RESEARCH PAPER

Bayesian Fixed-Effect Interaction Modelling for Comparative Assessment of two Dental Treatments

Pranjal Kumar Pandey[1*], Akanksha Gupta[2], Farhan Durrani[3], Abhishek Pathak[4], S.K Upadhyay[5]

[1]Department of Community Medicine, Heritage Institute of Medical Sciences, Varanasi 221311, Uttar Pradesh, India*
[2] Department of Statistics, Banaras Hindu University, Varanasi 221005, Uttar Pradesh, India
[3] Department of Dental Sciences, Banaras Hindu University, Varanasi 221005, Uttar Pradesh, India
[4] Department of Neurology, Banaras Hindu University, Varanasi 221005, Uttar Pradesh, India
[5]Department of Statistics, Banaras Hindu University, Varanasi 221005, Uttar Pradesh, India

***Corresponding Author**: Pranjal Kumar Pandey , Department of Community Medicine, Heritage Institute of Medical Sciences, Varanasi 221311, Uttar Pradesh, India, Email: pranjal998@gmail.com*

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ABSTRACT

The present study comparatively evaluates the efficacy of two dental treatment modalities, namely autogenous xenograft particulate grafts with a Bio-Gide membrane (AGB) and autogenous particulate grafts with a Ti-PTFE membrane (ATi), with respect to vertical bone gain and keratinised tissue width gain. Vertical bone loss and keratinised tissue width loss, along with their corresponding gain outcomes, were analysed using a Bayesian fixed effect interaction regression model with noninformative priors to assess the treatment effects. Posterior inference was performed using the Metropolis algorithm due to the analytical intractability of the resulting posterior distribution. The results demonstrate that ATi consistently provides superior outcomes compared with AGB, with greater improvements observed in both vertical bone gain and keratinised tissue width gain. These findings indicate that ATi is more effective than AGB in improving key clinical outcomes and highlight the utility of Bayesian regression as a robust framework for comparative treatment evaluation. The findings further support the clinical preference for ATi in enhancing bone and soft tissue outcomes and reinforce the role of Bayesian modelling in evidence-based decision-making in dentistry.

Keywords: Dental treatment, Vertical bone loss, Keratin tissue width loss, Regression, Interaction effect

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1. INTRODUCTION

Disease, whether minor or severe, is a common phenomenon that one faces in daily life, and can be mitigated through different treatments, provided it is curable. In healthcare, evaluating the efficacy of the treatments and comparing them plays a key role in deciding the best course of action for the patients. The comparative studies not only allow researchers to analyse the efficacy, safety, and cost-effectiveness of various treatment options, but also guarantee that the patients receive outstanding care specific to their personal requirements. It is also aimed at assisting healthcare providers to improve patient outcomes through evidence-based decision-making. Moreover, the comparison of two treatments enables the detection of potential side effects or adverse reactions related to each option, ultimately working towards reducing risks and enhancing benefits for the patients.

Additionally, understanding and examining the specific characteristics of a disease such as its genetic, molecular, and observational features is

crucial in creating tailored therapeutic approaches that improve treatment success rates while decreasing harmful side effects. For instance, individual differences in inflammatory response and composition of oral bacteria play a crucial role in determining the course of periodontal disease and how well it responds to treatment (*Abusleme et al. 2021*). Likewise, when it comes to managing diabetes, comprehending the genetic foundations of monogenic types such as MODY (Maturity Onset Diabetes of the Young) allows clinicians to categorise individuals into separate groups depending on their unique genetic changes (*Dzhemileva et al. 2025*). Accounting these characteristics in treatment planning not only enhances the precision of medical interventions but also adheres to the principles of personalised medicine, which aims to customise healthcare based on each patient's distinct characteristics. By embracing this method, healthcare could undergo a revolutionary shift towards more impactful, streamlined, and patient-focussed treatment.

The literature offers a wide spectrum of statistical procedures for situations in which the central aim is to compare outcomes across two or more treatment groups. Among these, the t-test, analysis of variance (ANOVA), and its extended form, analysis of covariance (ANCOVA), etc. constitute the most frequently employed techniques (see (*Montgomery and Runger 2019*) and (*Keselman et al. 1998*), etc.). These methods primarily assess whether observable differences in group means are statistically meaningful. However, data arising from real-world applications often display considerable structural complexity, where multiple predictors simultaneously influence the outcome and simple group-comparison frameworks become insufficient to capture such relationships.

To address such multifaceted settings, researchers increasingly rely on regression-based methodologies. Regression models facilitate the examination of how individual variables, as well as their combined effects, shape the outcome of interest. In particular, fixed-effect regression models incorporating interaction terms are widely applied when the objective is to explore the influence of a specific predictor or to understand how two variables jointly contribute to variations in the response (see (*Bai 2009*), (*Bolker et al. 2009*), (*Hardin and Hilbe 2013*), etc.). Thus, it offers a more comprehensive analytical perspective compared to traditional group-comparison techniques. Fixed effect model considers individual-specific effect that remain constant over time, such as individual attributes or unobserved heterogeneity. The usefulness of these models lies in their ability to control for unobservable individual factors that may skew estimation findings. By including fixed effect, one can better understand the relationship between the variables and isolate the specific effects of interest (*Allison 2009*). Yet, with inclusion of interaction terms, one has the ability to study how a variable effect varies based on the level or existence of another variable. It proves to be exceptionally beneficial in investigating how the relationship between variables can be influenced or adjusted by various external circumstances using interaction terms (see, for example, (*Siemsen, Roth, and Oliveira 2010*)). The present article attempts to utilise the fixed effect linear regression model with interaction terms for comparing two types of treatment procedures that are used to restore the depleted areas of dental bones. As the dataset analysed in this article is based on dental treatments, it is important to shed some light on the treatment procedures involved. An attempt has been made to explain both the procedures without making them too complex for the general audience.

With advancements in dental science, guided bone regeneration (GBR) has evolved into a fundamental method for restoring the depleted areas of bone, especially while planning for dental implant surgeries. Although a number of procedures can be found in the literature, a discussion on the same is beyond the scope of this work. Interested readers can, however, refer to (*Georgeanu et al. 2023*), where the authors have conducted a comprehensive literature-based analysis of current bone grafting materials used in dentistry, evaluating their biological properties, clinical applications, and limitations. We shall, however, restrict our study to two cutting-edge methods that are very popular among practitioners. The first method integrates natural bone from the patient and specially processed animal bone, paired with a protective Bio-gide membrane (see (*Abou Fadel, Samarani, and Chakar 2018*)). Made of collagen, this membrane supports the growth of new bone by shielding the healing area and gradually breaks down over time. The whole treatment is termed as Autogenous Xenograft Particulate Grafts with Bio-gide Membrane (AGB). Another treatment procedure called the Autogenous Particulate Graft with Ti-PTFE membrane (ATi), utilises tiny portions of the patient's bone mixed with a specialized non-absorbable membrane strengthened with titanium, commonly known as a Ti-PTFE membrane (see (*Cucchi et al. 2019*)). This sturdy titanium barrier maintains stability in the healing space and blocks soft tissue from hindering the growth of new bone.

It may be noted that the fixed-effect linear regression models that include interaction terms

have been extensively studied using both classical and Bayesian approaches across a wide range of application domains. Their application in the analysis of data related to dental research, however, remains relatively limited, particularly within the Bayesian framework. Existing studies in dentistry primarily rely on classical methods and are largely confined to longitudinal or panel data settings, although a few references have addressed more general analytical contexts (see, for example, (*Jain et al. 2016*), (*Huang et al. 2017*), and (*Matsuyama 2024*), etc.). Despite this restricted scope, Bayesian regression?based techniques have demonstrated considerable promise in dental research. Notable references include the studies by (*Gilthorpe, Maddick, and Petrie 2001*), (*Braga et al. 2005*), and (*Workie, Azene, and Dessie 2019*), among others, which illustrate the advantages of Bayesian methods in offering flexible modelling strategies and a principled approach to uncertainty quantification. Nevertheless, a closer examination of such contributions reveals several methodological and practical limitations. In particular, the systematic incorporation of interaction effects within Bayesian regression models has received very little attention in dental applications.

Against this backdrop, the present article employs a Bayesian linear regression framework incorporating interaction terms to analyse a dental dataset. In the absence of adequate prior knowledge and with the intention of allowing inferences to be driven primarily by the observed data, noninformative prior distributions are adopted for the model parameters. This modelling strategy is designed to more effectively capture the joint influence of covariates and to enhance the interpretability of the complex relationships inherent in dental data.

The rest of the article is structured as under. Section *2* outlines the structure of the dataset and details the specification of the statistical model within the Bayesian framework given in a separate subsection. Section *3* presents the application using real-world data to demonstrate the implementation and practical relevance of the proposed model. Finally, Section *4* concludes the study by summarising the main results and highlighting the key insights drawn from the analysis.

2. DATA STRUCTURE AND MODEL FORMULATION

Before introducing the model formulation, it is useful to first describe the typical structure of a dataset often encountered in some practical applications. As discussed earlier in the previous section, a disease often affects multiple characteristics, and effective recovery requires addressing all associated impairments. Let us assume that there are n individuals diagnosed with a particular disease. Consider a setting in which two distinct treatment procedures are available, each aimed at improving impairments in two specific disease-related characteristics. Each individual receives only one of these treatments, which naturally provides a further classification in the individuals. In most cases, these characteristics are measured on a quantitative scale. Table *1* summarises the level of impairment before treatment and the extent of recovery after treatment, recorded as continuous measurements (say, PIM and PTM) at certain fixed time point. The dataset also includes a variable labelled “Group", which is a binary indicator identifying the treatment assignment. This indicator takes the value 0 for individuals receiving treatment (say) AGB and 1 for those receiving treatment (say) ATi. Finally, the two disease characteristics under consideration are indexed by $C=c_1,c_2$.

Table 1: Pre- and Post-treatment measurements for the characteristics c_1 and c_2 at certain fixed time point

Characteristic c_1		**Characteristic c_2**		**Group**
PIM	PTM	PIM	PTM	
$F_1^{c_1}$	$M_1^{c_1}$	$F_1^{c_2}$	$M_1^{c_2}$	g_1
⋮	⋮	⋮	⋮	⋮
$F_n^{c_1}$	$M_n^{c_1}$	$F_n^{c_2}$	$M_n^{c_2}$	g_n

As presented in Table *1*, the columns are arranged under the headings characteristic c_1 and c_2 to indicate the specific characteristic to which each set of measurements belongs. The first two columns grouped under characteristic c_1 contain the observations $F_i^{c_1}$; i=1…,n and $M_i^{c_1}$; i=1…,n, which represent the PIMs and PTMs associated with the first characteristic for n individuals. Likewise, the subsequent two columns under charcateristic c_2 include the observations $F_i^{c_2}$; i=1…,n and $M_i^{c_2}$; i=1…,n, corresponding to the measurements for n

individuals for the second characteristic c_2. This structured layout distinctly separates the data for c_1 and c_2, allowing the PIM and PTM measurements to be interpreted accurately within the context of the characteristic to which they belong.

2.1. Bayesian Fixed Effect Regression Model with Interaction

As outlined in the previous discussion, the level of impairment for both characteristics c_1 and c_2 was quantified for each individual at a specific time point, both prior to and following the treatment. Moreover, in the fixed-effects framework, the effects of the covariates are assumed to be constant. The primary objective of this approach is to identify the influence of the explanatory variables included in the analysis, rather than to generalize the results beyond the study sample. Let M_i denote the response variable representing the PTM for individual i at a given time point, F_i represent the corresponding PIM value, and g_i indicate the treatment group to which the individual i belongs, i=1,2,…,n. With these presumptions, the fixed-effects linear regression model for each characteristic can be formulated separately as under.

$$M_i^{c1}=\beta_0^{c1}+\beta_1^{c1}F_i^{c1}+\beta_2^{c1}g_i+\epsilon_i^{c1};\ i=1,2,\ldots,n \qquad (1)$$

$$M_i^{c2}=\beta_0^{c2}+\beta_1^{c2}F_i^{c2}+\beta_2^{c2}g_i+\epsilon_i^{c2};\ i=1,2,\ldots,n \qquad (2)$$

where the intercept β_0^{c1} represents the fixed effect, common across all the observations, assuming the covariates F_i^{c1} to be zero. β_1^{c1} indicates the influence of covariate F_i^{c1} on outcome variable M_i^{c1}. The coefficient β_2^{c1} signifies the impact of covariate g_i on M_i^{c1} and ϵ_i^{c1} is the error term which is assumed to be independent and normally distributed. Within this study, β_0^{c1} represents the baseline PTM when there is no impairment and no treatment involved. β_1^{c1} specifies how much the PIM impacts the PTM and β_2^{c1} denotes the effect of treatment group on PTM. In the model presented in *1*, the regression coefficients β_0^{c1}, β_1^{c1}, and β_2^{c1} appear with the superscript c_1. This notation conveys that each coefficient is associated with, and estimated specifically for, the characteristic c_1. A similar interpretation holds for the coefficients in the model given in *2*, where the superscript c_2 denotes that the respective coefficients and their estimates correspond to the characteristic c_2.

Next, let us assume that one wants to observe the effect of treatment group (g_i) on PTM (M_i) given the covariate PIM (F_i). While the usual linear regression model can assess the individual effects, an interaction term, that is the product of the covariates ($F_i \cdot g_i$) must be incorporated for evaluating the joint effect occurring due to them. As such, the role of the interaction term showcasing the interactive effect of these variables becomes crucial. Furthermore, a model incorporating an interaction effect combines multiple predictors showcasing how the association between a predictor and the outcome may change given another such variable. Thus, the usual interaction effect model for both the characteristics c_1 and c_2 can be expressed separately as

$$M_i^{c1}=\beta_0^{c1}+\beta_3^{c1}F_i^{c1}\cdot g_i+\epsilon_i^{c1};\ i=1,2,\ldots,n \qquad (3)$$

$$M_i^{c2}=\beta_0^{c2}+\beta_3^{c2}F_i^{c2}\cdot g_i+\epsilon_i^{c2};\ i=1,2,\ldots,n \qquad (4)$$

where β_0^{c1} is the intercept term and the interaction coefficient β_3^{c1} in the model *3* reveals how the relationship between g_i and M_i^{c1} varies given F_i^{c1}. This means that β_3^{c1} denotes how the change in treatment group will affect the PTM, given that PIM is observed for that i^{th} individual for the characteristic c_1. In a similar fashion, coefficients given in the model *4* can be interpreted for the characteristic c_2.

Merging the formulations presented in *1* and *3*, as well as those in *2* and *4*, leads to the construction of two revised models. These models jointly capture the fixed effects and their corresponding interaction effects within a single analytical expression, which can be written as under.

$$M_i^{c1}=\beta_0^{c1}+\beta_1^{c1}F_i^{c1}+\beta_2^{c1}g_i+\beta_3^{c1}F_i^{c1}\cdot g_i+\epsilon_i;\ i=1,2,\ldots,n \qquad (5)$$

$$M_i^{c2}=\beta_0^{c2}+\beta_1^{c2}F_i^{c2}+\beta_2^{c2}g_i+\beta_3^{c2}F_i^{c2}\cdot g_i+\epsilon_i;\ i=1,2,\ldots,n \qquad (6)$$

where the coefficients $\beta_0^{c_1},\beta_1^{c_1},\beta_2^{c_1},\beta_3^{c_1},\beta_0^{c_2},\beta_1^{c_2},\beta_2^{c_2}$ and $\beta_3^{c_2}$ retain the same interpretations as defined in the models *1* to *6* corresponding to the characteristics c_1 and c_2, respectively.

Accounting for the assumption that errors are independent and normally distributed, the likelihood function can be written separately for the models given in *5* and *6*. So the likelihood functions corresponding to the characteristics c_1 and c_2 are given as

$$L^{c_1}=\prod_{i=1}^{n}\frac{1}{\sqrt{2\pi(\sigma^{c_1})^2}}\exp\left[-\frac{1}{2(\sigma^{c_1})^2}\left(M_i^{c_1}-\beta_0^{c_1}-\beta_1^{c_1}F_i^{c_1}-\beta_2^{c_1}g_i-\beta_3^{c_1}F_i^{c_1}\cdot g_i\right)^2\right], \qquad (7)$$

and

$$L^{c_2}=\prod_{i=1}^{n}\frac{1}{\sqrt{2\pi(\sigma^{c_2})^2}}\exp\left[-\frac{1}{2(\sigma^{c_2})^2}\left(M_i^{c_2}-\beta_0^{c_2}-\beta_1^{c_2}F_i^{c_2}-\beta_2^{c_2}g_i-\beta_3^{c_2}F_i^{c_2}\cdot g_i\right)^2\right], \qquad (8)$$

respectively. In this study, the two characteristics, c_1 and c_2, represent two distinct outcome parameters that are obtained independently for each subject. The measurements corresponding to the two characteristics are recorded separately and do not influence one another at the time of observation. Owing to this measurement independence, the corresponding random variables $M_i^{c_1}$ and $M_i^{c_2}$ are treated as statistically independent for each individual, i, i=1,2,…,n. Because the two characteristics are independent, and their measurements are recorded separately, the contribution of the likelihood from one characteristic does not affect the other. Therefore, the overall likelihood for the dataset is obtained by taking the product of the likelihood functions corresponding to the characteristics c_1 and c_2. Thus, one can write.

$$\begin{aligned}L \;=&\prod_{i=1}^{n}\frac{1}{\sqrt{2\pi(\sigma^{c_1})^2}}\exp\left\{-\frac{1}{2(\sigma^{c_1})^2}(M_i^{c_1}-\beta_0^{c_1}-\beta_1^{c_1}F_i^{c_1}-\beta_2^{c_1}g_i-\beta_3^{c_1}F_i^{c_1}\cdot g_i)^2\right\}\\ &\times\prod_{i=1}^{n}\frac{1}{\sqrt{2\pi(\sigma^{c_2})^2}}\exp\left\{-\frac{1}{2(\sigma^{c_2})^2}(M_i^{c_2}-\beta_0^{c_2}-\beta_1^{c_2}F_i^{c_2}-\beta_2^{c_2}g_i-\beta_3^{c_2}F_i^{c_2}\cdot g_i)^2\right\}.\end{aligned} \qquad (9)$$

Once the likelihood function is formulated, the subsequent step is to choose appropriate prior distributions for the model parameters. The literature offers a wide range of approaches for prior selection. When prior knowledge about the parameters is available, it is natural to adopt informative priors, which may be constructed or adjusted through subjective elicitation based on existing information. For instance, after selecting a suitable prior family, its hyperparameters can be determined using expert knowledge or historical evidence. On the other hand, when such prior information is limited or unavailable, non-informative or weakly informative priors may be employed so that the resulting inference is driven primarily by the observed data, with minimal influence from the prior (see, for example, (*Chesley 1975*), (*Datta and Ghosh 1995*), (*P. K. Pandey et al. 2024*) and (*P. Pandey et al. 2025*).

In this article, the model parameters are assumed to be mutually independent *a priori*. Accordingly, normal prior distributions with zero mean and large variances are assigned to all regression coefficients, including the intercept. The use of large variances yields proper yet weakly informative priors, reflecting limited prior knowledge about the regression effects. The zero-mean specification further implies prior symmetry, assigning equal probability mass to positive and negative values of each coefficient. For the error variance components, $(\sigma^{c_1})^2$ and $(\sigma^{c_2})^2$, Jeffreys type priors are adopted as the non-informative choices. The theoretical motivation and practical usefulness of Jeffreys priors are well documented in the Bayesian literature (see, for example, (*Box and Tiao 1973*), (*Bernardo and Smith 2000*), and (*Gelman et al. 2013*), among others). Thus, the considered priors are

$$\begin{aligned}&\beta_l^c\sim N\left(0,\sigma_l^2\right);\ l=0,\ldots,3\ ;\ c=c_1,c_2\\ &\pi(\sigma^c)\propto\frac{1}{(\sigma^c)^2};\ c=c_1,c_2\end{aligned} \qquad (10)$$

Now combining the likelihood function given in *9* with the priors in *10* using Bayes theorem, yields the posterior distribution, specified up to proportionality, as

$$
\begin{aligned}
&p(\beta_0^{c_1},\beta_1^{c_1},\beta_2^{c_1},\beta_3^{c_1},\beta_0^{c_2},\beta_1^{c_2},\beta_2^{c_2},\beta_3^{c_2},(\sigma^{c_1})^2,(\sigma^{c_2})^2 \mid M_i^{c_1},M_i^{c_2},F_i^{c_1},F_i^{c_2},g_i) \\
&\propto \prod_{i=1}^{n} \frac{1}{\sqrt{2\pi(\sigma^{c_1})^2}} \exp\left\{-\frac{\left(M_i^{c_1}-\beta_0^{c_1}-\beta_1^{c_1}F_i^{c_1}-\beta_2^{c_1}g_i-\beta_3^{c_1}F_i^{c_1}\cdot g_i\right)^2}{2(\sigma^{c_1})^2}\right\} \\
&\times \prod_{i=1}^{n} \frac{1}{\sqrt{2\pi(\sigma^{c_2})^2}} \exp\left\{-\frac{\left(M_i^{c_2}-\beta_0^{c_2}-\beta_1^{c_2}F_i^{c_2}-\beta_2^{c_2}g_i-\beta_3^{c_2}F_i^{c_2}\cdot g_i\right)^2}{2(\sigma^{c_2})^2}\right\} \\
&\times \exp\left\{-\frac{\left(\beta_0^{c_1}\right)^2+\left(\beta_1^{c_1}\right)^2+\left(\beta_2^{c_1}\right)^2+\left(\beta_3^{c_1}\right)^2+\left(\beta_0^{c_2}\right)^2+\left(\beta_1^{c_2}\right)^2+\left(\beta_2^{c_2}\right)^2+\left(\beta_3^{c_2}\right)^2}{2\sigma^2}\right\} \\
&\times \frac{1}{(\sigma^{c_1})^2} \times \frac{1}{(\sigma^{c_2})^2} \quad .
\end{aligned} \tag{11}
$$

The posterior distribution given in *11* is analytically intractable and therefore cannot be evaluated in closed form. As a result, posterior samples are generated using the Metropolis algorithm, which are then used to obtain the required sample-based inferences. A comprehensive discussion of this algorithm has been provided below.

Acknowledged as a pivotal advancement within computational science, the Metropolis algorithm introduced by (*Metropolis et al. 1953*) plays a vital role in stochastic simulation methods used prominently in statistical mechanics and Bayesian inference. It forms a Markov chain with the intention of approaching a particular target distribution by iteratively proposing transitions between states. At every iteration, a candidate state is formed from a symmetric proposal distribution and its acceptance is based on the ratio of target distribution values at both the current and proposed states. Its refinement shines through in its adeptness at estimating intricate, high-dimensional distributions without the need for knowledge of the normalizing constant (*Upadhyay, Vasishta, and Smith 2001*). Steps for implementing the Metropolis algorithm are as follows:

1. Choose an initial state x_0 from the domain of the target distribution. This initial value may be arbitrary or informed by prior knowledge.

2. At iteration p, generate a proposed state x' from a symmetric proposal distribution $q(x'|x_p)$. Common choices include Gaussian or uniform distributions centered at the current state x_p.

3. Compute the acceptance probability α as:

$$\alpha=\min\left(1,\frac{\pi(x')}{\pi(x_p)}\right) \tag{12}$$

where $\pi(\cdot)$ denotes the target distribution at x' and x_p.

4. Draw a uniform random number $u\sim U(0,1)$. If $u\leq\alpha$, accept the proposal and set $x_{p+1}=x'$. Otherwise, reject the proposal and retain the current state: $x_{p+1}=x_p$.

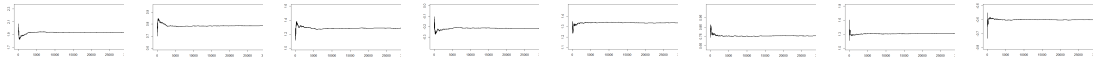
3. NUMERICAL ILLUSTRATION

This section demonstrates the Bayesian modelling framework described in the previous section using an actual clinical dataset obtained from dental patients at the Department of Dental Sciences, Banaras Hindu University. The dataset consists of observations from 22 patients and focusses on the measurements of two specific characteristics, namely, vertical bone loss (VBL) and keratinised tissue width loss (KTL), during the dental implant procedure. In implant therapy, vertical bone gain (VBG) is achieved by augmenting the jawbone to enhance implant stability and long-term success. Similarly, keratinised tissue width gain (KTG) refers to the surgical expansion of the resilient gum tissue surrounding teeth and implants. For each patient, VBL and KTL were recorded as PIMs. Two alternative treatment strategies were subsequently administered, namely, ATi and AGB (see Section *1*). Based on the treatment received, patients were equally allocated into two groups (Group 0 and Group 1) of size 11 each. The ATi group serves as the control group, with the group indicator g_i=0, while patients treated with AGB constitute the case group, with g_i=1. After that, VBG and KTG were recorded as PTMs after a follow-up period of five years. The above dataset is then incorporated into the model specification given in *9*, with observations from n=22 individuals for both the characteristics. Weakly informative normal priors are assigned to all the regression parameters in the model, each with a mean zero and variance 900. Large and equal variance is taken for the normal prior of each regression coefficient simply for the ease of implementation,

although different values of large variances in the weakly informative priors are not going to have any considerable impact on the final results.

To proceed with the Bayesian implementation, the Metropolis algorithm was employed for generating samples of the model parameters from the corresponding posterior distribution in *11*. *MVN* kernel as the candidate generating density was considered with the mean vector of the normal distribution adjusted to match the ML estimates of the parameters, while the variance-covariance matrix was calculated using a Hessian-based approximation calculated at the ML estimates. To ensure a relatively high acceptance probability, a scaling constant c_s was taken into account as a multiplier of the normal standard deviation (see also Section *3*). Convergence was monitored based on ergodic averages at about 5K iterations with a value of c_s as 0.5 and, consequently, the ergodic average plots corresponding to each regression coefficient is presented in Figure *1*.

Convergence diagnostic plots, based on the ergodic averages, shown in Figure *1* clearly demonstrate the perfect convergence for the variates under consideration. Upon establishing convergence, a total of 1,000 posterior samples were extracted by retaining every 10^{th} iteration. The inferential results based on these samples are presented in Table *2* in the form of estimated posterior means, SDs, and 0.95 HI for each regression parameter. These estimates are given for illustration only. One can, of course, consider other posterior characteristics of interest from the finally generated sample of size 1000.



(a): β_0^{c1} (b): β_1^{c1} (c): β_2^{c1} (d): β_3^{c1} (e): β_0^{c2} (f): β_1^{c2} (g): β_2^{c2} (h): β_3^{c2}

Figure 1: Convergence diagnostic plots based on ergodic averages for various regression coefficients.

Table 2: Some of the sample based posterior characteristics for various regression coefficients

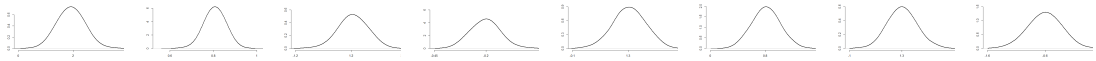
Coefficients	Posterior mean	Posterior SD	0.95 HI
β_0^{c1}	1.94	0.22	(1.31, 2.58)
β_1^{c1}	0.81	0.03	(0.72, 0.88)
β_2^{c1}	1.26	0.42	(0.06, 2.49)
β_3^{c1}	-0.21	0.06	(-0.37, -0.04)
β_0^{c2}	1.34	0.16	(0.88, 1.80)
β_1^{c2}	0.75	0.22	(0.65, 1.28)
β_2^{c2}	1.28	0.42	(0.06, 2.48)
β_3^{c2}	-0.59	0.10	(-0.83, -0.31)

For characteristic c_1 , which examines the influence of VBL on VBG, the intercept parameter β_0^{c1} represents the baseline level of VBG when VBL is absent and no treatment effect is applied. The coefficient β_1^{c1} captures the direct contribution of VBL and suggests that increase in VBL is accompanied by corresponding increases in VBG. The group effect reflected in β_2^{c1} further indicates that, in the absence of VBL, transitioning from group 0 to group 1 leads to an elevation in VBG. In contrast, the interaction term β_3^{c1} demonstrates that when VBL is present, shifting from treatment ATi to AGB results in reduction in VBG.

A similar interpretation applies to characteristic c_2, which examines the relationship between KTL and KTG. The intercept parameter β_0^{c2} represents the expected level of KTG when KTL is zero and treatment effects are not considered. The regression coefficient β_1^{c2} shows that an increase in KTL is associated with an increase in KTG, indicating a positive association between the two, that is, greater tissue loss corresponds to higher tissue gain. The term β_2^{c2} suggests that, in the absence of KTL, movement from group 0 to group 1 produces an increase in KTG. However, the interaction coefficient β_3^{c2} indicates that in the presence of KTL, transitioning from ATi to AGB leads to a decrease in KTG, consistent with the pattern observed for VBL and VBG scenario. Collectively, the findings show that in the presence of VBL or KTL, changing the treatment from ATi to AGB is associated with lower values

of the respective outcomes, VBG and KTG. This recurring trend indicates that ATi yields more favourable improvements in the outcome measures compared with AGB.

Furthermore, Figure *2* present the kernel density estimates of the marginal posterior distributions corresponding to various regression coefficients associated with VBL and KTL. Examination of these plots reveals that the posterior distributions are quite close to symmetry, with no noticeable leaning towards either tail. This near-symmetry indicates that the uncertainty surrounding the parameter estimates is evenly distributed about their most probable values, rather than being biased in a particular direction. Consequently, posterior inferences for these coefficients are more robust and stable. In particular, the near-symmetric form of the posterior distributions supports the use of estimated posterior modes (or means) as meaningful point estimates and justifies reliance on standard summary statistics, facilitating a clear and methodologically sound interpretation of the effects of covariates on the outcome variable of interest.



(a): β_0^{c1} (b): β_1^{c1} (c): β_2^{c1} (d): β_3^{c1} (e): β_0^{c2} (f): β_1^{c2} (g): β_2^{c2} (h): β_3^{c2}

Figure 2: Kernel density estimates for the various regression coefficients associated with VBL and KTL.

4. CONCLUSION

In healthcare research, comparing therapeutic strategies in terms of their clinical effectiveness is central to understanding treatment performance and improving patient outcomes. Rigorous evaluation of competing interventions enables data-driven conclusions that support informed clinical decision-making. In this context, the present article examines a dental dataset in which two treatment strategies, namely ATi and AGB, are compared using two independently recorded clinical characteristics, VBL and KTL, along with their corresponding outcomes, VBG and KTG. The analysis is carried out within a Bayesian framework using a fixed effect interaction regression model, with noninformative prior distributions specified for the model parameters. The results reveal a clear difference in treatment performance, with ATi demonstrating greater effectiveness in improving the outcomes VBG and KTG in comparison to the treatment AGB. These findings underscore the usefulness of considered Bayesian modelling in comparative treatment analysis and provide relevant insights for evidence-based decision making in healthcare settings.

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Declarations

Conflict Of Interest: On behalf of all authors, the corresponding author states that there is no conflict of interest.

Data Availability: It is important to mention that the dataset is not shown in the paper because of its confidentiality and space restrictions, although interested readers may approach one of the co-authors from the Department of Dental Sciences, Banaras Hindu University, for the purpose of academic use only.

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