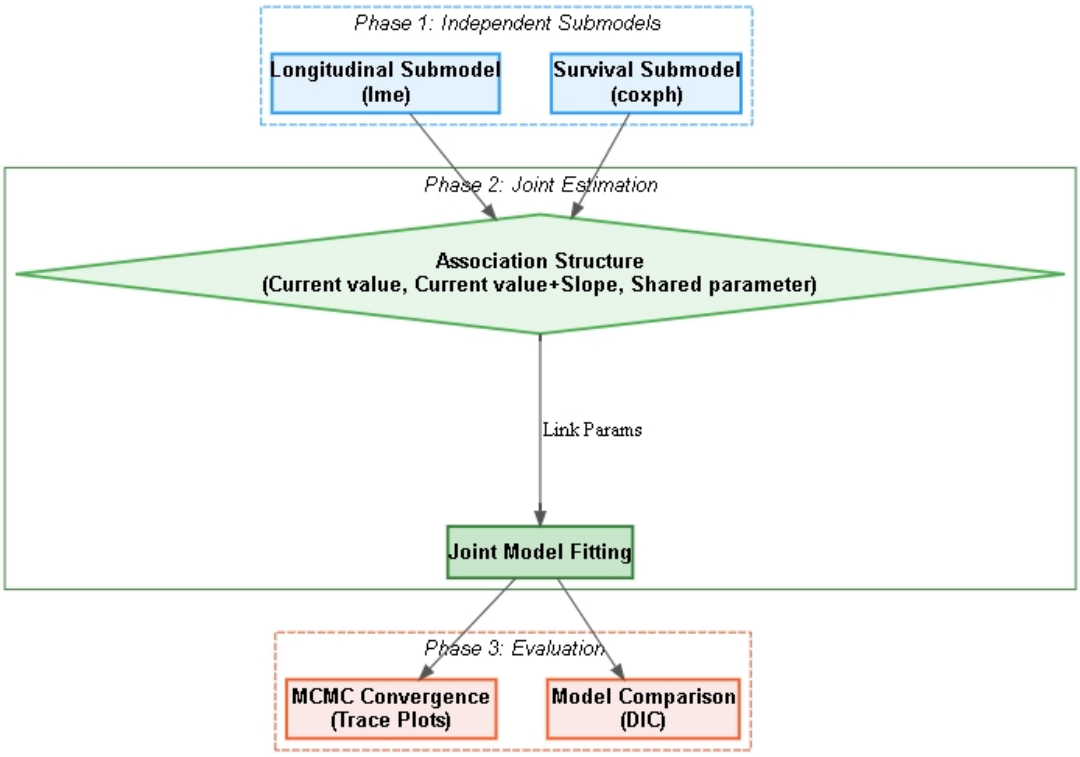

Figure 1. *Modeling framework for the joint modeling of longitudinal and survival process.*

2.5. Bayesian Estimation

Bayesian estimation was adopted because it provides a unified framework for jointly modeling longitudinal and survival outcomes while fully accounting for uncertainty in all model parameters [26]. Unlike frequentist approaches, Bayesian inference yields full posterior distributions and credible intervals and is particularly suitable for complex hierarchical joint models [27]. The Bayesian framework also allows straightforward incorporation of prior information when available, although weakly informative priors were used in this study so that inference was driven primarily by the observed data.

2.5.1. Likelihood Function

The parameters of the joint model were estimated under the Bayesian estimation approach using Markov chain Monte Carlo (MCMC) algorithms [28, 29]. The time-to-event (T_i) is independent of the longitudinal measurements (Y_{ij}) and

conditional on the random effects b_i in the joint model. Let θ_1 and θ_2 denote the combined parameter vector for survival and longitudinal process respectively, and $\theta = (\theta_1, \theta_2)$, then the likelihood [26] can be written as

$$f(\theta|T_i, \delta_i, Y_{ij}) = \prod_{i=1}^{n}\prod_{j=1}^{n_i} f(T_i, \delta_i, Y_{ij}|b_i, \theta)$$

$$= \prod_{i=1}^{n}\prod_{j=1}^{n_i} f(Y_{ij}|b_i, \theta_2)\, S(T_i, \delta_i|\ b_i, \theta_1) f(b_i|\theta) \quad (12)$$

2.5.2. Posterior Distribution

The fundamental principle is that the posterior distribution is a function of the likelihood function and the prior distribution [27]. The posterior distribution of the model parameters given the observed data $\pi(\theta|Y_{ij}, T_i, \delta_i)$ is derived under the assumptions that the longitudinal outcomes are independent of the event time, the longitudinal outcomes are independent of each others, longitudinal responses of each subject for outcomes are independent, and conditional on the random effects. Under these assumptions the posterior distribution is analogous to:

$$\pi(\theta, b_i|Y_{ij}, T_i, \delta_i) = \frac{\prod_{i=1}^{n}\prod_{j=1}^{n_i} f(Y_{ij}|b_i,\theta_2) S(T_i,\delta_i|\ b_i,\theta_1) f(b_i|\theta)\ \pi(\theta)}{\int f(Y_{ij},T_i,\delta_i|\theta,b_i)\ d\theta\ db}$$

$$\propto \prod_{i=1}^{n}\prod_{j=1}^{n_i} f(Y_{ij}|b_i, \theta_2)\, S(T_i, \delta_i|\ b_i, \theta_1) f(b_i|\theta)\ \pi(\theta) \quad (13)$$

where $\pi(\theta)$ is a prior distributions, and $\theta = (\theta_1, \theta_2)$ is a parameters vector. Similarly, δ_i is indicator function, $\delta_i = I(T_1^* \leq C_i)$ such that $\delta_i = 1$ when the event is observed and 0 when censored.

In the Bayesian approach, priors were selected to have minimal influence on posterior inference. For the parameters of the longitudinal submodel, standard default weakly informative priors recommended in the literature [30] were used. For the regression parameters of the survival submodel, independent normal priors with mean zero and variance 1000, $N(0,1000)$ were specified to represent weak prior information, allowing the observed data to dominate the posterior estimates.

The B-spline approximation for the baseline hazard was implemented using the default specification in the Bayesian joint modeling framework, providing sufficient flexibility to capture the underlying hazard over time [2, 25].

3. Results

3.1. Cardiac Dataset

The data for this study come from a clinical follow-up of cardiac patients at cardiac center Ethiopia monitored for biomarkers and survival outcomes. This longitudinal study tracks cardiac patients to examine the relationship between clinical biomarkers and the risk of death. From total of children treated at the center, the study considers a sample of 323 children [31].

This analysis focused on pulse rate as the longitudinal marker, which serves as an indicator of physiological stability in cardiac patients. Biomarkers were assessed up to six visit times for approximately 2-year period. Here, we investigate whether an individual's baseline level of and the subsequent change in pulse rate during the follow-up period predicts their risk of death. Pulse rate was assessed through repeated measurements recorded during the follow-up period. Conceptual relationship between variable were given in Figure 2. Variables included in the current analyses were:

Longitudinal Submodel: age at assessment, nutritional status, types of cardiac disease (CD), and anemia status.

Time-to-Event Submodel: Age, nutritional status, types of cardiac disease (CD), corrective surgery, and pulmonary hypertension.

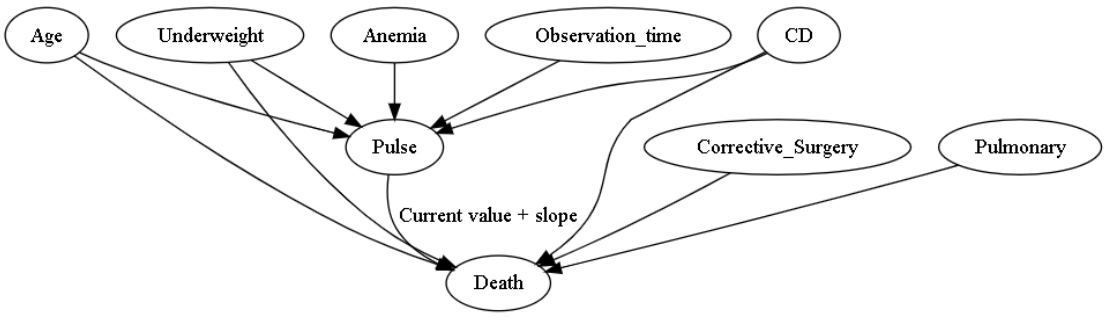

Figure 2. *Conceptual relationship between covariates and outcome variables.*

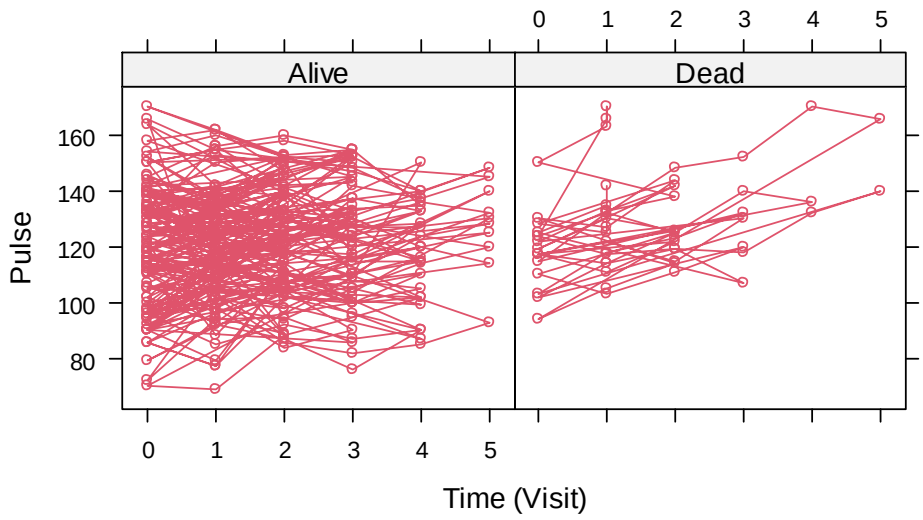

Figure 3. *Profile of longitudinal pulse rate data over observation or visit time.*

3.2. Analysis Approach

Linear mixed-effects model is used to the longitudinal outcome measured across time on each individual. Cox proportion hazard (Cox PH) regression model was used to model time-to-event (death) outcome. Indeed, the joint model directly includes parameters of the longitudinal fit in the survival submodel. If the longitudinal submodel does not fit the longitudinal data properly, the joint model will estimate invalid joint coefficients. Therefore, the submodel quality was checked to ensure that the joint model gives a reliable estimation.

3.3. Model Selection

Before estimating the joint model, we perform initial model selection steps independently for the longitudinal and the time-to-event data.

3.3.1. Longitudinal Sub-Model Selection

First, model for the longitudinal data is selected. Linear mixed-effects model was used and test for six different models. The first model is intercept and the linear slope model, the second is quadratic slope model, the third is cubic slope model, and the fourth to sixth is spline effects of time with different numbers of nodes. To do so, models are fitted and compared them on the basis of their Bayesian Information Criterion (BIC), where smaller values are preferable [32].

Table 1. *Random effect model selection for longitudinal submodels.*

Models	BIC
Polynomial of first order	1166.142
Polynomial of second order	1170.313
Polynomial of third order	1175.104
splines with nodes 2	1170.505
splines with nodes 3	1174.846
splines with nodes 4	1181.712

Based the result Table 1, we retain sub-model Polynomial of first order, which will be joined to the upcoming time-to-event sub-model in the final joint model.

3.3.2. Survival Submodel Selection

An optimal time-to-event submodel was selected. To do so, Cox PH model [23] was selected that relates the time and occurrence of death. A variables selection is done, and for each variable the PH assumption was checked. Analysis of these data showed that the PH assumption was met for selected variables as in Table 2.

Table 2. *Test of proportional hazard assumption.*

Variables	Chisquare	p-value
Age	0.129	0.720
Nutrition: under weight	2.364	0.271
CD: Congenital	1.762	0.543
Corrective surgery: Yes	0.086	0.770
Pulmonary: Yes	1.342	0.452
Global	2.136	0.910

Table 2 shown the global test of proportional hazard assumption, the test is not statistically significant for each of the covariates, and the global test is also not statistically significant. Therefore, a proportional hazard was assumed [33].

3.3.3. Joint Model

Finally, the two submodels are combined in joint modeling. Various association structures were considered and compared in the estimated joint models. To do so, the Deviance Information Criterion (DIC) was used, with smaller values indicating better model adjustments to the data. Three different association structures were considered and, again, use a model comparison approach to select the final model.

Shared Random Effect Association

The joint model revealed a significant association between pulse rate trajectory and survival outcome. Both subject-specific baseline pulse rate $(\gamma_{b_{0i}} = 0.065, \mathrm{p} < 0.001)$ and the rate of change in pulse rate over time $(\gamma_{b_{0i}} = 0.332, \mathrm{p} = 0.003))$ were positively associated with the hazard of death. This indicates that patients with higher initial pulse rates and those experiencing a more rapid increase in pulse rate over time are at significantly greater risk of mortality (Table 3).

Table 3. *Joint model with shared random effect association parameter.*

Covariates	Longitudinal process			
	Value	**Std. Dev**	**95% CI**	**Sign.**
Intercept	116.708	1.893	(111.11, 122.31)	<0.001
Obs.time	1.858	0.227	(1.400, 2.298)	<0.001
Age	-1.216	0.222	(-1.634, -0.775)	<0.001
CD: Congenital	3.121	2.232	(-1.092, 7.632)	0.153
Nutrition: Under weight	4.646	1.837	(1.072, 8.212)	0.012
Anemia: Yes	2.304	1.950	(-1.526, 6.084)	0.235
	Event Process			
Age	-0.008	0.048	(-0.112, 0.084)	0.873
Nutrition: Under weight	1.897	0.682	(0.698, 3.410)	<0.001
CD: Congenital	0.961	0.617	(-0.121, 2.224)	0.085
Surgery: Yes	-0.124	0.768	(-0.724, -0.248)	0.004
Pulmonary: Yes	1.343	0.416	(0.497, 2.160)	0.003
$\gamma_{b_{0i}}$	0.065	0.018	(0.031, 0.105)	<0.001
$\gamma_{b_{1i}}$	0.3321	0.139	(0.097, 0.622)	0.003

In the longitudinal submodel, pulse rate increased significantly over time $(\beta = 1.858, p < 0.001)$ and was higher among underweight patients (β = 4.646, p = 0.012), while older age was associated with lower pulse rate $(\beta = -1.216, p < 0.001)$. In the survival submodel, underweight status $(\beta = 1.897, p < 0.001)$ and pulmonary complications $(\beta = 1.343, p = 0.003)$ significantly increased the hazard of death, whereas undergoing surgery significantly reduced mortality risk $(\beta = -0.124, p = 0.004)$ as give in Table 3.

Current Value Association

The joint model with current value association revealed a significant relationship between the longitudinal pulse rate and survival outcome. The association parameter $(\gamma 1 = 0.028, p = 0.003)$ indicates that higher current values of pulse rate are associated with an increased hazard of death. This suggests that patients experiencing elevated pulse rates at a given time point are at significantly greater risk of mortality (Table 4).

Table 4. Joint model with current value association structures.

Covariates	Event process			
	Mean	StDev	95% CI	Sign.
Age	0.032	0.067	(-0.100, 0.157)	0.633
Nutrition: Under weight	1.864	0.698	(0.593, 3.375)	0.002
CD: Congenital	0.944	0.637	(-0.262, 2.209)	0.120
Surgery: Yes	-1.506	0.702	(-1.608, -1.396)	0.015
Pulmonary: Yes	1.357	0.392	(0.587, 2.125)	0.002
γ_1	0.028	0.011	(0.007, 0.051)	0.003
	Longitudinal process			
Intercept	126.434	3.063	(120.294,132.398)	0.001
Obs. Time	1.784	0.253	(1.292, 2.287)	0.001
Age	-1.734	0.233	(-2.192, -1.272)	0.001
CD: Congenital	-2.637	2.335	(-7.252, 1.939)	0.262
Nutrition: Under weight	2.820	1.969	(-0.977, 6.740)	0.147
Anemia: Yes	1.012	2.090	(-3.047, 5.069)	0.624

In the survival submodel, underweight status ($\beta = 1.864, p = 0.002$) and pulmonary complications ($\beta = 1.357, p = 0.002$) were significant predictors of increased mortality, whereas surgery was associated with a significant reduction in risk ($\beta = -1.506, p = 0.015$). In the longitudinal submodel, pulse rate increased significantly over time ($\beta = 1.784, p = 0.001$) and decreased with age ($\beta = -1.734, p = 0.001$), while other covariates were not statistically significant (Table 4).

Current Value plus Slope Association

The joint model incorporating both current value and slope association demonstrated that pulse rate is a significant time-dependent predictor of mortality. The current value of pulse rate was positively associated with the hazard of death ($\gamma 1 = 0.027, p = 0.003$), indicating that each unit increase in pulse rate corresponds to a 2.7% increase in mortality risk. Conversely, the slope of pulse rate was negatively associated with the hazard ($\gamma 2 = -0.703, p = 0.007$), suggesting that, conditional on the current pulse rate level, patients with increasing pulse rate trajectories have a lower risk of death compared to those with decreasing or stable trajectories (Table 5).

In the survival submodel, underweight status ($HR \approx 6.09, p = 0.002$) and pulmonary complications ($HR \approx 3.93, p = 0.001$) were associated with increased mortality, while surgery significantly reduced the hazard of death ($HR \approx 0.26, p = 0.025$). The longitudinal submodel indicated that pulse rate increased significantly over time ($\beta = 1.769, p = 0.001$) and decreased with age ($\beta = -1.736, p = 0.001$) as given in Table 5.

Table 5. Joint model with current value and slope association structures.

Covariates	Event process			
	Mean	StDev	95% CI	Sign.
Age	0.033	0.065	(-0.102, 0.152)	0.571
Nutrition: Under weight	1.807	0.664	(0.603, 3.202)	0.002
CD: Congenital	0.915	0.613	(-0.291, 2.152)	0.131

	Event process			
Covariates	**Mean**	**StDev**	**95% CI**	**Sign.**
Surgery: Yes	-1.346	0.680	(-1.727, -0.969)	0.025
Pulmonary: Yes	1.369	0.379	(0.619, 2.097)	0.001
γ_1	0.027	0.010	(0.006, 0.048)	0.003
γ_2	-0.703	0.126	(-1.312, -0.108)	0.007
	Longitudinal process			
Intercept	126.361	1.035	(120.34, 132.22)	0.001
Obs. Time	1.769	0.254	(1.267, 2.262)	0.001
Age	-1.736	0.232	(-2.190, -1.279)	0.001
CD: Congenital	-2.569	0.304	(-7.154, 1.866)	0.267
Nutrition: Under weight	2.833	0.938	(-0.964, 6.606)	0.145
Anemia: Yes	1.081	0.039	(-2.931, 5.053)	0.592

Joint Model Comparison

Finally, association structures of the estimated joint models were compared. To do so, the Deviance Information Criterion (DIC) was used, with smaller values indicating better model adjustments to the data [33].

Table 6. *model comparison and selection of joint model.*

Joint Models	**LPML**	**DIC**	**pD**
Shared random effect	-7821.416	15493.36	639.1473
Current value	-7832.877	15506.92	642.3408
Current value + slope	-7816.790	15482.36	639.0599

Based on the DIC values, the evidence is strong to prefer the joint model with current value and slope association structures, which includes the current value and the current rate of change as predictors of the risk of death (Table 6). Which indicate the current true value and the current rate of change of pulse rate as predictors of the risk of death. Recall that within this model, the effect of the current true value in pulse rate on the hazard of dying was not different from zero, whereas the association between the rate of change in speed and survival was strong (Table 6).

Convergence of the Bayesian joint model was assessed using trace plots, autocorrelation functions, and posterior density plots (Figure 4 and Figure 5). The trace plots exhibited good mixing with no apparent trends, indicating stationarity of the chains. The autocorrelation functions showed rapid decay, suggesting low serial correlation and efficient sampling. Additionally, the posterior density plots were smooth and unimodal. Overall, these diagnostics confirm that the Markov chains converged satisfactorily and that the parameter estimates are reliable.

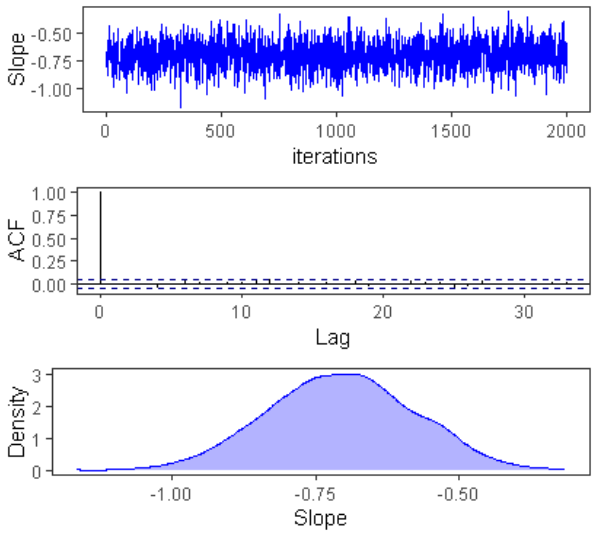

Figure 4. *convergence assessment for current value in joint model.*

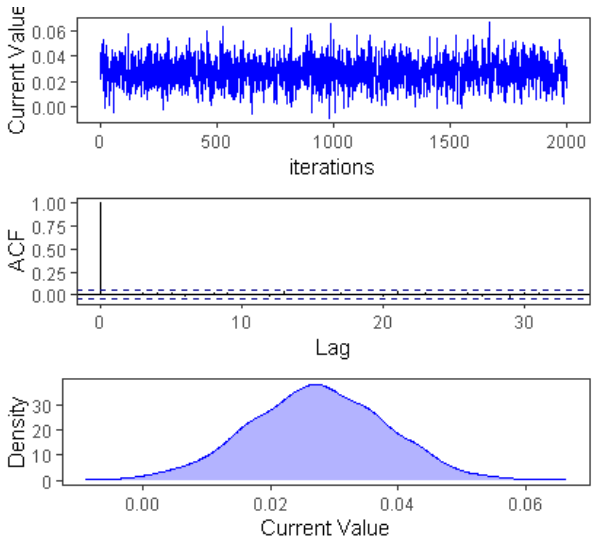

Figure 5. *convergence assessment for rate change (slope) in joint model.*

4. Conclusion

The present study underscores the methodological superiority and practical utility of the Bayesian joint modeling framework in clinical research where longitudinal biomarkers and survival outcomes are interdependent. By analyzing a cohort of cardiac patients, the researcher demonstrated that treating these processes separately ignores critical endogeneity, whereas joint models provide a unified probabilistic approach that yields more efficient and less biased estimates.

Our findings reveal that the Current Value plus Slope association structure offers the most robust fit, indicating that the instantaneous rate of change in pulse rate rather than just its absolute value is a critical, time-dependent predictor of cardiac mortality. These results underscore the necessity of moving toward personalized, dynamic risk prediction in clinical practice.

Pulse rate acts as a significant time-dependent predictor of mortality. Furthermore, factors such as underweight status and pulmonary hypertension significantly elevate the hazard of death, while corrective surgery remains a vital protective factor against mortality.

Ultimately, this research bridges the gap between complex statistical theory and clinical application. We recommend the wider adoption of joint modeling in medical and psychological research to better utilize rich longitudinal datasets and improve the accuracy of health outcome predictions.

Abbreviations

BIC	Bayesian Information Criterion
HR	Hazard Ratio
CD	Cardiac Disease
DIC	Deviance Information Criterion
CI	Confidence Interval
StDev	Standard Deviation

Author Contributions

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Conflicts of Interest

The author declares no conflict of interests.

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