KCOR: A depletion-neutralized framework for retrospective cohort comparison under latent frailty

Manuscript metadata

- **Article type**: Methods / Statistical method
- **Running title**: KCOR under selection-induced cohort bias
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- **Word count**: 12,100 (excluding Abstract and References)

- **Keywords**: selection bias; frailty model; gamma mixture model; frailty inversion; frailty heterogeneity; depletion of susceptibles; non-proportional hazards; cumulative hazard; hazard normalization; cumulative hazards; estimands; gamma frailty; negative controls; observational studies; observational cohort studies

Abstract

Selection-induced depletion under latent frailty heterogeneity can generate non-proportional hazards and curvature in observed cumulative hazards, biasing standard survival estimands in retrospective cohort studies using registry and administrative data. KCOR is a depletion-neutralized cohort comparison framework based on gamma-frailty normalization. It estimates cohort-specific depletion geometry during prespecified quiet periods and applies an analytic inversion to map observed cumulative hazards into a common comparison scale prior to computing cumulative contrasts. Across simulations spanning frailty heterogeneity and selection strength and across negative and positive controls, Cox proportional hazards regression can exhibit systematic non-null behavior under selection-only regimes. In contrast, KCOR-normalized trajectories remain stable and centered near the null while detecting injected effects. KCOR provides a diagnostic and descriptive framework for comparing fixed cohorts under selection-induced hazard curvature by separating depletion normalization from outcome comparison and improving interpretability of cumulative outcome analyses under minimal-data constraints.

1. Introduction

1.1 Retrospective cohort comparisons under selection

Randomized controlled trials (RCTs) are the gold standard for causal inference, but are often infeasible, underpowered for rare outcomes, or unavailable for questions that arise after rollout. As a result, observational cohort comparisons are widely used to estimate intervention effects on outcomes such as all-cause mortality.

Although mortality is used throughout this paper as a motivating and concrete example, the method applies more generally to any irreversible event process observed in a fixed cohort, including hospitalization, disease onset, or other terminal or absorbing states. Mortality is emphasized here because it is objectively defined, reliably recorded in many national datasets, and free from outcome-dependent ascertainment biases that complicate other endpoints.

However, when intervention uptake is voluntary, prioritized, or otherwise selective, treated and untreated cohorts are frequently **non-exchangeable** at baseline and evolve differently over follow-up. This problem is not limited to any single intervention class; it arises whenever the same factors that influence treatment uptake also influence outcome risk.

This manuscript is a methods paper. Real-world registry data are used solely to demonstrate estimator behavior, diagnostics, and failure modes under realistic selection-induced non-proportional hazards; no policy conclusions are drawn.

1.2 Curvature (shape) is the hard part: non-proportional hazards from frailty depletion

Selection does not merely shift mortality **levels**; it can alter mortality **curvature**—the time-evolution of cohort hazards. Frailty heterogeneity and depletion of susceptibles naturally induce curvature of the cumulative hazard (reflecting time-varying hazard) even when individual-level hazards are simple functions of time. When selection

concentrates high-frailty individuals into one cohort (or preferentially removes them from another), the resulting cohort-level hazard trajectories can be strongly non-proportional.

One convenient way to formalize "curvature" is in cumulative-hazard space: if the cumulative hazard $H(t)$ were perfectly linear in time, then its second derivative would be zero, whereas selection-induced depletion generally produces negative concavity (downward curvature) in observed cumulative hazards during otherwise stable periods.

This violates core assumptions of many standard tools:

- **Cox PH**: assumes hazards differ by a time-invariant multiplicative factor (proportional hazards).
- **IPTW / matching**: can balance measured covariates yet fail to balance unmeasured frailty and the resulting depletion dynamics.
- **Age-standardization**: adjusts levels across age strata but does not remove cohort-specific time-evolving hazard shape.

KCOR is designed for this failure mode: **cohorts whose hazards are not proportional because selection induces different depletion dynamics (curvature).** Approximate linearity of cumulative hazard after adjustment is therefore not assumed, but serves as an internal diagnostic indicating that selection-induced depletion has been successfully removed.

The methodological problem addressed here is general. The COVID-19 period provides a natural empirical regime characterized by strong selection heterogeneity and non-proportional hazards, serving as a useful illustration for the proposed framework. However, KCOR is not specific to COVID, vaccination, or infectious disease. KCOR refers to the method as presented here; earlier internal iterations are not material to the estimand or results and are omitted for clarity.

Two mechanisms often lumped as the 'healthy vaccinee effect' (HVE) are distinguished here:

- **Static HVE:** baseline differences in latent frailty distributions at cohort entry (e.g., vaccinated cohorts are healthier on average). In the KCOR framework, this manifests as differing depletion curvature (different θ_d) and is the primary target of frailty normalization.
- **Dynamic HVE:** short-horizon, time-local selection processes around enrollment that create transient hazard suppression immediately after enrollment (e.g., deferral of vaccination during acute illness, administrative timing, or short-term behavioral/health-seeking changes). Dynamic HVE is operationally addressed by prespecifying a skip/stabilization window (§2.7) and can be evaluated empirically by comparing early-period signatures across related cohorts in multi-dose settings.

1.3 Related work (brief positioning)

KCOR builds on the frailty and depletion-of-susceptibles literature in which unobserved heterogeneity induces deceleration of cohort-level hazards over follow-up (a standard working model is gamma frailty)[1]. KCOR's distinct contribution is not additional hazard flexibility, but a **diagnostics-driven normalization** of selection-induced depletion geometry in cumulative-hazard space prior to defining a cumulative cohort contrast. Related approaches that address non-proportional hazards (time-varying effects, flexible parametric hazards, additive hazards) or time-varying confounding (MSM/IPW/g-methods) target different estimands and typically require richer longitudinal covariates than are available in minimal registry data[2–9]. Additional discussion is provided in the Supplementary Information (SI).

1.4 Evidence from the literature: residual confounding despite meticulous matching

Motivating applied studies show that even careful matching and adjustment can leave substantial residual differences in non-COVID mortality and time-varying "healthy vaccinee effect" signatures, consistent with selection and depletion dynamics not captured by measured covariates[10,11].

1.5 Contribution of this work

This work makes four primary contributions: (i) it formalizes selection-induced depletion under latent frailty heterogeneity as a source of non-proportional hazards and curvature that can bias common survival estimands;

(ii) it defines a diagnostics-first normalization that fits depletion geometry in quiet periods and maps observed cumulative hazards into a depletion-neutralized space; (iii) it validates operating characteristics using synthetic and empirical controls, including a synthetic null under selection-only regimes; and (iv) it separates normalization from comparison by permitting standard post-normalization cumulative estimands.

A central implication is identifiability: in minimal-data retrospective cohorts, interpretability depends on an epidemiologically quiet window and on internal diagnostics that indicate depletion geometry has been estimated and removed, rather than absorbed into a time-varying effect estimate.

Together, these contributions position KCOR not as a replacement for existing survival estimands, but as a prerequisite normalization step that addresses a source of bias arising prior to model fitting in many retrospective cohort studies.

1.6 Target estimand and scope (non-causal)

Box 1. Target estimand and scope (non-causal).

- **Primary estimand (KCOR)**: For two fixed enrollment cohorts A and B, we define

$$\mathrm{KCOR}(t) = \tilde{H}_{0,A}(t)/\tilde{H}_{0,B}(t),$$

  where $\tilde{H}_{0,d}(t)$ is cohort d's **depletion-neutralized baseline cumulative hazard** obtained by fitting depletion geometry in a prespecified quiet window and applying the gamma-frailty inversion (Methods §2).
- **Interpretation**: KCOR is a time-indexed **cumulative** contrast on the depletion-neutralized scale. Values above/below 1 indicate greater/less cumulative event accumulation in cohort A than B by time t after depletion normalization. KCOR is not an instantaneous hazard ratio.
- **What it is not**: KCOR is **not** a causal effect estimator (no ATE/ATT) and does not recover counterfactual outcomes under hypothetical interventions.
- **When interpretable**: Interpretation is conditional on explicit assumptions (fixed cohorts; shared external hazard environment; adequacy of the working frailty model; existence of an epidemiologically quiet window) **and** on internal diagnostics (quiet-window fit quality; post-normalization linearity within the quiet window; parameter stability to small window perturbations).
- **If diagnostics fail**: treat the analysis as not identified and do not report KCOR as a "corrected effect".

1.7 Paper organization and supporting information (SI)

The main text presents the KCOR estimator, a single canonical demonstration of Cox bias under frailty-driven depletion, and two main validation examples (negative control and stress test). Additional validations (including positive controls), extended diagnostics (Supplementary Information §S2; Tables S2.1–S2.3), and detailed simulation/control specifications are provided in the Supplementary Information (SI) document.

2. Methods

Mortality is used as the primary example throughout this section because it is objectively defined and reliably recorded in many administrative datasets.

Table **??** defines the notation used throughout the Methods section.

For COVID-19 vaccination analyses, intervention count corresponds to the number of vaccine doses received; more generally, this can index any discrete exposure level.

2.1 Conceptual framework and estimand

Retrospective cohort differences can arise from two qualitatively different components:

- **Level differences**: cohort hazards differ by an approximately time-stable multiplicative factor (or, equivalently, cumulative hazards have different slopes but similar shape).
- **Depletion (curvature) differences**: cohort hazards evolve differently over time because cohorts differ in latent heterogeneity and are **selectively depleted** at different rates.

This framework targets the second failure mode. Under latent frailty heterogeneity, high-risk individuals die earlier, so the surviving risk set becomes progressively "healthier." This induces **downward curvature** (deceleration) in cohort hazards and corresponding concavity in cumulative-hazard space, even when individual-level hazards are simple and even under a true null treatment effect. When selection concentrates frailty heterogeneity differently across cohorts, the resulting curvature differences produce strong non-proportional hazards and can drive misleading contrasts for estimands that condition on the evolving risk set.

The strategy is therefore:

1. **Estimate the cohort-specific depletion geometry** (via curvature) during prespecified epidemiologically quiet periods.
2. **Map observed cumulative hazards into a depletion-neutralized space** by inverting that geometry.
3. **Compare cohorts only after normalization** using a prespecified post-adjustment estimand; ratios of depletion-neutralized cumulative hazards (KCOR) are used here.

All analyses are performed using discrete weekly time bins; continuous-time notation is used solely for expositional convenience.

2.1.1 Target estimand

Scope and interpretation are summarized in Box 1 (§1.6); the formal definition used throughout is provided here.

Let $\tilde{H}_{0,d}(t)$ denote the **depletion-neutralized baseline cumulative hazard** for cohort d at event time t since enrollment (Table **??**). For two cohorts A and B, KCOR is defined as

$$\mathrm{KCOR}(t) = \frac{\tilde{H}_{0,A}(t)}{\tilde{H}_{0,B}(t)}. \tag{1}$$

For visualization, an **anchored KCOR** is sometimes reported to show post-reference divergence:

$$\mathrm{KCOR}(t; t_0) = \mathrm{KCOR}(t)/\mathrm{KCOR}(t_0),$$

with prespecified t_0 (e.g., 4 weeks).

2.1.2 Identification versus diagnostics

Scope and interpretation are summarized in Box 1 (§1.6).

Interpretability of a KCOR trajectory is assessed via prespecified diagnostics (Supplementary Information §S2; Tables S2.1–S2.3), and analyses are treated as not identified when those diagnostics fail. Checks include:

- stability of $(\hat{k}_d, \hat{\theta}_d)$ to small quiet-window perturbations,
- approximate linearity of $\tilde{H}_{0,d}(t)$ within the quiet window,
- absence of systematic residual structure in cumulative-hazard space.

Diagnostics corresponding to each assumption are summarized in Supplementary Table S1 and discussed in detail in Supplementary Information §S2.

2.1.3 KCOR assumptions and diagnostics

These assumptions define when KCOR normalization is interpretable.

The KCOR framework relies on the following assumptions, which are framed diagnostically:

1. **Fixed cohort enrollment.** Cohorts are defined at a common enrollment time and followed forward without dynamic entry or rebalancing.
2. **Multiplicative latent frailty.** Individual hazards are assumed to be multiplicatively composed of a baseline hazard and an unobserved frailty term, with cohort-specific frailty distributions.
3. **Quiet-window stability.** A prespecified epidemiologically quiet period exists during which external shocks to the baseline hazard are minimal, allowing depletion geometry to be estimated from observed cumulative hazards.
4. **Independence across strata.** Cohorts or strata are analyzed independently, without interference, spillover, or cross-cohort coupling.
5. **Sufficient event-time resolution.** Event timing is observed at a temporal resolution adequate to estimate cumulative hazards over the quiet window.

These assumptions are evaluated empirically using post-normalization diagnostics. Violations are expected to manifest as residual curvature, drift, or instability in adjusted cumulative hazard trajectories.

2.2 Cohort construction

We define KCOR for **fixed cohorts at enrollment**. Required inputs are minimal: enrollment date(s), event date, and optionally birth date (or year-of-birth) for age stratification. Analyses proceed in discrete event time t (e.g., weeks) measured since cohort enrollment.

Cohorts are assigned by intervention state at the start of the enrollment interval. In the primary estimand:

- **No post-enrollment switching** is allowed (individuals remain in their enrollment cohort),
- **No censoring** is applied (other than administrative end of follow-up),
- analyses are performed on the resulting fixed risk sets.

Censoring or reclassification due to cohort transitions (e.g., moving between exposure groups over time) is not permitted, because such transitions alter the frailty composition of the cohort in a time-dependent manner. Allowing transitions would introduce additional, endogenous selection that changes cohort mortality trajectories in unpredictable ways, confounding depletion effects that KCOR is designed to normalize.

This fixed-cohort design is intentional. It avoids immortal-time artifacts and prevents outcome-driven switching rules from creating time-dependent selection that is difficult to diagnose under minimal covariate availability. Extensions that allow switching or censoring are treated as sensitivity analyses (§5.2) because they change the estimand and introduce additional identification requirements.

Conceptual requirements of the KCOR framework are distinguished from operational defaults, which are reported separately for reproducibility (Supplementary Section S4).

Throughout this manuscript the failure event is **all-cause mortality**. KCOR therefore targets cumulative mortality hazards and is not framed as a cause-specific competing-risks analysis.

2.3 Hazard estimation and cumulative hazards in discrete time

For each cohort d, let $N_d(0)$ denote the number of individuals at enrollment. Let $d_d(t)$ denote deaths occurring during interval t, and let

$$D_d(t) = \sum_{s \le t} d_d(s)$$

denote cumulative deaths up to the end of interval t.

Define the risk set size at the start of interval t as

$$N_d(t) = N_d(0) - \sum_{s<t} d_d(s) = N_d(0) - D_d(t-1).$$

In the primary estimand, individuals do not switch cohorts after enrollment and there is no loss to follow-up; therefore $N_d(t)$ is the risk set used to define all discrete-time hazards and cumulative hazards in this manuscript.

Define the interval mortality ratio

$$\mathrm{MR}_{d,t} = \frac{d_d(t)}{N_d(t)}.$$

We compute the discrete-time cohort hazard as

$$h_{\mathrm{obs},d}(t) = -\ln\bigl(1 - \mathrm{MR}_{d,t}\bigr) = -\ln\Bigl(1 - \frac{d_d(t)}{N_d(t)}\Bigr). \tag{2}$$

This transform is standard: it maps an interval event probability into a continuous-time equivalent hazard under a piecewise-constant hazard assumption. For rare events, $h_{\mathrm{obs},d}(t) \approx \mathrm{MR}_{d,t} = d_d(t)/N_d(t)$, but the log form remains accurate and stable when weekly risks are not negligible.

All hazard and cumulative-hazard quantities used in KCOR are discrete-time integrated hazard estimators derived from fixed-cohort risk sets; likelihood-based or partial-likelihood formulations are not used for estimation or for the subsequent frailty-based normalization.

Observed cumulative hazards are accumulated over event time after an optional stabilization skip (§2.7):

$$H_{\mathrm{obs},d}(t) = \sum_{s \le t} h_d^{\mathrm{eff}}(s), \qquad \Delta t = 1. \tag{3}$$

Discrete binning accommodates tied events and aggregated registry releases. Bin width is chosen based on diagnostic stability (e.g., smoothness and sufficient counts per bin) rather than temporal resolution alone.

In addition to the primary implementation above, $\hat{H}_{\mathrm{obs},d}(t)$ was computed using the Nelson–Aalen estimator $\sum_{s \le t} d_d(s)/N_d(s)$ as a sensitivity check; results were unchanged.

2.4 Selection model: gamma frailty and depletion normalization

2.4.1 Individual hazards with multiplicative frailty

Within cohort d, individual i is modeled as having hazard

$$h_{i,d}(t) = z_{i,d}\, h_{0,d}(t), \qquad z_{i,d} \sim \mathrm{Gamma}(\mathrm{mean} = 1,\ \mathrm{var} = \theta_d). \tag{4}$$

Here $h_{0,d}(t)$ is the cohort's depletion-neutralized baseline hazard and $z_{i,d}$ is a latent multiplicative frailty term. The frailty variance θ_d governs the strength of depletion-induced curvature: larger θ_d yields stronger deceleration at the cohort level due to faster early depletion of high-frailty individuals.

Gamma frailty is used because it yields a closed-form link between observed and baseline cumulative hazards via the Laplace transform[1]. In KCOR, gamma frailty is a **working geometric model** for depletion normalization, not a claim of biological truth. Adequacy is evaluated empirically via fit quality, post-normalization linearity, and stability diagnostics.

2.4.2 Gamma-frailty identity and inversion

Let

$$H_{0,d}(t) = \int_0^t h_{0,d}(s)\, ds \tag{5}$$

denote the baseline cumulative hazard. Integrating over gamma frailty yields the gamma-frailty identity

$$H_{\mathrm{obs},d}(t) = \frac{1}{\theta_d}\,\log\bigl(1 + \theta_d H_{0,d}(t)\bigr), \tag{6}$$

which can be inverted exactly as

$$H_{0,d}(t) = \frac{\exp\big(\theta_d H_{\mathrm{obs},d}(t)\big) - 1}{\theta_d}. \tag{7}$$

This inversion is the **normalization operator**: given an estimate $\hat{\theta}_d$, it maps the observed cumulative hazard $H_{\mathrm{obs},d}(t)$ into a depletion-neutralized cumulative hazard scale.

2.4.3 Baseline shape used for frailty identification

To identify θ_d, KCOR fits the gamma-frailty model within prespecified epidemiologically quiet periods. In the reference specification, the baseline hazard is taken to be constant over the fit window:

$$h_{0,d}(t) = k_d, \qquad H_{0,d}(t) = k_d\, t. \tag{8}$$

This choice intentionally minimizes degrees of freedom: during a quiet window, curvature is forced to be explained by depletion (via θ_d) rather than by introducing time-varying baseline hazard terms. If the observed cumulative hazard is near-linear over the fit window, the model naturally collapses toward $\hat{\theta}_d \approx 0$, signaling weak or absent detectable depletion curvature for that cohort over that window.

2.4.4 Quiet-window validity as the key dataset-specific requirement

Frailty parameters are estimated using only bins whose corresponding calendar weeks lie inside a prespecified quiet window (defined in ISO-week space). The quiet window is prespecified to avoid sharp, cohort-differential hazard perturbations (e.g., epidemic waves or policy shocks) that would confound depletion-geometry estimation. A window is acceptable only if diagnostics indicate (i) good fit in cumulative-hazard space, (ii) post-normalization linearity within the window, and (iii) stability of $(\hat{k}_d, \hat{\theta}_d)$ to small boundary perturbations. If no candidate window passes, KCOR is treated as not identified for that analysis rather than producing a potentially misleading normalized contrast. All diagnostics are computed over discrete event-time bins (weekly intervals since enrollment) whose corresponding calendar weeks fall within the prespecified quiet window.

Quiet-window selection protocol (operational)

Quiet-window selection is prespecified and evaluated using diagnostic criteria summarized in Supplementary Information §S2 (Tables S2.1–S2.3).

2.5 Estimation during quiet periods (cumulative-hazard least squares)

KCOR estimates $(\hat{k}_d, \hat{\theta}_d)$ independently for each cohort d using only time bins that fall inside a prespecified quiet window in calendar time (see §2.4.4). The quiet window is applied consistently across cohorts within an analysis. Let $\mathcal{T}_d$ denote the set of event-time bins t whose corresponding calendar week lies in the quiet window, with t also satisfying $t \geq$ SKIP_WEEKS.

Under the default baseline shape, the model-implied observed cumulative hazard is

$$H_d^{\mathrm{model}}(t; k_d, \theta_d) = \frac{1}{\theta_d} \log(1 + \theta_d k_d t)\,. \tag{9}$$

Identifiability of $(\hat{k}_d, \hat{\theta}_d)$ comes from curvature in cumulative-hazard space: observed cumulative hazards are nonlinear in event time when $\theta_d > 0$. When depletion is weak (or the quiet window is too short to show curvature), the model smoothly collapses to a linear cumulative hazard, since $H_d^{\mathrm{model}}(t; k_d, \theta_d) \to k_d t$ as $\theta_d \to 0$. Operationally, near-linear observed cumulative hazards naturally drive the fitted frailty variance toward zero; fit diagnostics such as n_{obs} and RMSE in H-space provide a practical check on whether the selection parameters are being identified from the quiet-window data. In practice, lack of identifiable curvature naturally manifests as fitted frailty variance estimates approaching zero, providing an internal diagnostic for non-identifiability over short or sparse follow-up.

In applied analyses, this behavior is most commonly observed in vaccinated cohorts, whose cumulative hazards during quiet periods are often close to linear. In such cases, the gamma-frailty fit collapses naturally, indicating minimal detectable depletion. This outcome is data-driven and reflects the absence of observable selection-induced curvature rather than a modeling assumption. When residual time-varying risk contaminates a nominally quiet window, fitted frailty variance estimates naturally shrink toward zero, signaling limited identifiability rather than inducing spurious correction.

Parameters are estimated by constrained nonlinear least squares:

$$(\hat{k}_d, \hat{\theta}_d) = \arg \min_{k_d>0,\ \theta_d\geq 0} \sum_{t\in\mathcal{T}_d} \left[H_{\mathrm{obs},d}(t) - H_d^{\mathrm{model}}(t; k_d, \theta_d)\right]^2 . \tag{10}$$

We fit in cumulative-hazard space rather than maximizing a likelihood because the primary inputs are discrete-time, cohort-aggregated hazards and the objective is stable estimation of selection-induced depletion curvature during quiet periods. Least-squares fitting is used as a numerical estimating equation rather than as a likelihood-based estimator. Least squares on observed cumulative hazards is numerically robust under sparse events, emphasizes shape agreement over the fit window, and yields diagnostics (e.g., RMSE in H-space) that directly reflect the quality of the depletion fit. Likelihood-based fitting can be treated as a sensitivity analysis, but is not required for the normalization identity itself.

All analyses use a prespecified reference implementation with fixed operational defaults; full details are provided in Supplementary Section S4.

2.6 Normalization (depletion-neutralized cumulative hazards)

After fitting, KCOR computes the depletion-neutralized baseline cumulative hazard for each cohort d by applying the inversion to the full post-enrollment trajectory:

$$\tilde{H}_{0,d}(t) = \frac{\exp\left(\hat{\theta}_d\, H_{\mathrm{obs},d}(t)\right) - 1}{\hat{\theta}_d}. \tag{11}$$

This normalization maps each cohort into a depletion-neutralized baseline-hazard space in which the contribution of gamma frailty parameters $(\hat{\theta}_d, \hat{k}_d)$ to hazard curvature has been factored out. This normalization defines a common comparison scale in cumulative-hazard space; it is not equivalent to Cox partial-likelihood baseline anchoring, but serves an analogous geometric role for cumulative contrasts. In this space, cumulative hazards are directly comparable across cohorts, and remaining differences reflect real differences in baseline risk rather than selection-induced depletion. The core identities used in KCOR are given in Equations (**??**), (**??**), (**??**), and (**??**). Normalization defines a common comparison scale; the scientific estimand is then computed on that scale (Box 1).

2.6.1 Computational considerations

KCOR operates on aggregated event counts in discrete time and cumulative-hazard space. Computational complexity scales linearly with the number of time bins and strata rather than the number of individuals, making the method feasible for very large population registries. In practice, KCOR analyses on national-scale datasets (millions of individuals) are memory-bound rather than CPU-bound and can be implemented efficiently using standard vectorized numerical libraries. No iterative optimization over individual-level records is required.

2.6.2 Internal diagnostics and 'self-check' behavior

KCOR includes internal diagnostics intended to make model stress visible rather than hidden.

1. **Post-normalization linearity in quiet periods.** Within the prespecified quiet window (see §2.4.4), the depletion-neutralized cumulative hazard should be approximately linear in event time after inversion. Systematic residual curvature indicates window contamination (external shocks, secular trends) or misspecified depletion geometry for that cohort.

2. **Fit residual structure in cumulative-hazard space.** Define residuals over the fit set $\mathcal{T}_d$:

$$r_d(t) = H_{\text{obs},d}(t) - H_d^{\text{model}}(t; \hat{k}_d, \hat{\theta}_d). \tag{12}$$

KCOR expects residuals to be small and not systematically time-structured. Strongly patterned residuals indicate that the curvature attributed to depletion is instead being driven by unmodeled time-varying hazards.

3. **Parameter stability to window perturbations.** Under valid quiet-window selection,

$$(\hat{k}_d, \hat{\theta}_d)$$

should be stable to small perturbations of the quiet-window boundaries (e.g., ±4 weeks). Large changes in fitted frailty variance under small boundary shifts signal that the fitted curvature is sensitive to transient dynamics rather than stable depletion.

4. **Non-identifiability manifests as:**

$$\hat{\theta}_d \to 0.$$

When the observed cumulative hazard is near-linear (weak curvature) or events are sparse, θ is weakly identified. In such cases, KCOR should be interpreted primarily as a diagnostic (limited evidence of detectable depletion curvature) rather than a strong correction.

These diagnostics are reported alongside KCOR(t) curves. The goal is not to assert that a single parametric form is always correct, but to ensure that when the form is incorrect or the window is contaminated, the method signals this explicitly rather than silently producing a misleading 'corrected' estimate. Failure of these diagnostics indicates that the depletion-based normalization is inappropriate, in which case KCOR should not be interpreted.

2.7 Stabilization (early weeks)

In many applications, the first few post-enrollment intervals can be unstable due to immediate post-enrollment artifacts (e.g., rapid deferral, short-term sorting, administrative effects). KCOR supports a prespecified stabilization rule by excluding early weeks from accumulation and from quiet-window fitting. The skip-weeks parameter is prespecified and evaluated via sensitivity analysis to exclude early enrollment instability rather than to tune estimates.

In discrete time, define an effective hazard for accumulation:

$$h_d^{\text{eff}}(t) = \begin{cases} 0, & t < \text{SKIP\_WEEKS} \\ h_{\text{obs},d}(t), & t \geq \text{SKIP\_WEEKS}. \end{cases} \tag{13}$$

Then compute observed cumulative hazards from $h_d^{\text{eff}}(t)$ as in §2.3:

$$H_{\text{obs},d}(t).$$

2.8 KCOR estimator

With depletion-neutralized cumulative hazards in hand, the primary KCOR trajectory is defined as:

$$\text{KCOR}(t) = \frac{\tilde{H}_{0,A}(t)}{\tilde{H}_{0,B}(t)}. \tag{14}$$

This ratio is computed after depletion normalization and is interpreted conditional on the stated assumptions and diagnostics (Box 1; §2.1.2).