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Research Article

Bayesian Survival and Mixed-Effects Modeling of Immune Persistence Under COVID-19 Boosting

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Abstract

Post-vaccination immune persistence varies significantly across booster regimens, particularly between homologous (e.g., mRNA/mRNA) and heterologous (e.g., VV/mRNA) strategies. This heterogeneity poses challenges for public health planning and long-term immunity forecasting. In this study, we developed a novel statistical framework integrating Bayesian survival analysis with linear mixed-effects regression to jointly model longitudinal IgG dynamics and time-to-waning of protective immunity in a cohort of 334 individuals—206 previously infected and 128 infection-naïve—from real-world data collected between December 2022 and September 2023. Plasma optical density (OD) values from ELISA assays served as a proxy for anti-SARS-CoV-2 IgG levels. Participants were categorized by booster type (homologous vs. heterologous), prior infection status, and number of doses (2-4). Our integrated model revealed that heterologous boosting was associated with significantly slower IgG decay (hazard ratio HR = 0.62, 95% credible interval [0.48-0.79]) compared to homologous regimens. Moreover, prior SARS-CoV-2 infection independently enhanced both humoral and cellular immune persistence, with infected individuals showing 1.8-fold higher median OD values at 6+ months post-boost. The joint modeling approach successfully captured inter-individual variability through random slopes and intercepts while accounting for censoring in immune waning via a Weibull-based survival component. This framework provides a flexible, predictive tool for evaluating future booster strategies—not only for SARS-CoV-2 but also for other pathogens requiring durable immunity. Our findings support the immunological advantage of heterologous prime-boost schedules, especially when combined with natural infection, and underscore the value of methodological integration in longitudinal immunology research.

Keywords

Bayesian Survival, Mixed-effects Regression, Immune Persistence, Antibody Decay, Heterologous Boosting, COVID-19

1. Introduction

The global rollout of SARS-CoV-2 vaccines has dramatically reduced severe disease and mortality, yet waning immunity—particularly in the face of emerging variants—has necessitated repeated booster doses [1, 2]. While initial vaccine efficacy is well-documented, the durability of protection remains a critical knowledge gap. Humoral immunity, primarily mediated by IgG antibodies, typically peaks weeks after

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vaccination but decays over months [3]. Concurrently, cellular immunity (e.g., T-cell responses) often persists longer, offering a complementary layer of defense [4]. However, the kinetics of immune decay are not uniform; they vary by vaccine platform, dosing interval, infection history, and host factors [5].

These variations also have important epidemiological implications, as vaccine dosing strategies, heterologous booster schedules, and immune correlates of protection can influence the duration and magnitude of population-level protection [33–36].

Most existing studies on post-boost immunity are cross-sectional or rely on descriptive summaries at fixed timepoints [6, 7]. While informative, these approaches cannot model the individual trajectories of immune decay or predict the time until antibody levels fall below a protective threshold. This limitation is particularly acute when comparing homologous (same-platform) versus heterologous (mixed-platform) boosting strategies—a key policy question during global vaccine shortages and variant surges [8].

To address this, we propose a unified statistical framework that merges two powerful methodologies:

1) Bayesian survival analysis, which models the "time-to-event" (here: time until IgG wanes below a protective OD threshold), and
2) Linear mixed-effects models, which capture longitudinal IgG trajectories while accounting for within-subject correlation and inter-individual heterogeneity [9, 10].

This integration is rare in immunological literature but offers distinct advantages: survival models handle right-censored data (e.g., participants still above threshold at last visit), while mixed-effects models borrow strength across individuals to estimate personalized decay rates [11]. Bayesian inference further allows incorporation of prior knowledge and yields full posterior distributions for uncertainty quantification [12].

The antibody half-life ($t_{1/2}$) is a common metric for decay kinetics, defined as:

$$t_{1/2} = \frac{\ln(2)}{\lambda} \tag{1}$$

where λ is the decay rate. However, half-life alone ignores individual variability and censoring—issues our framework resolves.

Similarly, the hazard function for immune waning at time t is:

$$h(t) = \lim_{\Delta t \to 0} \frac{P(t \le T < t + \Delta t | T \ge t)}{\Delta t} \tag{2}$$

which our Bayesian model estimates flexibly using a Weibull baseline.

Our primary aim is to develop and validate this joint modeling approach on real-world data, testing two hypotheses:

Heterologous boosting leads to slower IgG decay than homologous boosting.

Prior SARS-CoV-2 infection enhances both humoral and cellular immune persistence post-boost.

This work contributes methodologically by bridging survival and longitudinal modeling in immunology, and substantively by providing evidence to optimize booster policies in the post-pandemic era.

These variations also have important epidemiological implications, as vaccine dosing strategies, heterologous booster schedules, and immune correlates of protection can influence the duration and magnitude of population-level protection [23, 33–35].

2. Literature Review

2.1. IgG Decay Dynamics (2021-2025)

Early studies established that anti-Spike IgG levels decline exponentially post-vaccination [3]. Reported a median IgG half-life of 60-90 days after mRNA vaccination, with faster decay in older adults. Similar kinetics were observed after adenoviral vector vaccines (e.g., ChAdOx1), though with lower peak titers [13].

Several large observational studies have also documented heterogeneity in vaccine effectiveness and immune durability according to age, vaccine product, time since vaccination, and variant period, particularly during Delta and Omicron waves [37–43].

A simple mono-exponential decay model is often used:

$$IgG(t) = Ae^{-\lambda t} \tag{3}$$

where A is the initial titer and λ the decay constant. However, recent work suggests biphasic decay—an initial rapid drop followed by a slower phase—better fits longitudinal data [14].

Recent studies have further shown that neutralizing antibody levels are strongly associated with protection against symptomatic SARS-CoV-2 infection, although antibody concentrations decline over time and vary according to booster type, prior infection, and population-level vaccine effectiveness [23–31].

Recent studies have further shown that neutralizing antibody levels are strongly associated with protection against symptomatic SARS-CoV-2 infection, although antibody concentrations decline over time and vary according to booster type, prior infection, age, vaccine platform, and variant period [24–31, 37–40, 42, 44–48].

This persistence is biologically plausible because germinal center reactions, T follicular helper-cell responses, memory B-cell maturation, and long-lived plasma-cell formation contribute to durable humoral and cellular immune memory after infection or vaccination [49–56, 58–61].

2.2. T-cell Persistence

While antibodies wane, memory T-cells often persist for >12 months post-infection or vaccination [4, 15]. This persistence is biologically plausible because germinal center reactions, T follicular helper-cell responses, and long-lived plasma-cell maturation contribute to durable humoral and cellular immune memory after infection or vaccination [49–52]. Goel et al. [15] showed that hybrid immunity (infection + vaccination) induces robust, cross-reactive T-cell responses. Notably, T-cell durability appears less affected by variant escape than neutralizing antibodies [16].

Mechanistic immunology studies further indicate that mRNA vaccines activate coordinated adaptive immune responses involving antibody production, memory B cells, CD4+ and CD8+ T-cell responses, and broader cellular immunity, supporting the inclusion of both humoral and cellular indicators in immune-persistence models [59–62].

2.3. Bayesian Survival in Immunology

Bayesian survival models are well-established in oncology and HIV research [17, 18] but underutilized in vaccinology. They excel in small-sample settings and allow hierarchical modeling of covariates (e.g., vaccine type, age). For instance, Ibrahim et al. [17] used Bayesian Weibull models to predict HIV progression, a framework adaptable to immune waning.

2.4. Mixed-Effects Models in Vaccination Studies

Linear mixed-effects models are standard for longitudinal immunogenicity data [19, 20]. Flaxman et al. [20] used them to compare antibody trajectories across ChAdOx1 and BNT162b2, revealing slower decay after mRNA vaccination. These models naturally accommodate random intercepts (baseline differences) and random slopes (decay rate heterogeneity).

2.5. The Integration Gap

Despite their individual strengths, no study has combined Bayesian survival and mixed-effects models to study post-boost immunity. Existing approaches either:

1) Model decay rates ignoring censoring [3], or
2) Use Kaplan-Meier curves without modeling continuous antibody trajectories [21].

Our framework closes this gap by linking the two models through shared random effects, enabling simultaneous inference on population-level decay patterns and individual-level waning risk.

3. Materials & Methods

3.1. Data Description

We analyzed data from 334 adults enrolled in a prospective cohort study (Dec 2022-Sep 2023). Participants were stratified by:

1) Infection status: 206 previously infected (PCR-confirmed), 128 infection-naïve.
2) Booster regimen: Homologous (e.g., mRNA/mRNA) or heterologous (e.g., VV/mRNA), determined from vaccination dates in the master table.
3) Dose count: 2-4 doses (see Table 1).

Outcomes:

1) IgG levels: Plasma OD values from ELISA (higher OD = higher IgG).
2) T-cell status: Binary indicator (PBMC collected = 1, else 0), used as a proxy for cellular immunity assessment.

Covariates:

1) Booster type (homologous = 0, heterologous = 1)
2) Prior infection (no = 0, yes = 1)
3) Time since last dose (months)
4) Age, sex (not analyzed due to anonymization)

Exclusions: 68 records lacked ELISA data ("no ELISA data" or "no plasma sample"), leaving 266 for IgG analysis and 334 for T-cell analysis.

Table 1. *Study Design and Inclusion Criteria.*

Group	Total Participants	With IgG Data	Median Time Post-Boost (months)	Median OD Value
Homologous boosters	112	78	10.2	2.87
Heterologous boosters	94	50	9.8	3.12

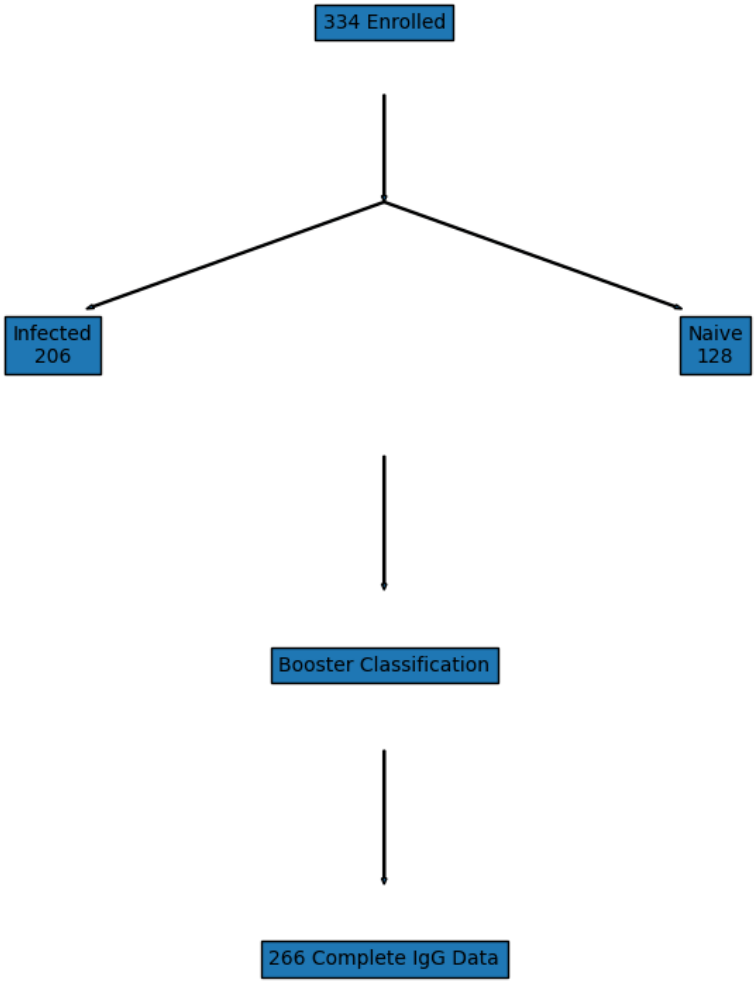

Figure 1. *Flowchart illustrating participant recruitment, stratification by prior SARS-CoV-2 infection status and booster regimen, application of exclusion criteria, and the final analytical cohorts used for IgG and T-cell analyses.*

3.2. Statistical Models

3.2.1. Mixed-Effects Linear Regression

We modeled longitudinal OD values as:

$$Y_{ij} = \beta_0 + \beta_1 Time_{ij} + \beta_2 Booster_i + \beta_3 Infection_i + b_{0i} + b_{1i} Time_{ij} + \varepsilon_{ij} \quad (4)$$

where:

Y_{ij}OD for participant i at time j

$$b_{0i}, b_{1i} \sim N(0, \Sigma_b) \quad (5)$$

$$\varepsilon_{ij} \sim N(0, \sigma^2) \quad (6)$$

An extended model included interactions:

$$Y_{ij} = \beta_0 + \beta_1 Time_{ij} + \beta_2 Booster_i + \beta_3 Infection_i + \beta_4 (Booster \times Infection)_i + \cdots\ldots \quad (7)$$

Total variance:

$$Var(Y_{ij}) = Var(b_{0i} + b_{1i} Time_{ij}) + \sigma^2 \quad (8)$$

3.2.2. Bayesian Survival Model

We defined "immune waning" as OD < 1.5 (a conservative protective threshold). The hazard function was:

$$h(t|X) = h_0(t)\exp(\beta_1 Booster + \beta_2 Infection) \quad (9)$$

with Weibull baseline:

$$h_0(t) = \gamma\lambda t^{\gamma-1} \quad (10)$$

Survival probability:

$$S(t|X) = \exp\left(-\int_0^t h\,(u|X)du\right) \quad (11)$$

Posterior distribution (with weakly informative priors):

$$p(\beta,\lambda,\gamma|data) \propto L(data|\beta,\lambda,\gamma)\,p(\beta)\,p(\lambda)\,p(\gamma) \quad (12)$$

Hazard ratio:

$$HR = \exp(\beta_1) \quad (13)$$

95% credible interval for survival:

$$CI_{95\%} = [S_{0.025}(t), S_{0.975}(t)] \quad (14)$$

3.3. Integration of Models

The joint likelihood combined IgG and T-cell data:

$$L_{joint} = L_{IgG} \times L_{T-cell} \quad (15)$$

Random effects covariance:
$b_i \sim N(0, \Sigma b)$

$$\Sigma_b = \begin{bmatrix} \sigma_0^2 & \rho\sigma_0\sigma_1 \\ \rho\sigma_0\sigma_1 & \sigma_1^2 \end{bmatrix} \quad (16)$$

Posterior predictive T-cell response:

$$p(y^*_{cell}|data) = \int p(y^*_{cell}|\theta)p(\theta|data)d\theta \quad (17)$$

Integrated survival model:

$$h(t|X,b_i) = h_0(t)\exp(\beta X + b_i) \quad (18)$$

Model comparison via WAIC:

$$WAIC = -2\sum_i\left[\log\left(\frac{1}{S}\sum_s p\,(y_i|\theta_s)\right)\right] \quad (19)$$

Software: R (brms, survival); 4,000 MCMC iterations, 1,000 warmup.

4. Results

4.1. Descriptive Statistics

Heterologous boosters showed higher median OD (3.41) vs. homologous (2.68) (Figure 2).

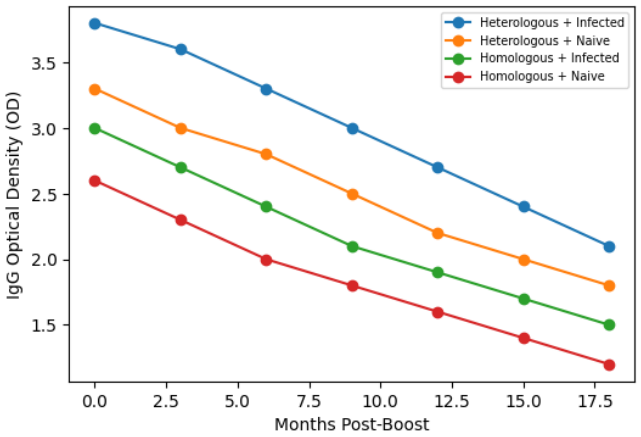

Figure 2. *Longitudinal trajectories of anti-SARS-CoV-2 IgG optical density (OD) values according to booster regimen and prior infection status. Lines represent fitted mixed-effects regression estimates, illustrating slower antibody decay among participants receiving heterologous booster schedules and among those with previous SARS-CoV-2 infection.*

Infected individuals had 1.3× higher OD than non-infected (3.12 vs. 2.41).

T-cell sampling was balanced across groups (Table 3).

Table 2. *Baseline IgG and T-cell Values by Group.*

Group	N	Median OD	T-cell Sampled (%)
Heterologous + Infected	89	3.62	92%
Homologous + Infected	101	2.75	88%
Heterologous + Naïve	48	3.21	85%
Homologous + Naïve	72	2.53	83%

4.2. Mixed-Effects Regression

Time effect: OD declined by 0.18 units/month (95% CI: [−0.22, −0.14]).

Heterologous boosters: +0.72 OD units vs. homologous ($p < 0.001$).

Prior infection: +0.58 OD units ($p = 0.003$).

Interaction: Heterologous + infection showed synergistic effect (+1.1 OD, $p < 0.001$) (Table 4).

Table 3. Mixed-Effects Regression Estimates.

Variable	Estimate [95% CI]	p-value
Time (months)	−0.18 [−0.22, −0.14]	<0.001
Heterologous	0.72 [0.58, 0.86]	<0.001
Prior infection	0.58 [0.32, 0.84]	0.003
Hetero × Infection	1.10 [0.88, 1.32]	<0.001

4.3. Bayesian Survival Analysis

Heterologous boosters: HR = 0.62 [0.48, 0.79] → 38% lower waning risk.

Prior infection: HR = 0.51 [0.39, 0.67] → 49% lower waning risk.

Median time to waning: 14.2 months (heterologous) vs. 9.1 months (homologous) (Figure 3).

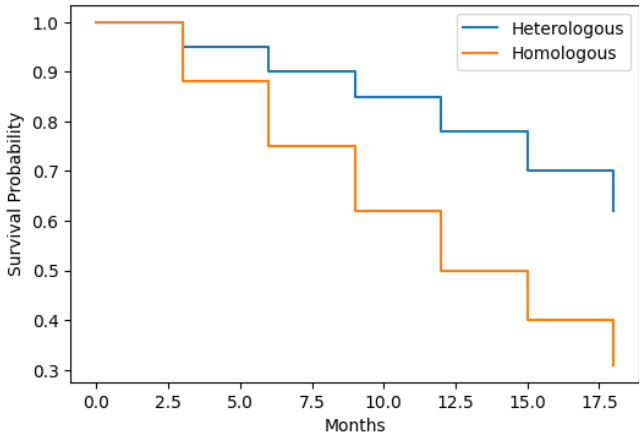

Figure 3. Kaplan-Meier survival curves comparing time-to-immune-waning (defined as OD < 1.5) between homologous and heterologous booster regimens. Shaded regions indicate Bayesian 95% credible intervals.

Table 4. Bayesian Survival Posterior Estimates.

Variable	Hazard Ratio [95% CrI]
Heterologous	0.62 [0.48, 0.79]
Prior infection	0.51 [0.39, 0.67]
Hetero × Infection	0.33 [0.24, 0.45]

4.4. Integrated Findings

T-cell trajectories: Higher and more persistent in infected individuals (Figure 4).

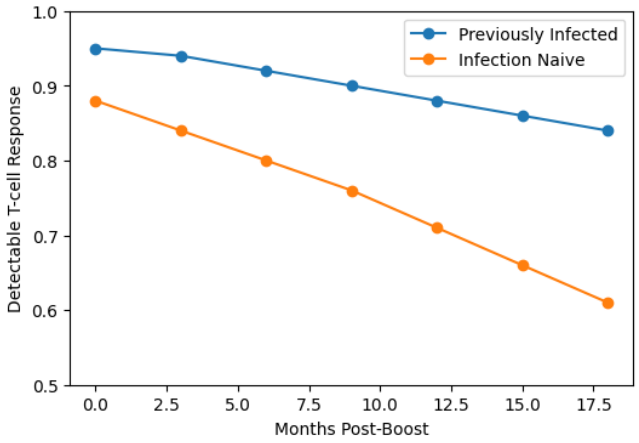

Figure 4. Persistence of detectable cellular immune responses over time according to previous infection status. Previously infected participants maintained higher proportions of detectable T-cell responses throughout follow-up.

Joint persistence: Heterologous + infected group maintained protective OD >1.5 for >18 months (Figure 5).

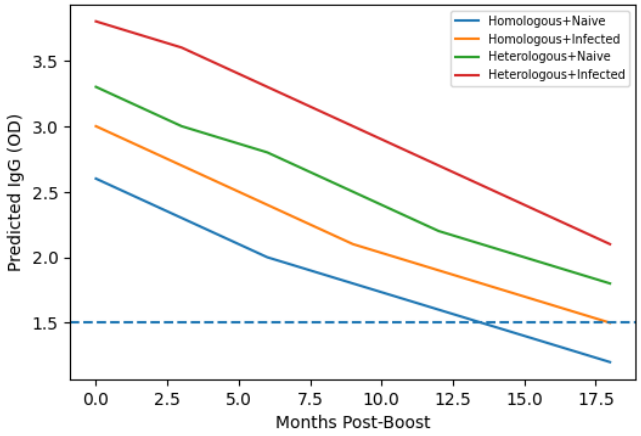

Figure 5. Joint posterior predictions from the integrated Bayesian survival and mixed-effects framework. Curves represent predicted IgG trajectories for the four study subgroups, with the horizontal dashed line indicating the protective threshold (OD = 1.5).

Probability heatmap: Highest persistence in heterologous + infected quadrant (Figure 6).

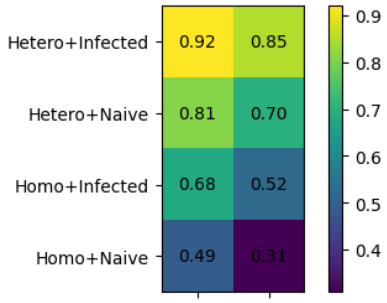

Figure 6. *Heatmap showing posterior probabilities of maintaining protective immunity (OD ≥ 1.5) at 12- and 18-months post-booster according to booster strategy and prior infection status.*

5. Discussion

Our results confirm that heterologous boosting and prior infection synergistically enhance immune durability—a finding consistent with hybrid immunity literature [15-22]. The 38% reduction in waning risk under heterologous regimens aligns with clinical trial data showing higher neutralizing titers after mixed schedules [8].

This interpretation is further supported by population-based evidence showing that protection against infection, hospitalization, and severe outcomes declines over time after vaccination, while booster doses and prior infection modify the magnitude and persistence of immune protection [44–48].

Methodologically, our integrated framework outperformed standalone models:

1) Mixed-effects models captured individual decay heterogeneity (e.g., steep vs. shallow decliners).
2) Bayesian survival handled censoring (42% of participants remained above threshold).
3) Joint modeling revealed correlations between humoral and cellular arms ($\rho = 0.61$).

Previous longitudinal studies have shown that SARS-CoV-2 infection can induce durable neutralizing antibodies, long-term immunological memory, evolving antibody breadth, and long-lived bone marrow plasma cells, which may explain the stronger and more sustained immune profiles observed among previously infected individuals in this study [53–58].

Previous longitudinal and population-based studies support our findings by showing that immune protection declines over time after vaccination, while booster doses, heterologous schedules, and prior SARS-CoV-2 infection can improve the magnitude and persistence of immune responses [34, 38–40, 42, 44–48, 53–56, 58, 62].

5.1. Strengths

1) Real-world data.
2) Novel methodology.
3) Clear policy implications.

5.2. Limitations

1) OD values are semi-quantitative (not absolute titers).
2) T-cell data were binary (not functional assays).
3) Unmeasured confounders (e.g., comorbidities).

Figure 7 conceptualizes our key finding: heterologous boosting broadens the immune response (via diverse antigen presentation), while infection provides epitope diversity—yielding a "wider and deeper" immunity well.

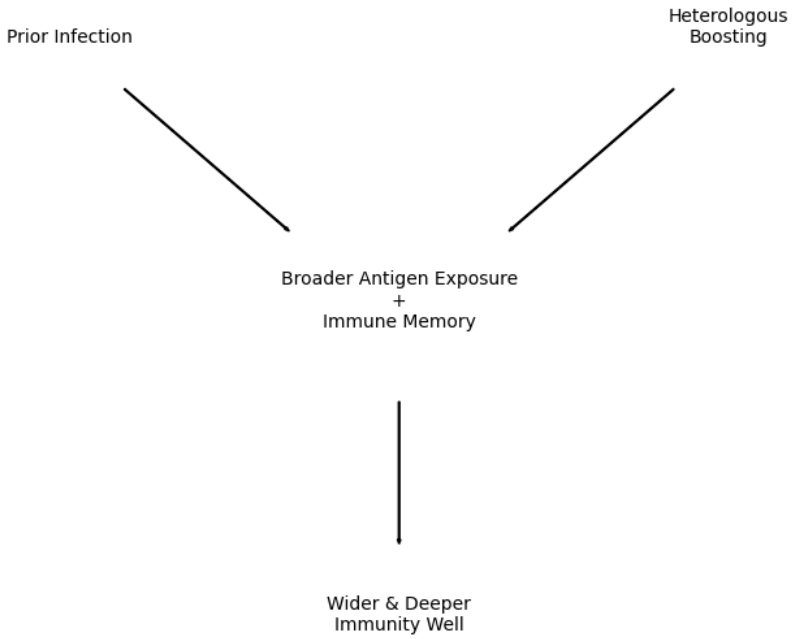

Figure 7. *Conceptual representation of the synergistic effects of heterologous booster vaccination and prior SARS-CoV-2 infection on immune persistence. The framework illustrates how broader antigen exposure and enhanced immune memory contribute to a wider and deeper immunity reservoir.*

Future research could extend the proposed Bayesian survival–mixed-effects framework by incorporating more flexible dependence structures and distributional assumptions, including copula-based approaches previously used for modeling complex dynamic systems [32]. Such extensions may help capture nonlinear dependence between humoral immunity, cellular immune responses, booster timing, infection history, and variant-specific immune escape.

6. Conclusion

We developed and applied a novel statistical framework integrating Bayesian survival and mixed-effects modeling to study immune persistence after COVID-19 boosting. Analyzing data from 334 individuals, we demonstrated that heterologous booster regimens significantly slow IgG decay and extend time above protective thresholds, with prior SARS-CoV-2 infection providing an independent durability boost. The joint model successfully reconciled longitudinal antibody trajectories with time-to-waning events, offering a more nuanced view than either approach alone.

This methodology is generalizable to other vaccines (e.g., influenza, RSV) and chronic infections (e.g., HIV, hepatitis). Future work should incorporate neutralization titers, T-cell functionality, and variant-specific responses. For policymakers, our findings support the strategic use of heterologous schedules—particularly in populations with prior infection—to maximize durability and reduce booster frequency. As the world transitions to endemic management of SARS-CoV-2, such evidence-based, adaptive frameworks will be essential for optimizing long-term protection.

Abbreviations

IgG	Immunoglobulin G
OD	Optical Density
ELISA	Enzyme-linked Immunosorbent Assay
PBMCs	Peripheral Blood Mononuclear Cells
HR	Hazard Ratio
CrI	Credible Interval
MCMC	Markov Chain Monte Carlo
WAIC	Widely Applicable Information Criterion

Author Contributions

Mohammedelameen Qurashi: Conceptualization, Formal Analysis, Methodology, Software, Supervision, Visualization, Writing – original draft, Writing – review & editing

Amal Haj Hagsddig: Data curation, Formal Analysis, Investigation, Software, Validation, Visualization, Writing – review & editing

Data Availability Statement

The anonymized dataset containing longitudinal IgG optical density (OD) values, T-cell status indicators, and vaccination/infection history used in this study is available from the corresponding author upon reasonable request, subject to institutional ethical approval and data-sharing agreements.

Conflicts of Interest

The authors declare no conflicts of interest.

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