

Epidynamix: From Force to Field in Real-World Epidemiology

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Abstract

Modern causal inference in epidemiology treats interventions as external forces acting on passive subjects to produce average outcomes—a “Newtonian” abstraction that works well in randomized trials but falters in Real-World Data (RWD). In RWD, clinical guidelines create structural positivity violations, treatment decisions are entangled with prognosis, and effects are inherently heterogeneous across patient states. We propose Epidynamix, a framework that shifts focus from “Force” to “Field.” By viewing health states as points within a Risk Potential Field $\Phi(s) = -\log \lambda(s)$, we reinterpret causal effects as directional gradients, the ATE as a first-order projection, and positivity violations as structural boundaries—not estimation failures, but geometric features of the clinical landscape. Through simulation, we demonstrate that while standard methods (Cox regression, IPTW) collapse effect heterogeneity into scalar summaries, the field-based approach preserves clinically meaningful variation (~3.5-fold across state space) and explicitly maps regions where comparison is structurally impossible. This geometric perspective offers a complementary lens for RWD analysis: **a map, not a number.**

Introduction: The Newtonian Discomfort

Modern causal inference in epidemiology typically asks:

$$\text{ATE} = \mathbb{E}[Y(1) - Y(0)]$$

This formulation assumes: well-defined potential outcomes $Y(a)$, interventions acting as external causes, and counterfactual symmetry between $a = 0$ and $a = 1$. This works extremely well in randomized controlled trials.

However, in Real-World Data (RWD), the system is high-dimensional, time-varying, policy- and guideline-constrained, and observer-influenced. Yet we still model intervention as a force:

$$A \longrightarrow Y$$

Why now? The explosion of RWD—electronic health records, claims databases, registries—has exposed the limitations of this paradigm. Unlike curated trial populations, RWD reflects the full

complexity of clinical practice: treatment decisions are constrained by guidelines, contraindications create structural zeros in the propensity score, and effects vary systematically across patient states (Petersen et al. 2012). We increasingly face situations where:

- Treatment is mandated for high-risk patients (structural positivity violation)
- The “same” intervention varies by context, timing, and patient state
- Treatment decisions are entangled with prognosis (confounding by indication)

Traditional methods respond by restricting analysis to “overlap regions” or trimming extreme weights—effectively discarding the very patients for whom treatment decisions matter most.

We propose an alternative: **a map, not a number.**

The Geometric Turn: From Force to Field

Einstein did not reinterpret gravity by refining the force. He removed the force. Objects do not respond to gravity—they follow geodesics in a curved spacetime.

We propose an analogous shift:

- Causal effects are not primitive forces
- Outcomes arise from motion within a structured field
- Interventions reposition systems inside that field

Notation

Throughout this paper, we use the following conventions:

Symbol	Meaning
$S_t = (X_t, A_t)$	System state at time t
\mathcal{S}	State space
$\lambda(s)$	Instantaneous hazard at state s
$\Phi(s)$	Risk potential function
$\nabla \Phi$	Gradient (for continuous variables)
$\delta_A \Phi(x)$	Finite difference for binary A : $\Phi(x, 1) - \Phi(x, 0)$
$K(s, s')$	Transition kernel

Symbol	Meaning
\mathcal{I}_a	Intervention operator

Note on binary treatment: When $A \in \{0, 1\}$, expressions like $\partial\Phi/\partial a$ should be understood as the finite difference $\delta_A\Phi$, not a true derivative.

State Space and Risk Field

State Definition

We define the system state as:

$$S_t = (X_t, A_t) \in \mathcal{S}$$

where treatment A_t is a coordinate of the system, not an external action. The system evolves according to a transition kernel:

$$P(S_{t+1} | S_t) = K(S_t, S_{t+1})$$

Regions where K assigns zero probability are **structural**, not violations.

Risk Potential Function

Define a local risk field:

$$\lambda(s) = \lim_{\Delta t \rightarrow 0} \frac{P(T \in [t, t + \Delta t) | S_t = s)}{\Delta t}$$

This is estimable using standard survival models. We then define the risk potential:

$$\Phi(s) = -\log \lambda(s)$$

Interpretation: low $\Phi \rightarrow$ high risk; high $\Phi \rightarrow$ relative stability. Φ is not an outcome—it is a

geometric property of the state space.

Time Treatment

In this framework, we treat $\Phi(s)$ as either:

1. **Landmark approach:** Evaluated at a fixed reference time t^*
2. **Stationary assumption:** Time-invariant for a given state
3. **Cumulative formulation:** Integrated over a horizon $[0, \tau]$

For the examples in this paper, we adopt the **stationary assumption** for simplicity.

In non-stationary hazard settings, consider landmark or time-specific fields, or a cumulative definition of risk potential over $[0, \tau]$.

Intervention as a Transition Operator

Instead of a do-operator $\text{do}(A = a)$, we define intervention as a state transformation:

$$\mathcal{I}_a : \mathcal{S} \rightarrow \mathcal{S}, \quad \mathcal{I}_a(x, a') = (x, a)$$

Interventions move the system across the field. They do not directly generate outcomes.

Vector Field Interpretation

Gradient of the Potential

Φ defines a scalar field on \mathcal{S} . Its gradient defines a **vector field**:

$$\nabla \Phi(s) = \left(\frac{\partial \Phi}{\partial x_1}, \dots, \frac{\partial \Phi}{\partial x_p}, \frac{\partial \Phi}{\partial a} \right)$$

This **risk-gradient field** encodes directions of maximal risk increase and local instability structure. For binary treatment, interpret the treatment axis as finite differences $\delta_A \Phi$ rather than a true derivative.

Connection to Cox regression: In the Cox model, $\log \lambda(s) = \log h_0(t) + \beta' s$. Since $\Phi = -\log \lambda$,

we have $\nabla_x \Phi = -\beta$ (independent of baseline hazard h_0). The field framework generalizes this to nonlinear $\Phi(s)$.

Directional Effects as Local Geometry

An intervention induces a local displacement $\Delta s = (0, \dots, 0, \Delta a)$. The induced change in potential is:

$$\Delta \Phi \approx \nabla \Phi(s) \cdot \Delta s$$

Definitions:

- **Risk-increasing effect:** $\Delta \Phi < 0$
- **Protective effect:** $\Delta \Phi > 0$

These effects are local, state-dependent, and directional. They are **not global scalar quantities**.

ATE as a First-Order Projection

The true object is the field $\Phi(x, a)$.

For binary treatment $A \in \{0, 1\}$, the treatment effect is exactly the finite difference:

$$\delta_A \Phi(x) = \Phi(x, 1) - \Phi(x, 0)$$

Averaging over X yields the ATE:

$$\text{ATE} = \mathbb{E}_X[\delta_A \Phi(X)]$$

ATE is a first-order projection of a high-dimensional geometry.

Survival scale note: For survival outcomes, $\delta_A \Phi(x) = \Phi(x, 1) - \Phi(x, 0) = \log\{\lambda(x, 0)/\lambda(x, 1)\}$ is the negative log hazard ratio. The HR field is $\exp\{-\delta_A \Phi(x)\}$.

Case 1: Homogeneous effects ($\text{Var}_X[\delta_A \Phi] \approx 0$)

When $\delta_A \Phi(x) \approx c$ (constant across x), $\text{ATE} \approx c$. No information is lost.

Case 2: Heterogeneous effects ($\text{Var}_X[\delta_A \Phi]$ large)

ATE remains well-defined but collapses a distribution into a single number, losing information about where effects are strong vs weak, positive vs negative.

Case 3: Positivity violation (structural discontinuity)

When $P(A = 0 | X = x) = 0$ for some x , the field $\Phi(x, 0)$ is undefined in that region. ATE involves integration over regions where $\Phi(x, 0)$ does not exist. This is not an estimation problem—it is a **domain problem**.

Positivity Reinterpreted Geometrically

Standard positivity requires $0 < P(A = 1 | X = x) < 1$.

In the field view: positivity violations correspond to **disconnected regions** of \mathcal{S} —geometric constraints, not estimation failures. Rather than treating these as problems to fix, we map them as the defining boundaries of the clinical landscape.

Relation to MSM and the g-formula

The g-formula as Integrated Flow

The g-formula computes the expected accumulation of local risk along trajectories constrained to follow a specific path through state space.

MSMs as Average Projections

MSMs estimate the average directional flow of the system through the risk field, projected onto the treatment axis (Cole and Hernán 2008).

Weight Instability as a Geometric Signal

When positivity is violated, inverse probability weights diverge. Geometrically, this corresponds to disconnected regions where admissible trajectories do not exist. Weight explosion is a signal that the causal projection is being forced across regions with no valid geometric connection.

Simulation Study

We simulated a 2D state space with survival outcomes:

- X_1 : Systolic blood pressure (100–200 mmHg)
- X_2 : Inflammatory marker (0–10 mg/L)
- A : Binary treatment (antihypertensive)
- Structural constraint: If $X_1 > 160$, then $A = 1$ (mandatory per guideline)
- Heterogeneous effect: Treatment benefit increases with X_1

Estimation notes: We enforce the structural rule $X_1 > 160 \Rightarrow A = 1$ deterministically to induce a structural positivity boundary. We estimate the hazard surface $\hat{\lambda}(s)$ using generalized additive models (GAMs) with tensor product smooths, fitting separate surfaces for $A = 0$ and $A = 1$. From these, we obtain $\hat{\Phi}(s) = -\log \hat{\lambda}(s)$ and compute $\delta_A \Phi(x) = \hat{\Phi}(x, 1) - \hat{\Phi}(x, 0)$. We report both $\delta_A \Phi$ and the HR field $\exp\{-\delta_A \Phi\}$. Visualization is restricted to regions with empirical support; disconnected regions (no joint support for both $A = 0$ and $A = 1$) are rendered as boundaries where $\delta_A \Phi$ is undefined. Alternative estimators (e.g., neural networks, random survival forests) could be substituted; the conceptual framework does not depend on a specific method.

Results (N = 3,000):

Method	Output	Interpretation
Cox HR	0.55	Treatment reduces hazard by 45%
IPTW ATE	0.20	Treatment increases 1-year survival by 20 percentage points
HR field $\exp(-\delta_A \Phi)$	0.19–0.66	Effect varies ~3.5-fold across state space

Traditional methods produce single numbers. The Field approach reveals that treatment benefit varies ~3.5-fold across the state space, with the largest effects in high-BP patients—precisely those for whom the guideline mandates treatment.

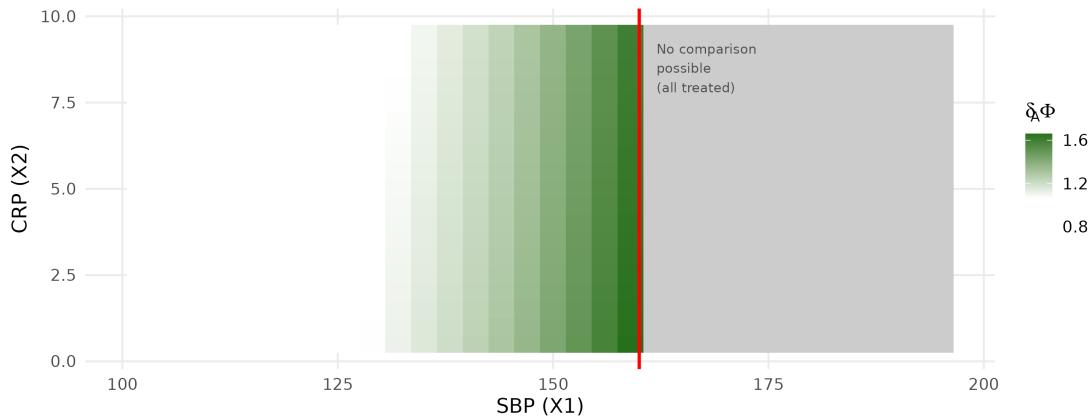
Key Messages:

1. Traditional methods are not wrong—Cox and IPTW correctly estimate average effects
2. But averages hide structure—HR = 0.55 does not reveal ~3.5-fold variation
3. Positivity violation is information—the gray zone reflects clinical logic

Field-Based Approach: Structure Beyond Average Effects

Treatment Effect Field

Cox HR = 0.55 / IPTW ATE = 0.2 (single numbers) vs Field shows structure



Effect Gradient by SBP

Treatment benefit increases with blood pressure

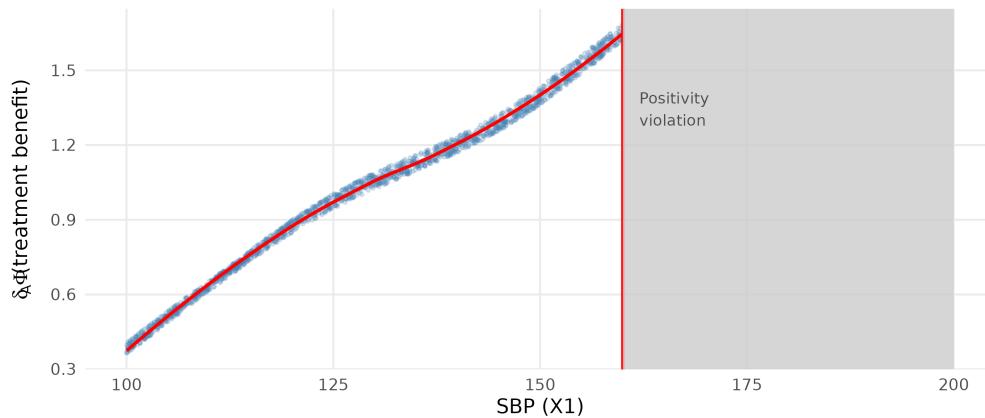


Figure 1: Treatment effect field showing heterogeneity and structural boundary at $X_1 > 160$. Gray region indicates positivity violation where comparison is structurally impossible.

- Field-based output is a map, not a number

Relation to Existing Heterogeneity Research

Differentiation from CATE

Aspect	CATE / HTE Research	Epidynamix
Core question	“What is the effect for subgroup $X = x$?”	“What is the structure of the risk field?”
Output	$\tau(x) = \mathbb{E}[Y(1) - Y(0) \mid X = x]$	$\Phi(s), \nabla\Phi$, structural boundaries
Positivity violation	Estimation problem (trim, extrapolate)	Information (map the boundary)
Counterfactuals	Required	Not required
Goal	Better effect estimation	Different question entirely

Key distinction: CATE still asks “what is the effect?”— just conditional on X (Wager and Athey 2018). Epidynamix asks “what does the risk landscape look like, and where can we not go?” Crucially, CATE treats positivity violations as missing data; Epidynamix reports boundaries explicitly as part of the result.

Connection to Structural Nested Models

The local effect $\delta_A \Phi(x)$ shares conceptual ground with the “blip function” in Structural Nested Mean Models (Robins 1994), which also models treatment effects as functions of patient state. The key difference is interpretive: SNMM aims to estimate causal effects under sequential exchangeability, while Epidynamix treats the effect surface as a geometric object to be mapped.

Novelty Claim

We do not claim to invent new mathematics. Potential landscapes exist in physics and systems biology (e.g., Waddington’s epigenetic landscape (Waddington 1957)). Concurrent work by Leizerman (Leizerman 2025) develops a “Unified Causal Field Theory” using differential geometry and

fiber bundles—a more general mathematical formalization applicable across domains. Our contribution is complementary: applying this geometric lens specifically to clinical RWD, where positivity violations are common, effects are entangled with state, and the average-effect question may be structurally unanswerable.

This is a **reinterpretation**, not an invention—a new language for an old problem.

Discussion

The Epidynamix framework offers a complementary perspective to standard causal inference (Rubin 1974; Hernán and Robins 2020). It does not replace existing methods; rather, it clarifies their domain of validity.

When ATE works: If the risk field is smooth, low-curvature, and well-connected (positivity holds everywhere), the ATE is an accurate summary.

When ATE fails: If effects are highly heterogeneous, or if structural constraints create disconnected regions, the ATE may be misleading or undefined. The field approach provides richer output—a map rather than a number.

Practical implications:

1. Report effect heterogeneity alongside average effects
2. Visualize the treatment effect landscape when possible
3. Treat positivity violations as findings, not errors
4. Consider whether the causal question is answerable before estimating
5. Report HR field maps ($\exp\{-\delta_A \Phi\}$) alongside scalar summaries

Limitations: The framework is conceptual; practical estimation of $\Phi(s)$ requires flexible models (e.g., GAMs, neural networks) and careful validation. Extension to time-varying treatments and high-dimensional states remains an open challenge.

Conclusion

Newtonian mechanics is not wrong—it is flat-space physics. Causal inference is not false—it is a low-curvature approximation.

Real-world epidemiology, with its structural constraints and heterogeneous effects, may require a geometric view. The Epidynamix framework offers one such lens: treating causal effects not as forces, but as directional movements within a structured risk field.

When the standard causal question cannot be answered, perhaps we should ask a different question.

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