

Vaccines Don't Cause Autism

Cohort-Aligned Evidence from Vaccine Policy Changes

Jordan Lasker
Texas Tech University

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Abstract

Vaccines do not cause autism according to the wider evidence. We assess the contribution and limits of public population data by aligning recorded autism with vaccine schedule changes by birth cohort. The aggregate event study fits continuous jurisdiction-specific trends with a change at the event and pools results across countries. Across 39,753 overlapping comparisons around 404 changes in 40 countries, the uptake-scaled model estimate is 0.973 (95% random-effects interval 0.836-1.131). Adding the assumed shared-bias allowance widens the interval to 0.699-1.354. A matched autism-versus-control contrast is 0.913 (0.759-1.098) on overlapping data. Deterministic recovery checks and pooled fake-date checks support specific features of the pipeline, while a failed dose-response diagnostic limits interpretation as an individual causal risk ratio. The cohort-slope sensitivity describes change across successive birth cohorts, not accumulation within individuals. A complementary hierarchical analysis gives an exponentiated shared mean of 0.937 (95% generalized-profile credible interval 0.599-1.458), conditional on exposure, trend and bias assumptions. Published cohorts, local coverage gradients, interaction analyses and other health outcomes are examined separately on their appropriate scales. The evidence shows no coherent vaccine-linked autism signature. Positive associations and failed diagnostics limit conclusions about particular products, outcomes and subgroups. The population analyses complement the wider evidence; they do not identify every individual effect or establish a universal zero-risk bound.

JEL codes: I12, I18, C23, C54

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1. Introduction

The wider evidence does not support a causal link between vaccines and autism. A meta-analysis of more than 1.25 million cohort children reported a vaccination-autism odds ratio of 0.99 (95 percent CI 0.92-1.06) (Taylor, Swerdfeger, and Eslick 2014). A Danish cohort of 1,224,176 children reported an autism spectrum disorder hazard ratio of 0.93 (0.89-0.97) per milligram of vaccine aluminum (Andersson et al. 2025). These observational associations inform the overall evidence; inverse estimates do not establish protection.

This paper tests whether changes in vaccination uptake are followed by changes in recorded autism in the affected birth cohorts. The primary aggregate event study combines comparisons within countries, then pools country summaries. Complementary analyses examine individual policy changes, shared effects across vaccine-and-place contexts, local coverage gradients and vaccine-pair interactions. A context is a particular vaccine or schedule contrast in a particular setting. The 17- and 23-context models have different membership and answer a different question from the primary schedule-change model.

The primary estimate is close to no abrupt change, but its interpretation depends on exposure measurement, the expected outcome trend and bias assumptions. The dose-response diagnostic fails, and the shared-bias allowance leaves a 25 percent increase compatible with the data. Table 1 summarizes the distinct analyses. Their overlapping observations and different effect scales preclude treating the rows as independent replications of one universal vaccine effect.

Table 1. Principal findings across distinct analyses

Analysis and scope	Estimate	95% interval	Measure and interval type
Schedule changes: primary model; 40 countries; 39,753 comparisons	0.973	0.836-1.131	Uptake-scaled ratio; random-effects interval
Autism compared with control outcomes; 25 countries; 49,966 comparisons	0.913	0.759-1.098	Autism-minus-control model ratio; random-effects interval
Additional cohort-slope analysis; 39 countries; 35,365 comparisons	-1.73%	-8.05% to +5.02%	Percent change per cohort; placebo-calibrated interval
Shared mean across 23 contexts; 23 contexts	0.937	0.599-1.458	Shared mean ratio; generalized-profile credible interval
Shared mean across 17 contexts; 17 contexts; 8,538 cells	0.987	0.638-1.519	Shared mean ratio; credible interval
Vaccine-pair interactions; 27 vaccine-pair contrasts	0.967	0.881-1.063	Interaction multiplier; credible interval
Kindergarten MMR coverage; 14 states	1.002	0.950-1.057	Autism share of special education per 10 coverage points; credible interval
Early-childhood disability records; 6 states	1.002	0.761-1.321	Autism share of disability classifications per 10 coverage points; modified Hartung-Knapp interval

Notes: For a ratio, 1 means no association and 1.25 means a 25 percent increase on the stated outcome scale. The additional cohort-slope row gives percent change across successive birth cohorts, not accumulation within individuals. An interval containing 1 (or zero for percent change) is compatible with no association. A context is a vaccine or schedule contrast in a particular setting. Rows overlap and must not be pooled. The primary shared-bias interval is 0.699-1.354. A ratio below 1 does not establish protection.

Section 2 describes the claim's predictions and the wider evidence, and Section 3 describes the data and methods. Sections 4.1-4.8 report the complementary analyses; Section 4.9 reports the aggregate event study. Sections 5 and 6 examine positive signals and other health outcomes. Sections 7 and 8 give the interpretation, limitations and research priorities. Detailed inventories, model comparisons and reproducibility records are in the supplement.

2. What the vaccine-autism claim predicts

2.1 Observational cohorts provide triangulation, not policy identification

The person-level literature is large but mostly observational. Linked-register, sibling-risk, case-control and ingredient-dose studies rarely identify vaccination through randomization, a valid instrument or a policy discontinuity. Their adjusted estimates triangulate the conclusion but do not enter the policy hierarchy or narrow its intervals. Residual confounding limits individual comparisons; replication across populations, exposures and measured-risk strata remains informative. Randomized developmental endpoints are not autism: the Danish Calmette trial found no difference in 12-month Ages and Stages scores among 4,262 newborns randomized to BCG or no vaccine (Kjærgaard et al. 2016; Supplement S39.2a); MATISSE recorded developmental delay through 24 months in 0.1 percent of maternal RSV-vaccine-arm and fewer than 0.1 percent of placebo-arm infants (Simões et al. 2025).

Large MMR cohorts in Denmark, the United States and Britain (Madsen et al. 2002; Hviid et al. 2019; Jain et al. 2015; Smeeth et al. 2004; Burstain, Jacques, and Burstain 2026), with cohort-timing analyses (Farrington, Miller, and Taylor 2001), report no harmful association (Supplement S76.6).

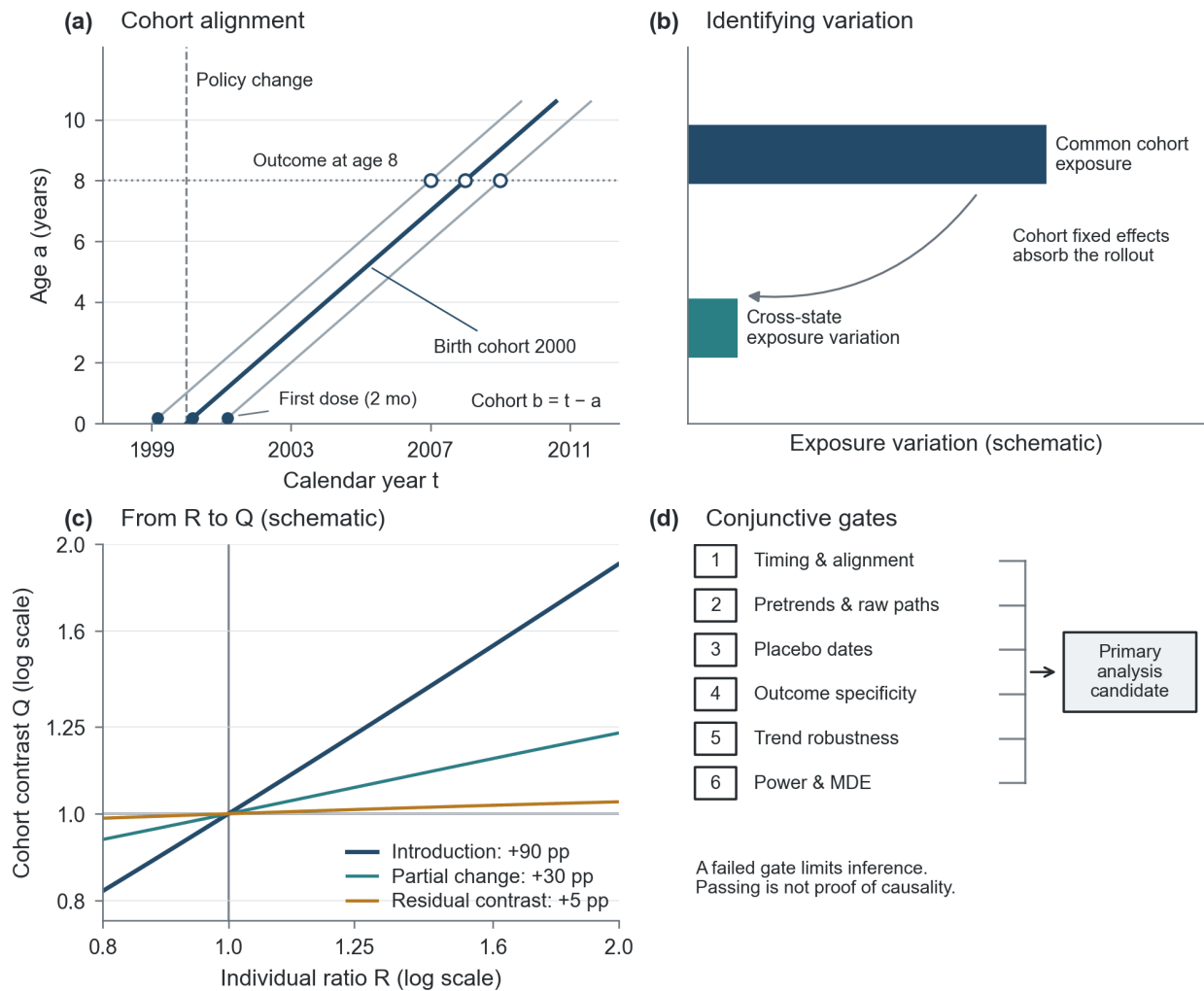


Figure 1. Cohort alignment and exposure scaling. (a) Vaccination is assigned by scheduled age and uptake, and outcomes are observed for the same birth cohorts later in childhood. (b) Cohort effects absorb common rollout, leaving cross-state differences. (c) Under the stated risk-mixture assumptions, the population contrast Q maps to an individual ratio R through the uptake change. Panels (b) and (c) are schematic; no empirical estimate is plotted. The conversion requires a valid exposure contrast and credible comparison.

Ingredient and schedule studies generally do not support the proposed autism gradients. Hviid et al. (2003) found neither an autism association nor an ethylmercury dose response, Price et al. (2010) found no harmful relation with prenatal or infant thimerosal, and DeStefano, Price, and Weintraub (2013) found none with cumulative antigens. Becerra-Culqui et al. (2018) reported HR 0.85 (0.77-0.95) after prenatal Tdap among 81,993 mother-child pairs; the inverse association is not interpreted as prevention. Reverse selection pushes the same way: after an autism diagnosis, children were less often fully vaccinated for vaccines due at ages 4-6 (adjusted RR 0.87, 0.85-0.88), as were their younger siblings (Zerbo et al. 2018). Such selection biases naive vaccinated-versus-unvaccinated contrasts toward protection. In an Israeli cohort of 153,321 children, a crude prenatal-influenza HR of 1.22 attenuated to 0.97 (0.91-1.05) after adjustment (Neeman et al. 2025). Andersson et al. (2025) examined fifty chronic outcomes among 1,224,176 Danish children; its main autism hazard ratio was inverse, but its

supplementary categorical risk differences and subgroup results were not uniformly null or inverse (Section 6.3; Supplement S72). A 2026 systematic review located only three of fifty-nine human studies reporting autism, two of them ecological correlations at critical risk of bias, so its autism conclusion rests on that Danish cohort (Doyon-Plourde et al. 2026).

Published syntheses reach the same conclusion (National Academies/IOM 2012; Di Pietrantonj et al. 2021; Gidengil et al. 2021; WHO 2026; WHO Global Advisory Committee on Vaccine Safety 2025). A dated PubMed search through 30 August 2026 screened 377 unique records (Supplement S39.2a). Search records are not effect estimates. The acquired published-study layer retains 4,713 contrast records with 87 specified participant/source identifiers and fits 37 overlapping native-scale or explicitly bridged families (Supplement S67 and S69). Under the reference bias allowances, the two-cohort childhood-MMR hazard-ratio family gives 0.954 (0.647-1.415); a three-cohort sensitivity bridging incidence-rate and hazard ratios gives 0.947 (0.694-1.293)¹. Four prenatal-influenza cohorts give HR 1.012 (0.779-1.315), and the two-study MMR case-control family gives OR 0.926 (0.585-1.513). These summaries do not enter the policy-event hierarchy. Corrected Jain et al. estimates enter only one age/dose/familial-risk contrast per fit, avoiding repeated use of a single cohort model. This is a structured public search and overlap audit, not a duplicate-screened PRISMA systematic review. Thimerosal-removal series in Denmark, Sweden, California and Quebec did not establish a sustained decline attributable to removal (Madsen et al. 2003; Stehr-Green et al. 2003; Fombonne et al. 2006; Schechter and Grether 2008), but uncontrolled before-after comparisons remain descriptive.

2.2 Tests of proposed mechanisms

Proposed mechanisms predict persistence, dose response, timing or susceptibility, and the tests of those signatures are summarized in Supplement S76.11.

Known harms calibrate the evidence standard. First-dose MMRV versus same-day MMR plus varicella gives seizure RR 1.98 (1.43-2.73) on days seven through ten, about one additional case per 2,300 doses (Klein et al. 2010); the comparator still carries measles-vaccine fever/seizure risk. Surveillance detected RotaShield intussusception and later smaller second-generation rotavirus risks (Murphy et al. 2001; Yih et al. 2014; Weintraub et al. 2014). The 1976 influenza program's Guillain-Barre excess, just under one per 100,000 vaccinations, did not recur in later seasons (Schonberger et al. 1979; Hurwitz et al. 1981; Kaplan et al. 1982). Pandemrix narcolepsy gave HR 2.92 (1.78-4.79) among Swedes twenty or younger (Persson et al. 2014); second-dose BNT162b2 myocarditis concentrated in young males (Mevorach et al. 2021), and dengue vaccination harmed baseline-seronegative recipients (Sridhar et al. 2018). Credible signals specify product, phenotype, timing and subgroup, with record confirmation and replication. Passive reports alone generate signals, not causal estimates.

2.3 Why recorded autism prevalence rose

Recorded prevalence can rise through identification rather than vaccine injury. IDEA added autism as an educational category in 1990 (U.S. Department of Education 2024); screening and diagnosis age alter identification (Johnson and Myers 2007; Parner, Schendel, and Thorsen 2008). Fiscal incentives, insurance mandates, Medicaid waivers and provider capacity change classification and treated prevalence (Cullen 2003; Dhuey and Lipscomb 2011; LaClair et al. 2019; Shea et al. 2021). Criteria/reporting changes explain much of Denmark's rise (Hansen, Schendel, and Parner 2015); U.S. mandates increased treated prevalence (Mandell et al. 2016), alongside substitution and diagnostic accretion (Shattuck 2006; King and Bearman 2009). These mechanisms predict policy breaks, younger diagnosis, category substitution and growth after coverage stabilizes. They are causes of recorded prevalence, not variation to erase.

2.4 Policy-driven diagnosis is not vaccine injury

These designs observe identified disability, not latent biological incidence. Adding a reporting category, reimbursing treatment or reclassifying pupils can raise counts without changing incidence. A diagnosis policy can confound a vaccine comparison when its effect on recording is associated with the exposure contrast and is not adequately captured by the counterfactual model. We date insurance mandates and child-specific waivers, absorb common report-year and cohort shocks, combine substitutable categories and compare outcome systems. These steps keep institutional effects from being conflated with vaccination, not remove them from prevalence. A national diagnosis reform inseparable from a national vaccine change leaves the vaccine effect unidentified; that series is downgraded, not adjusted by assertion.

Harm predicts cohort differences after uptake changes when follow-up covers latency and ascertainment is comparable; withdrawal reverses that prediction. Interactions test departures from multiplicative separate effects, equivalently additivity on the log-risk scale. The predicted shape follows the exposure history and follow-up ages; a different trajectory requires an explicit exposure or effect model. Timing alone cannot distinguish vaccine from diagnosis effects; a credible comparison and adequate sensitivity remain necessary.

3. Testing the vaccine-autism claim

3.1 Populations, exposures, and autism outcomes

The main U.S. outcome is the IDEA Section 618 child count of children served under the autism category, combined with Census single-age denominators into state-by-birth-cohort rates; it is an administrative service measure, not clinical incidence. ADDM supplies records-based surveillance prevalence at age eight for a few sites, and NSCH and NHIS supply parent-reported diagnoses. International outcomes come from special-education and school-census counts, specialist-care, claims and hospital-discharge registers, and Brazil's 2022 Census. These systems are analyzed separately because their case definitions differ.

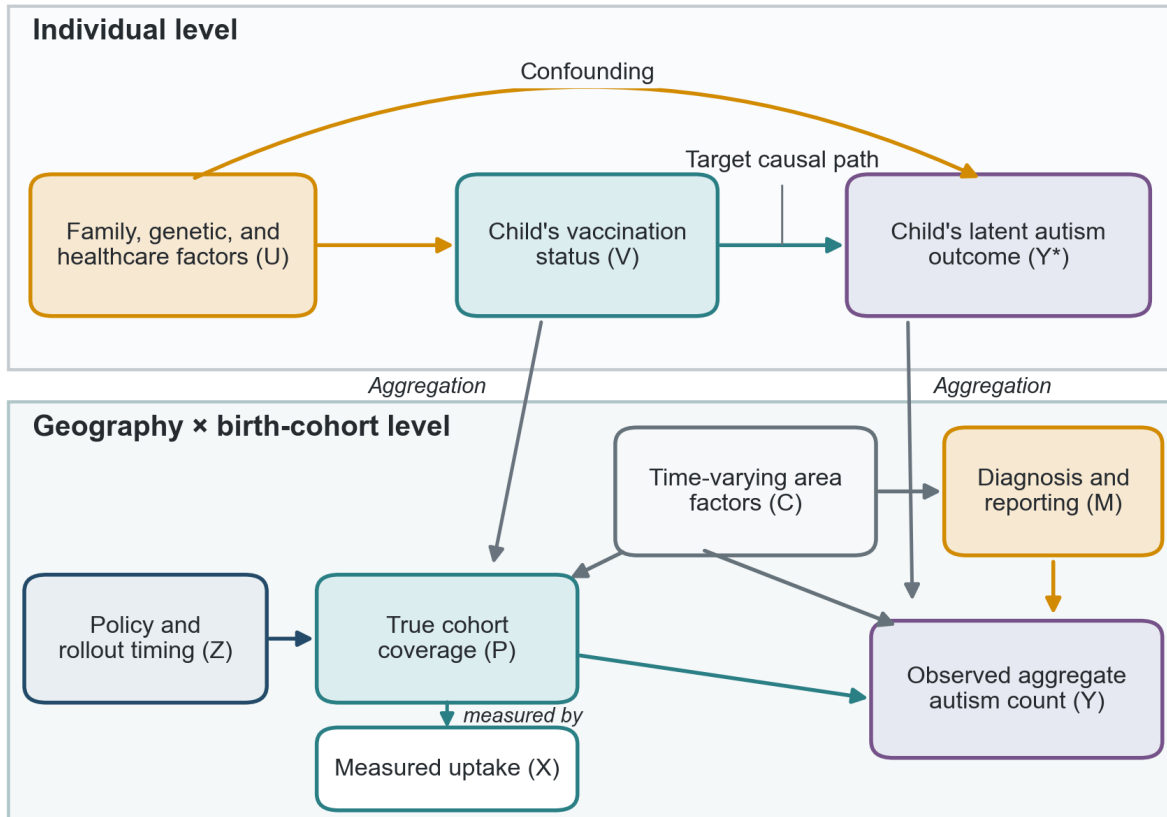


Figure 2. Routes from vaccination and diagnosis processes to recorded autism. Family, genetic and health-care factors can affect both vaccination and diagnosis. Diagnosis policy and reporting affect observed counts, and coverage is measured with error. The design must distinguish exposure-related differences from these other pathways.

Exposure is assigned to each birth cohort from scheduled age and measured uptake: provider-verified NIS receipt in the United States, and national, regional or municipal coverage series elsewhere, joined to outcomes only when geography and timing are compatible. Japan uses the continuous cohort MMR coverage path, because uptake fell several years before the formal withdrawal.

The schedule registry supplies a search frame of 3,379 national and registered subnational introductions, removals, dose changes, eligibility transitions, mandates and coverage shocks. A causal interpretation additionally requires verified timing, cohort-compatible outcomes, a change in uptake and a usable comparison. A recorded date alone does not establish eligibility, and the aggregate sample retains timing flags discussed in Section 8. An event without suitable outcome data supplies no estimate. Table 2 summarizes the principal data systems; Supplement S41 and S59.1 document eligibility and source coverage.

Table 2. Principal data systems and identifying variation

Setting	Exposure/event	Outcome	Sample	Design status
Japan	MMR withdrawal, 1988-96 cohorts	Cumulative ASD incidence to age 7	9 cohorts	Interrupted cohort trend; outcome-compatible
U.S.	PCV7 rollout intensity	IDEA autism at age 8 plus control categories	353 state-cohorts	Partly exposed reference; spline exposure mapping fails (S52)
U.S.	RotaShield 1998-99 measured uptake	IDEA autism at age 8	353 state-cohorts	Pulse intensity and spline; one clean lead
U.S.	Kindergarten MMR coverage, cohorts 2014-16	IDEA autism at age 8 plus control categories	144 state-cohorts	SchoolVaxView dose-response; only three pandemic-adjacent cohorts
U.S.	PCV7 and RotaShield in recurring ADDM sites	Records-based ASD prevalence at age 8	48 primary site-cohorts	Exact small-cluster event study; clinically closer but low power
England	Rotavirus 2013; MenB 2015; 6-in-1 2017	Primary-need ASD, ages 5-12	120 national cells	Flexible splines and nested schedule states
Scotland	Rotavirus cohort ramp and schedule interaction	Education-recorded autism ASN	512 LA-years	Intensity event study; low power
Wales	Rotavirus cohort ramp and PCV interaction	Education-recorded autism/ALN	220 LA-years	Pretrend fails; ALN system break
Northern Ireland	Rotavirus, MenB, and 6-in-1 schedule states	Autism prevalence by school grade	155 grade-years	Exact APC alias; event profile fails
Sweden	PCV and later schedule coverage	Annual specialist-care F84 patients	1,122 F84 cells	County intensity; outcome/pretrend caveats
Spain	Regional varicella convergence	ASD/PDD share of SEN pupils	266 region-years	DiD/event study; failed joint pretrend
Germany	PCV, MenC, varicella Land coverage	F84 inpatient discharges plus F80/F82/F90 controls	4,800 cells; 10 covered Länder	Independent falsification panel; not incidence
Italy	Varicella and rotavirus harmonization	No compatible regional fixed-age ASD panel	7 national cohorts	Exposure-ready; outcome-blocked
Canada	Eight staggered provincial rotavirus introductions	PHAC CCDSS autism incidence and prevalence, ages 1-19	382 province × year × outcome cells	Naive leads fail; province-trend cohort-saturation estimates are null
Czechia	District birth-cohort MMR coverage	District F84 treated-patient stock, ages 4-6	77 districts; 9 years	Nominal result fails older-cohort and trend diagnostics
Colombia	Municipality antigen coverage	School-recorded named disabilities	1,113 municipalities	Approximate cohorts; specificity and timing fail
Brazil	Municipality cohort-window MMR coverage	2022 Census autism/disability by age and sex	4,794 municipalities	Very precise exploratory one-Census cohort approximation
Peru	District immunization-service composition	Voluntary named disability registrations	300 districts; 7 years	Positive result fails future-exposure and specificity gates

Notes: Counts refer to the harmonized analysis panels. Outcome systems remain separate and are not treated as interchangeable clinical measures.

3.2 Jumps and the individual-scale mixture model

A jump is a sharp, dated change in the share of a birth cohort vaccinated, following an introduction, withdrawal, deferral or eligibility change. The individual-policy program inventories 56 candidate changes across fourteen countries and one multi-country entry; its alternative specifications and estimability assessments are documented in Supplement S60.1. This inventory is distinct from the broader aggregate event study in Section 3.5. National introductions supply some of the largest uptake changes: U.S. PCV7 increased any-dose uptake by 87.9 percentage points, England rotavirus by 88.3, and Sweden PCV reached 98.0 percent.

For geography g and birth cohort b , the count model is

$$\mathbb{E}[Y_{gb} \mid X] = \exp(\alpha_g + \lambda_b + \beta X_{gb} + h_g(b) + \log N_{gb})$$

where Y is the autism count, N is the cohort denominator and X is measured exposure; the geography- and cohort-indexed terms are fixed effects, and the geography-specific function of birth cohort is the chosen counterfactual path. A coefficient on an arbitrary X is a reduced-form rate ratio on its native contrast, not a vaccinated-versus-unvaccinated risk ratio. The jump program replaces the exponential exposure term with the mixture factor $1 + p(r - 1)$, where p is the measured share of the cohort exposed and r is the rate ratio of a vaccinated relative to an unvaccinated child. The homogeneous-mixture map $Q = [1 + pH(R - 1)]/[1 + pL(R - 1)]$ describes a specified high-versus-low comparison under common baseline risk and homogeneous effects. An arbitrary adjusted rollout coefficient need not equal Q . Illustrative power calculations under this map are not evidence that the actual PCV7 design detects or excludes the corresponding individual risk. Figure 3 shows the failure: harm injected in proportion to measured receipt can return a rollout coefficient below one. The conversion is therefore applied to measured jumps, not to arbitrary coefficients.

For two vaccines A and B , the ecological interaction is the departure of the joint-high cell from log additivity:

$$\log \mathbb{E}[Y] = \alpha + \beta_A A + \beta_B B + \theta(A \times B) + \mathbf{Z}'\gamma$$

The receipt-mixture interaction expresses this contrast on a conditional individual scale. A cohort's rate mixes four receipt states (neither, A only, B only, both) in their measured shares, with multipliers 1, r_A , r_B and $r_A \times r_B \times \theta$, so θ is the extra multiplier for a child who receives both vaccines beyond the product of the single-vaccine effects. A θ of 1 means multiplicative separate effects. A θ above 1 means a joint risk above their product; whether the joint risk exceeds the unexposed baseline also depends on r_A and r_B . Joint receipt must be measured or bounded; nearly simultaneous rollouts with empty off-diagonal states leave θ unidentified, not zero.

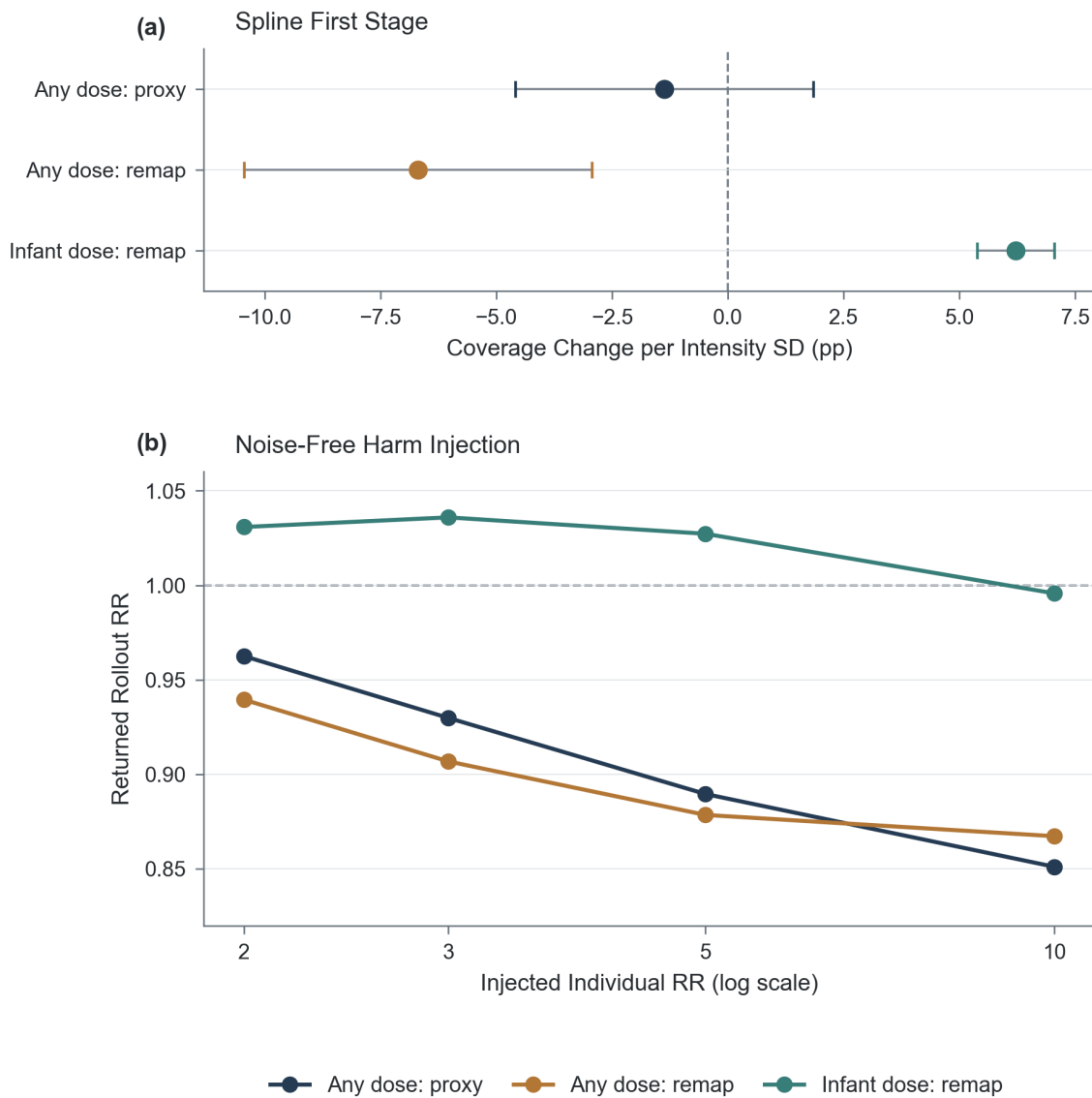


Figure 3. Exposure alignment limits the PCV7 spline. (a) Measured uptake changes with state-cluster CR1 95 percent intervals. (b) Twelve noise-free injections at hypothetical individual risk ratios of 2, 3, 5 and 10. Nine fitted rollout ratios fall below one, demonstrating a failure of the exposure-to-effect mapping. These are simulations. Any-dose coverage concerns pneumococcal-containing receipt; the birth-window remap assumes ages and interview timing within each band.

3.3 Calibration, gates and the hierarchy

Each jump's interval is calibrated against placebo breakpoints: the coverage path is shifted at least three cohorts earlier within pre-exposure data, and the calibrated standard error is the largest of the model standard error and the spread and root mean square of the placebo log r. Standard errors are clustered at the level of exposure assignment, with wild-cluster, sign-flip or permutation diagnostics when clusters are few (Cameron, Gelbach, and Miller 2008; Conley and Taber 2011). NIS sampling uncertainty is propagated by joint redraws (Kish 1965), and outcome families use Holm, Benjamini-Hochberg or stepdown adjustment (Holm 1979; Benjamini and Hochberg 1995; Romano and Wolf 2005). The estimator gate follows the staggered-adoption literature (Goodman-Bacon 2021; Callaway and Sant'Anna 2021; Sun and Abraham 2021; Borusyak, Jaravel, and Spiess 2024), and pretrend screening and sensitivity follow Roth (2022) and Rambachan and Roth (2023). A rejected pretrend challenges the chosen counterfactual; an untestable pretrend is not a passed pretrend.

A context enters the pooled synthesis only if it passes the D4 gate: an autism-specific outcome; no duplicated observations; a future-exposure placebo, with exposure shifted three cohorts later on identical cells, whose interval includes one under every specified trend kernel; no failed pretrend check; and correct response to synthetic harm, both with injected effects of 1, 2, 3 and 5 on noise-free data and with a doubling added to the observed outcome. A stricter two-sided placebo family, with future shifts of two to four cohorts and past shifts of two and three under four kernels, is a sensitivity. These analyses were not preregistered. Analysis choices were documented during the project, including after earlier results had been examined. Rule files and hash records document computational settings and provenance; they do not establish preregistration. The supplement identifies the choices and their timing; intervals condition on the gate decisions rather than accounting for all specification and selection uncertainty.

U.S. PCV and RotaShield share one joint likelihood of 1,469 cells. U.S. rotavirus 2006 remains inside it but is untied from the shared mean, because its future-exposure placebo passes only after a post-hoc reporting-break screen (unscreened 1.306, 1.012-1.675) and its identification diagnostics do not justify sharing the mean. Each context carries an assumed bias standard deviation of 0.35 or 0.70 on the log scale, shared bias has standard deviation 0.15, the shared mean has a Normal(0, log 2) prior, and product and context heterogeneity are estimated. The expanded model uses each original outcome block once and treats alternative specifications as alternatives, not additional studies.

3.4 Generalized-claim tests, power, negative controls and convergence

Generalized-claim tests impose common effects across contexts, within products or harm everywhere, using the restricted 15-context likelihoods and bias terms (Supplement S62). Exclusion requires a posterior upper 97.5th percentile below the threshold; robust exclusion additionally requires agreement across doubled-bias and omission fits. Whole-pipeline power uses seventeen scenarios, 400 replicates each, with common ratios 1.05-1.50 or product-specific harm, exposure/gate checks and serial/cohort bootstrap noise before likelihood and hierarchy refits. Candidate controls replace autism on identical cells. Their product-specific exclusion restrictions are assumptions, including for injuries, sensory/physical disability and asthma. The rule distinguishes UNINFORMATIVE intervals much wider than autism's, CONSISTENT intervals including one, and BIAS SIGNAL otherwise. Consistency is not validation; reused-cell synthetic controls check convergence, not replication.

3.5 The aggregate event study

The aggregate event study aligns dated vaccine schedule changes with recorded autism by birth cohort. Its data layer contains 3,145,790 primary autism cells for 29,170 jurisdictions in 33 countries, with cohort-level coverage and 3,905 adjudicated primary events. The design inventory contains 95,462 candidate comparisons in 41 countries; the estimating sample is reported in Section 4.9. Series with fewer than four distinct calendar years in the estimation window enter sensitivity analyses only. The minimum-calendar-span rule was chosen after inspecting diagnostics. Its model intervals do not capture all uncertainty from data-informed selection. Specification choices and their timing are documented in Supplement S74, S77 and EA2.

Each comparison fits a quasi-Poisson model to one jurisdiction's autism series over the six birth cohorts before and after the first affected cohort, with age or band effects, one continuous cohort trend (linear in the primary specification) and a step at the change (Supplement Figure S114). The raw step is the percentage shift in the autism rate. The uptake-scaled working parameter r rescales it by the change in uptake, p , assuming that the cohort's autism rate changes by the factor $1 + p(r - 1)$ (Section 3.2), linearized as $\log[1 + p(r - 1)] \approx p \log r$; this is a local approximation near $r = 1$. When both cohorts have nonzero uptake, the exact contrast uses the high- and low-uptake mixture ratio in Section 3.2. Uptake p is often modeled, broadcast from a higher level or set to zero before a programme; classical uptake error can attenuate the fitted log effect, but correlated, systematic or nonlinear exposure error can bias it in either direction (Sauerbrei et al. 2014); and the outcome is recorded diagnoses in administrative systems, not biological incidence. U.S. comparisons use measured sub-state coverage where available.

The time axis is birth cohort, not time since vaccination within a person. Under the same effect per vaccinated child and comparison at the same follow-up age, an abrupt uptake change predicts a step between cohorts. Within-person accumulation or latency does not by itself imply a slope across successive birth cohorts. Gradual uptake, changing exposure histories or an age-mixed reporting system require their own exposure and observation-time mapping.

Within a country, comparisons are pooled by profile likelihood with jurisdiction-clustered variance, widened to the envelope of the linear, quadratic and spline trends' intervals. Countries are pooled by random effects with a between-country standard deviation of 0.33 on the log scale, estimated from earlier placebo analyses and held fixed here (Supplement Figure S115). Intervals are reported three ways: random effects, the headline interval; and, as sensitivities, the assumed-allowance interval, which adds an assumed shared bias of 0.15 to the random-effects variance, and the placebo-calibrated interval, with bias components estimated at six fake dates seven to twelve cohorts before each change.

The co-primary matched contrast fits autism and specified control outcomes recorded by the same systems, for the same jurisdictions, ages and years, around the same changes with the same uptake paths, and takes the difference of their country steps; the 77 control categories were selected using record structure and exclude categories that absorb autistic children, and have their own gates (Supplement S77).

Validation uses deterministic effect injection, fake event dates and comparison outcomes. The headline model and matched contrast recover the specified injected effects within the specified 10 percent log-target tolerance, and their pooled placebo intervals cover zero at all six dates. These checks assess the specified perturbations and pooled diagnostics; they do not establish absolute-receipt validity or repeated-sample interval coverage. Brazil's school census fails its recovery check and is report-only. The school-entry-only check and dose-response check also fail (Sections 4.9 and 8; Supplement S74, S78 and EA1).

4. Results across policy contrasts

The aggregate event study (Section 4.9; Figure 13) reports an uptake-scaled model estimate of 0.973 (95 percent random-effects interval 0.836-1.131). Sections 4.1-4.8 report complementary designs and evidence layers.

4.1 Every jump on the individual scale

Of the 57 jump rows (the 56 inventoried jumps, with the NHIS part of the U.S. survey jump carried separately), 37 have a checked individual-scale estimate and 31 a placebo-calibrated interval. None of the 37 headline intervals excludes one in either direction. Among the calibrated estimates, 16 lie above one and 15 below.

The most precise single estimates, from large national introductions, are discussed in Supplement S76.7.

4.2 The hierarchical synthesis (complementary evidence)

The hierarchical synthesis fits original context likelihoods with explicit context and shared-bias allowances. It shares some source series with the event study and uses a different estimand, estimator and bias model. The 23-context shared-mean model, including a linked Peru context, uses a spline trend and context/shared bias standard deviations of 0.70 and 0.15; its exponentiated shared mean is 0.937 (95 percent generalized-profile credible interval 0.599-1.458). The 17-context reference model supplies the context comparisons below, while the common-effect tests use fifteen contexts. These model families overlap and are not independent confirmations; their sample definitions and provenance are tabulated in the supplement (S67 and EA2).

The 17-context model integrates 17 contexts and 21 represented vaccine or schedule contrasts in 16 original likelihood blocks, comprising 8,538 cells. Its conditional exponentiated shared mean log effect is 0.987 (95 percent credible interval 0.638-1.519). Posterior probabilities that the mean exceeds 1, 1.10 and 1.25 are 47.7, 31.0 and 13.9 percent. These are neither probabilities that any vaccine causes harm nor that all effects are zero. Brazil and Sweden contribute joint multiple-product likelihoods; NSCH and NHIS contribute separate survey-outcome likelihoods with shared U.S. bias sensitivity. Each outcome cell enters its own likelihood once. The target is an average log effect across represented products and settings, conditional on exposure reconstruction, trend models and bias priors, not a population schedule-risk ratio. Context posteriors combine unseparated context bias and heterogeneity and are not debiased individual causal risk ratios (Supplement S67).

The component series differ greatly in precision and identification. England rotavirus gives 0.94 (0.86-1.02) on its own data. U.S. PCV leans harmful, 1.17 (0.97-1.39), while its controls also lean harmful (Sections 4.4 and 5.1). U.S. rotavirus 2006 remains estimated but untied from the shared mean. Some blocks carry little information in either direction: Scotland's count design recovers only about a fifth of an injected effect in its gate test, and the German and Czech blocks respond weakly to an injected rate ratio of 1.5 in the restricted-model simulations. Adding cells does not remove those identification limits.

Different Designs, Same Contrasts: Robustness, Not Replication

In the U.S., Brazil and Hong Kong, the same designs also produce changes in candidate control outcomes

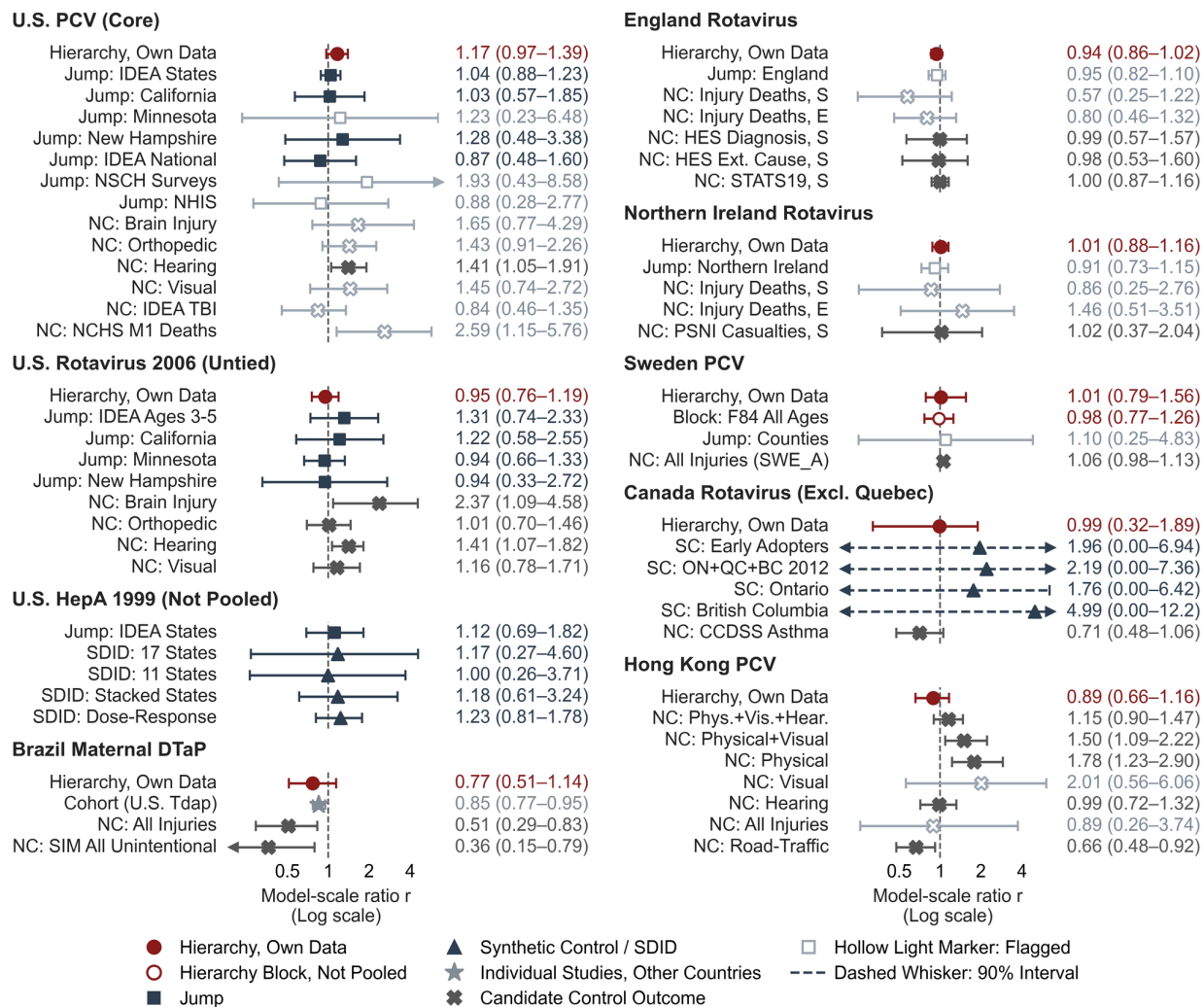


Figure 4. Different designs on the same vaccine-by-place contrasts, in the nine contexts discussed in Sections 4.4 and 4.7. Ratios use each study's exposure mapping and are not uniformly identified individual causal effects. Each panel shows the context's own-data hierarchy posterior (filled red circle), a not-pooled hierarchy block of the same series (hollow red circle), jump estimates (squares), synthetic-control designs (triangles), an individual-level study of the same product in another population (star) and candidate control outcomes (crosses), with 95 percent intervals. Hollow light markers are flagged as lacking a placebo-passing specification, uninformative, report-only or exploratory; dashed whiskers are 90 percent intervals. Control comparisons test specificity only under the no-effect and shared-bias assumptions in Section 4.4. Agreement on reused observations is robustness, not independent replication. The full panel, 108 rows in 20 contexts, is Supplement Figure S107.

Additional three- and twelve-context comparisons give broad intervals around one. Their different membership, weighting and assumptions are described in Supplement EA5; they supply no universal schedule-risk bound.

4.3 Common-effect comparisons and whole-pipeline power

The shared mean averages heterogeneous products and settings; generalized claims may instead predict a common effect, common product effects or harm everywhere. This section's common-effect tests, control calibration and simulations use the restricted 15-context, 14-block model with 5,133 cells. They do not replace the 17-context synthesis, for which the same simulation operating characteristics have not been established.

If vaccination multiplied autism risk by one factor in every pooled context, the data put that factor at 0.964 (0.637-1.455) under the assumed bias allowances. Its posterior probabilities of exceeding 1.10, 1.25 and 1.5 are 0.265, 0.108 and 0.018. With each context's placebo-calibrated allowance in place of the assumed floors it is 0.974 (0.713-1.330). Under the stated rule a common factor of 1.5 is excluded under the assumed allowances, but not robustly: doubling every allowance gives 0.977 (0.540-1.763), and leaving out Colombia's varicella context raises the upper limit to 1.553. Common factors of 1.05, 1.10 and 1.25 are excluded in no specification. The common-effect model fits the data at least as well as the heterogeneous one (Bayes factor 4.64 in its favour, conditional on the compared likelihoods and priors; this comparison does not validate causal identification).

Whole-pipeline simulations of this model, which describe sampling behaviour only, are in Supplement Figure S75.

One input, Colombia's school census in the pandemic school years, moves the synthesis materially (Supplement S76.8).

Control calibration uses 45 candidate outcomes in nine contexts to estimate bias, assuming their biases are exchangeable with autism's. The calibrated common-effect ratio is 0.871 (0.741-1.020), compared with 0.964 (0.637-1.455) under the assumed bias allowances. Adding residual shared log-bias SD 0.15 gives 0.876 (0.632-1.212). The tighter interval therefore depends on a comparable-bias assumption that controls cannot establish for autism-specific errors. The U.S. PCV association remains unresolved because its controls also lean harmful. Full calibration, residual-bias and numerical sensitivities are in Supplement S62.9, S65 and EA5.

These statements concern a common or product-level effect, the prediction of a generalized claim. They do not bound a harm confined to one product, one context or an unmeasured subgroup: with a rate ratio of 1.5 confined to MMR, the common-effect model would nevertheless place its upper limit below 1.5 in 56 percent of simulated replicates. Nor do they replace the heterogeneous shared mean, the more permissive estimand, which remains compatible with moderate increases.

4.4 Negative controls: designs that find "effects" on injuries reveal bias

Negative controls ask whether a design also finds associations with outcomes assumed to be unaffected by the vaccine. A non-null control estimate indicates bias only if that exclusion restriction holds. Injury categories can be more credible candidates than broad disability or asthma outcomes, but even injuries may respond indirectly through health or behaviour. Such findings test specificity; they do not, by themselves, prove that an autism association is confounding. Designs in three places fail this test, although the stated verdict for the U.S. core likelihood is MIXED.

Testing One Vaccine Effect on Autism Across 15 Contexts

Restricted 15-context model. A common effect is 0.96 (0.64–1.46): RR 1.5 is excluded under the assumed and placebo-calibrated allowances, but not if they double or without Colombia or its 2020–21 school years; RR 1.25 is not excluded under the assumed or placebo-calibrated (A2) allowances, or in any other row.

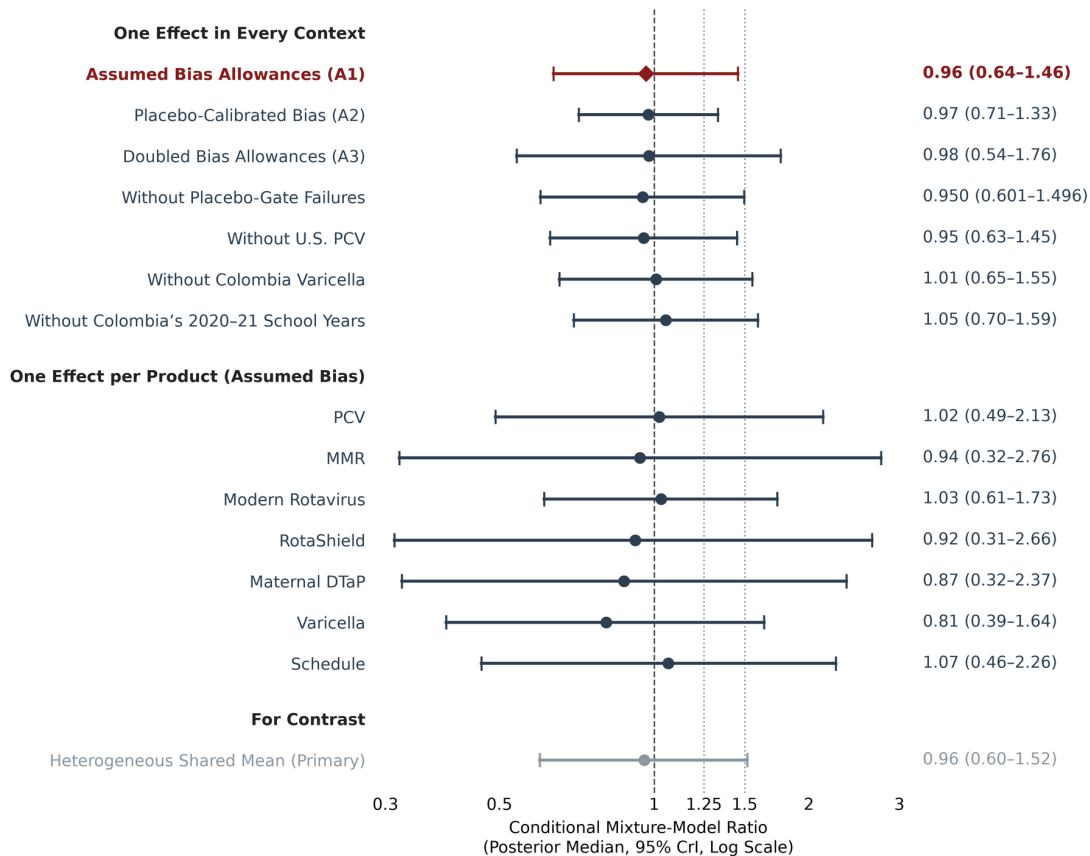


Figure 5. Common-effect and shared-mean models across fifteen contexts. Points show conditional posterior medians; whiskers show 95 percent credible intervals. Assumption labels are A1, assumed bias; A2, placebo-calibrated bias; and A3, doubled bias. Omission rows remove selected contexts or Colombia pandemic school years. The product-specific and heterogeneous shared-mean rows estimate different quantities from a common effect in every context. Dotted lines mark ratios of 1.25 and 1.50. Exclusion of 1.50 depends on bias allowances and context membership; no row excludes 1.25. Simulation operating characteristics are in Supplement Figure S75.

In the United States, the core PCV context and its candidate controls lean harmful together, so the stated verdict is MIXED; the Brazilian maternal DTaP and Hong Kong PCV designs return bias signals on injury or physical-disability controls, in both directions; and England's road-casualty and Sweden's injury control intervals include one on the same cells as autism. The context-by-context results are in Supplement S76.1.

What the negative controls imply for agreement among designs and for the hierarchy's bias terms is set out in Supplement S76.12.

4.5 Withdrawals, pulses and deferrals

Sign-reversing and pulse designs make predictions that a smooth confounding trend does not. Yokohama's MMR coverage fell across the 1988-1992 birth cohorts and was zero from 1993, a jump of -69.8 percentage points, yet cumulative ASD incidence to age seven kept rising (Figure 6). The illustrative predicted-decline path holds the unvaccinated background rate at its 1988-implied level; it is an assumption-indexed benchmark, not an estimated counterfactual. On the individual scale the nine cohorts carry little information: the Japan context's own data give 0.85 (0.28-2.32), the jump fit on Yokohama's Kohoku ward is uninformative, and the native coverage gradient is 0.981 (0.805-1.196) per ten points. The observed series lacks the decline in the fixed-background illustration, but the broad individual-scale interval does not establish a precise withdrawal-effect bound.

RotaShield was available nationally for about a year before withdrawal, and measured state uptake ranged from near zero to about fourteen percent. Including NIS exposure-sampling uncertainty, the IDEA estimate is RR 0.996 per ten points (0.945-1.051), and the individual-scale context gives 0.87 (0.36-1.83). The pulse supplies a distinct short-lived uptake contrast, with limited preperiod support and a failed strict two-sided placebo family. Its precision on the stated exposure scale is not a general individual-risk guarantee, and it does not test the vaccine's known acute intussusception risk.

The 2007-2009 Hib booster shortage cut on-schedule booster receipt in the 2007 birth cohort by 50.0 percentage points while other vaccines at the same visit barely moved. This is a timing contrast, a booster by 19 months versus late or never, not vaccination versus none. The corrected national estimate is 1.055 (0.717-1.553), and the cross-state dip-intensity design gives 0.992 (placebo-calibrated 0.811-1.212), with event-study leads that do not reject ($F = 1.09$, $p = 0.37$).

4.6 Interactions, schedule load and saturation

The pairwise interaction analysis tests the specific hypothesis that joint risk exceeds the product of separate-vaccine risks. A general schedule-load claim need not imply that particular interaction. Across 27 modeled pairs (24 U.S. vaccine pairs, two English sequences and the U.S. PCV-by-RotaShield core term), the interaction hyper-mean, the exponentiated mean log theta, is 0.967 (0.881-1.063). A mean stacking penalty of ten percent or more has posterior probability 0.46 percent, and of 25 percent or more less than one in 100,000. Between-pair heterogeneity is small, and a new exchangeable pair is predicted at 0.967 (0.762-1.229). U.S. state pairs carry 97.5 percent of the information; dropping them, treating them as independent rather than correlated, or dropping the one pair whose interval excludes one, influenza by Hib deferral (Section 5.1), leaves an interval that includes one (Supplement S64.1). The layer is pairwise and area-level: it does not establish same-child biology, and Japanese, Swedish and German pair terms are not identified.

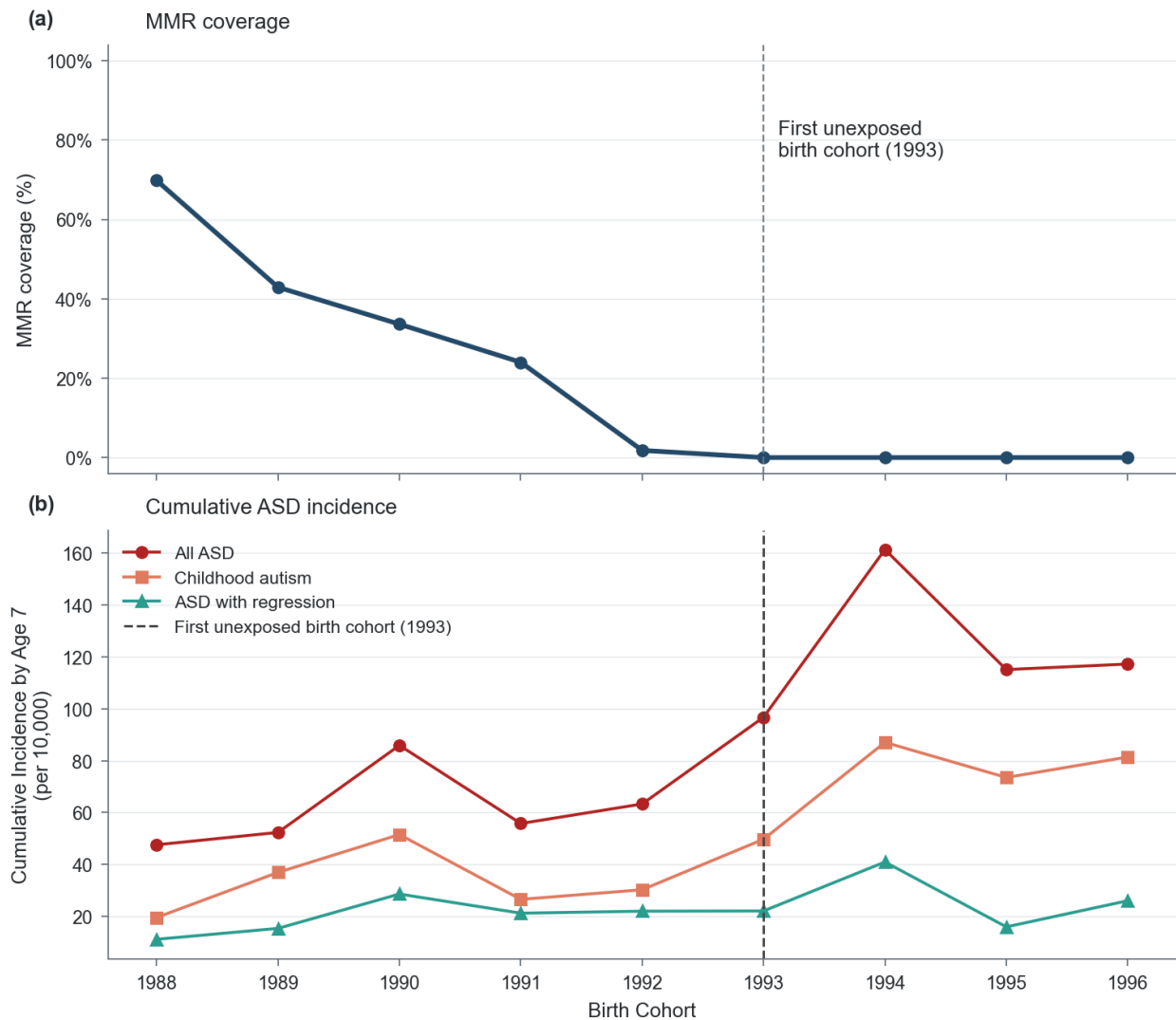


Figure 6. Yokohama MMR coverage and cumulative ASD incidence aligned by birth cohort. Coverage approaches zero while observed identification continues upward. The dashed line marks the 1993 birth cohort, the first cohort with no MMR coverage; the MMR vaccine was withdrawn in April 1993.

The older ecological test answers a population-scale question. The U.S. PCV-by-baseline-schedule interaction is an ecological departure from log additivity of 1.001 (0.989-1.012 after joint NIS uncertainty), on a joint ten-point coverage contrast rather than a per-child multiplier. Re-expressed on the same (+10-point) by (+10-point) contrast, the six-site ADDM analogue is 1.018 (0.280-3.700), with a ten-point PCV change extrapolating beyond observed support (Figure 7).

Load dependence is not informative. Across the ten pooled contexts with a verified schedule load, the ratio per five additional antigens is 0.999 (0.507-1.965) and per five injections 0.982 (0.516-1.865), but the posterior barely moves from the prior, so the data neither support nor exclude load dependence of the size the prior allows; the same holds for age at first dose (Supplement S64.2). Contexts of the same product agree, and U.S. PCV is consistent with the other PCV contexts (conflict $p = 0.108$; Supplement S64.3). The cumulative schedule-load

exercise lacks an identified estimand; absence of an estimable load coefficient is not evidence that its effect is zero.

Saturation tests a different prediction. If the schedule or measured uptake were the principal engine of autism growth, identification growth should slow once major additions and uptake stabilize. Instead, age-specific IDEA, NHIS and NSCH measures continue rising by roughly five to fifteen percent annually across their stable windows (Figure 8). Under an assumed individual RR of 2, observed uptake drift could generate only about 0.9 percent annual aggregate growth; even RR 3 yields about 1.3 percent. The comparison does not identify which diagnostic, social or environmental force drives the residual trend. Under these assumed risk ratios and measured uptake paths, uptake drift is insufficient to explain the observed growth. This comparison concerns changes over time; it does not bound a constant contribution to the level of recorded autism.

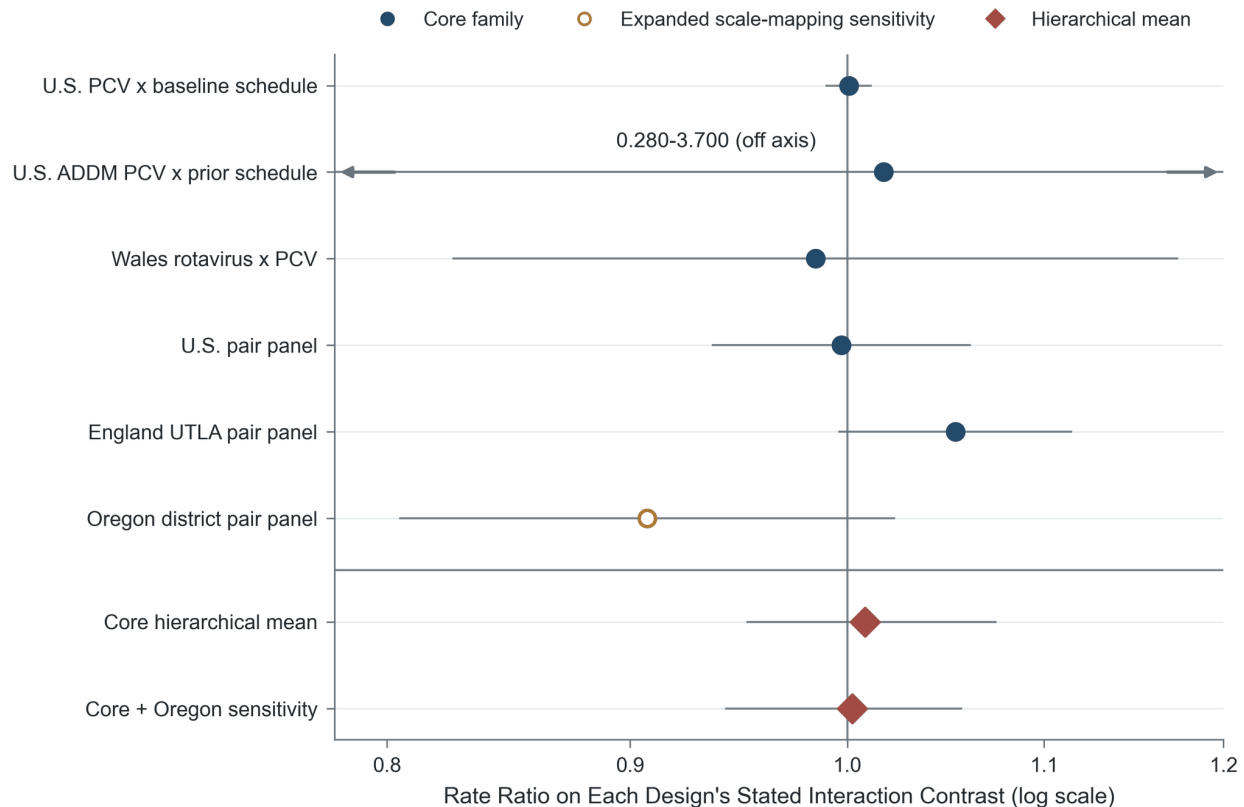


Figure 7. Exploratory schedule-addition and interaction estimates. The figure reports every core interaction-family summary on its stated joint contrast (the ADDM analogue is re-expressed on the U.S. (+10-point) by (+10-point) contrast, and its interval, 0.280-3.700, runs beyond the axis), the Oregon scale-mapping sensitivity, and the hierarchical summaries. These ecological diagnostics do not identify same-child biological interactions. The supplement reports the exploratory and failed-diagnostic interaction tests.

4.7 Convergence across designs, and the PCV7 rollout coefficient

Agreement among designs using the same cells assesses sensitivity to method choices, while disagreement can expose a specification problem. It does not provide independent data replication, and none of the synthetic-control rows enters the pool (Figure 4; Supplement S76.5).

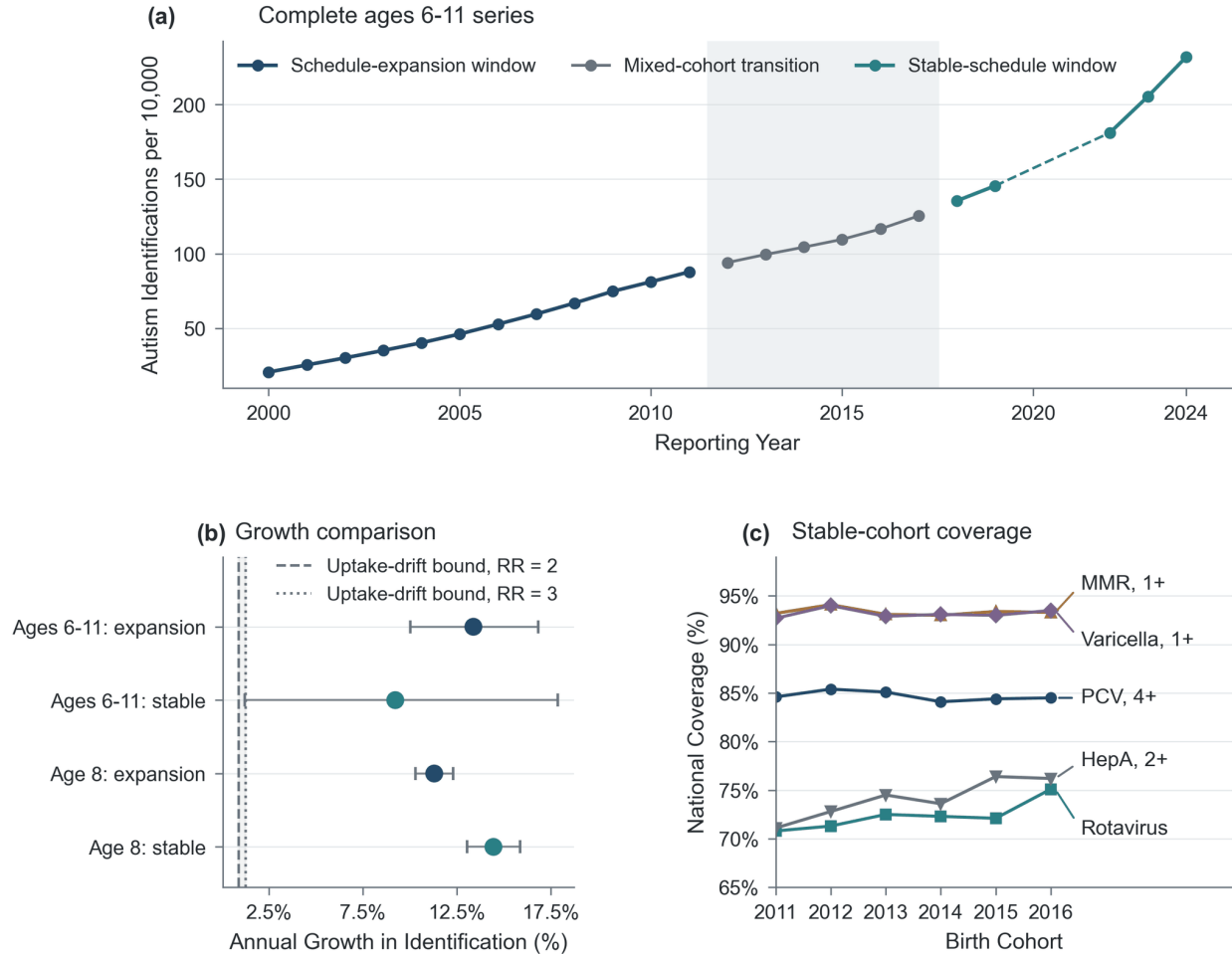


Figure 8. Autism identification keeps rising after schedule additions cease. Panel (a) uses every observable year in the ages-six-to-eleven IDEA series; the dashed connector marks unavailable state-level cells in 2020-2021. Panel (b) compares expansion- and stable-window growth in the pooled-age and exact-age-eight panels with model-implied growth from measured uptake drift at assumed individual RRs of two and three. Panel (c) shows the five national coverage trajectories underlying those bounds.

Across the estimator battery, estimates do not show a consistent positive pattern. Across six panels with sufficient variation, thirteen estimator families produce fifty-five identified estimator-panel cells; two isolated cells are nominally positive, neither replicates within its panel's battery, and none survives Holm correction. Eight exemption-event synthetic-control diagnostics have weak or wrong-signed first stages, broad outcomes and no treated-unit placebo rejection; they receive zero causal weight. Several national designs, including England, Spain, Wales, Northern Ireland and Sweden, are downgraded because cohort curvature, future exposure or alternative cutoffs reproduce their apparent effects. Figure 9 shows all six compatible event-study families. Figure 10 aligns 185 series-by-introduction instances and shows no

positive post-introduction bump; because it mixes products, repeats cohorts and has a nonzero lead, it receives zero causal weight.

The U.S. PCV7 rollout coefficient measures a native-scale contrast whose exposure mapping failed. The PCV7 rollout spline gives RR 1.013 per intensity standard deviation (Table 3). This is a state-ranking contrast, not a vaccination effect, and its 1999 reference cohort was already partly exposed: with the spline controls, the first stage on any-dose coverage is -1.37 percentage points per intensity standard deviation under the survey-year proxy and -6.69 under the birth-window remap. Noise-free, coverage-proportional harm injections return a negative spline coefficient in nine of twelve scenarios with hypothetical individual RRs of two, three, five and ten (Figure 3). Its narrow interval is therefore not a small individual-risk exclusion bound, and the PCV7 exposure audit limits the proposed small-effect exclusions. An event study referenced to 1997 gives an average 2000-2003 contrast of RR 1.025 (0.982-1.070; state-cluster CR1 with 50 degrees of freedom), still on the ranking scale. Joint NIS redraws give an upper bound near 1.046, and modeling insurance mandates and HCBS waivers leaves estimates between 1.009 and 1.019.

Obesity supplies the falsification. Treating youth obesity as an outcome PCV7 does not affect, the same index and design (cohorts 1997-2003, reference 1999) return post-2000 steps of 1.016-1.037 with the linear intensity trend and 1.024-1.048 with the spline, against autism steps of 1.016 and 1.013. A two-lead pretest rejects for obesity ($p = 0.003$ -0.027) but not for autism ($p = 0.636$), and it has only 8.5-12.7 percent power against obesity-sized linear drifts. Non-linear state-cohort drift of a few percent, the size of the 1.046 upper bound, cannot be excluded for autism (Supplement S48).

Fitting the full cohort exposure path moves the estimate with the trend model. In 353 state-cohort cells, a direct mixture model using remapped pneumococcal receipt gives conditional R estimates of 1.446 (nominal 95% score set 0.976-2.088) with state and cohort effects, 1.727 (1.084-2.642) with the uptake spline, 0.976 (0.543-1.462) with state-specific linear trends, and 1.008 (0.485-1.583) with both. A signed rollout-score inversion that does not divide by the weak first stage is wider: with the same remapped exposure and spline, the nominal 95% set is 0.017-1.367 under zero unobserved 1997 catch-up, 0.078-1.642 under half the state's 1999 receipt, and 0.258-2.275 under all of that receipt (Supplement S53). The model-native 80% threshold near 1.047 per rollout-ranking standard deviation is not an individual-risk threshold, and RotaShield's single-test 80% threshold of about 1.079 per ten uptake points and its familywise exposure-uncertainty threshold of 1.096 remain on their stated aggregate scale. The overlap-safe PCV7 kink gives 0.983 (0.928-1.043); Japan carries 53.9 percent of its weight, and without Japan it is 0.962 (0.857-1.081), whose upper limit exceeds 1.10 in nine of thirty alternative fixed-age choices. Table 3 lists these native-scale contrasts with their identification grades; none is converted to the individual scale.

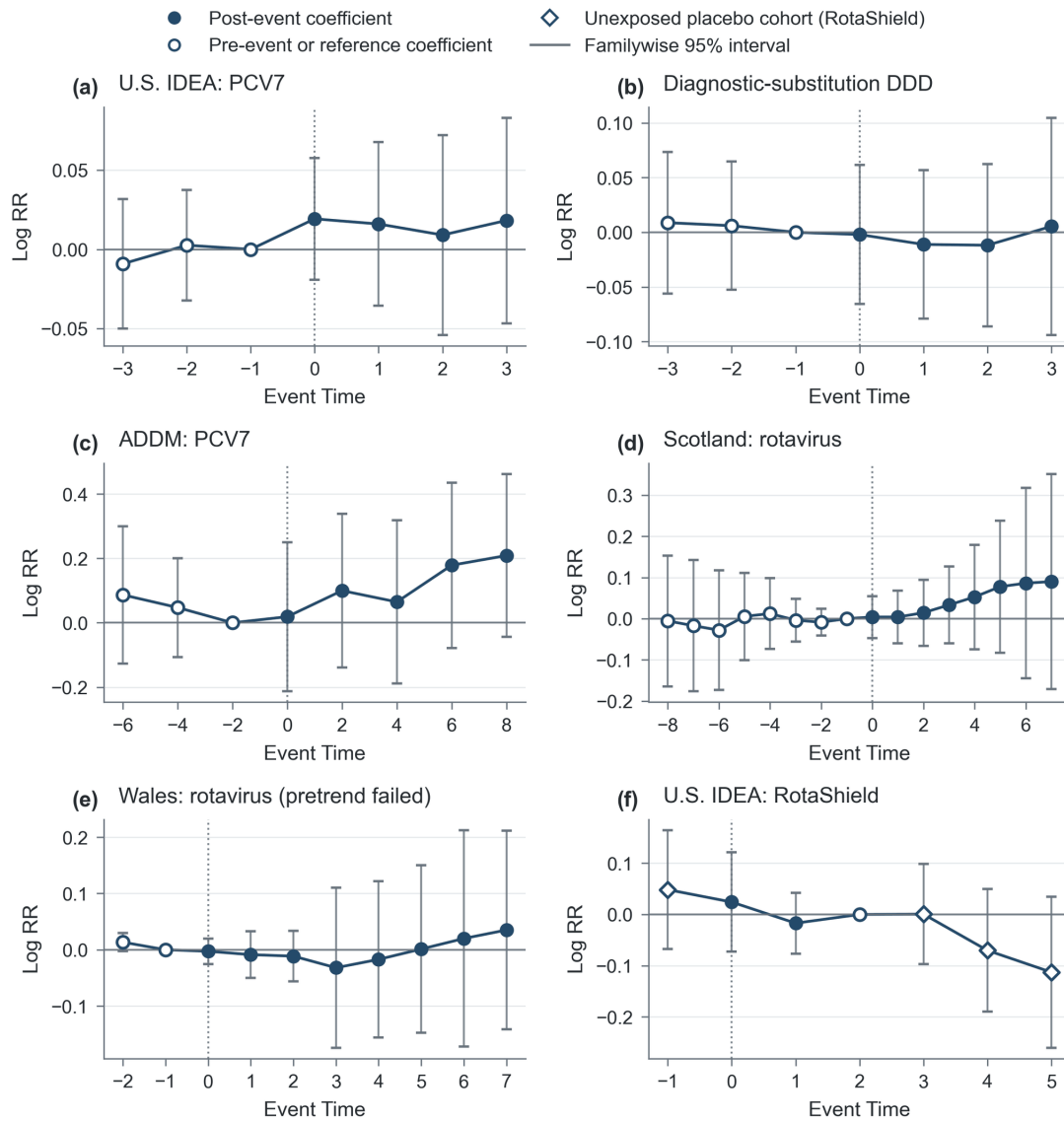


Figure 9. Event studies do not show a coherent post-introduction rise in autism. Every coefficient from all six compatible families is shown on its own intensity unit (ADDM: one six-site SD, 2.35 points; IDEA PCV7: one state SD, 8.91 points); open circles denote pre-event or reference coefficients, open diamonds in panel (f) denote the unexposed placebo cohorts of the RotaShield family (event times -1, 3, 4 and 5), filled markers denote post-event coefficients, and intervals are familywise 95 percent bands. All panels use one colour; panel (e) is labelled because its pretrend test failed. Wide bands and failed pretrends remain visible rather than being interpreted as favorable nulls.

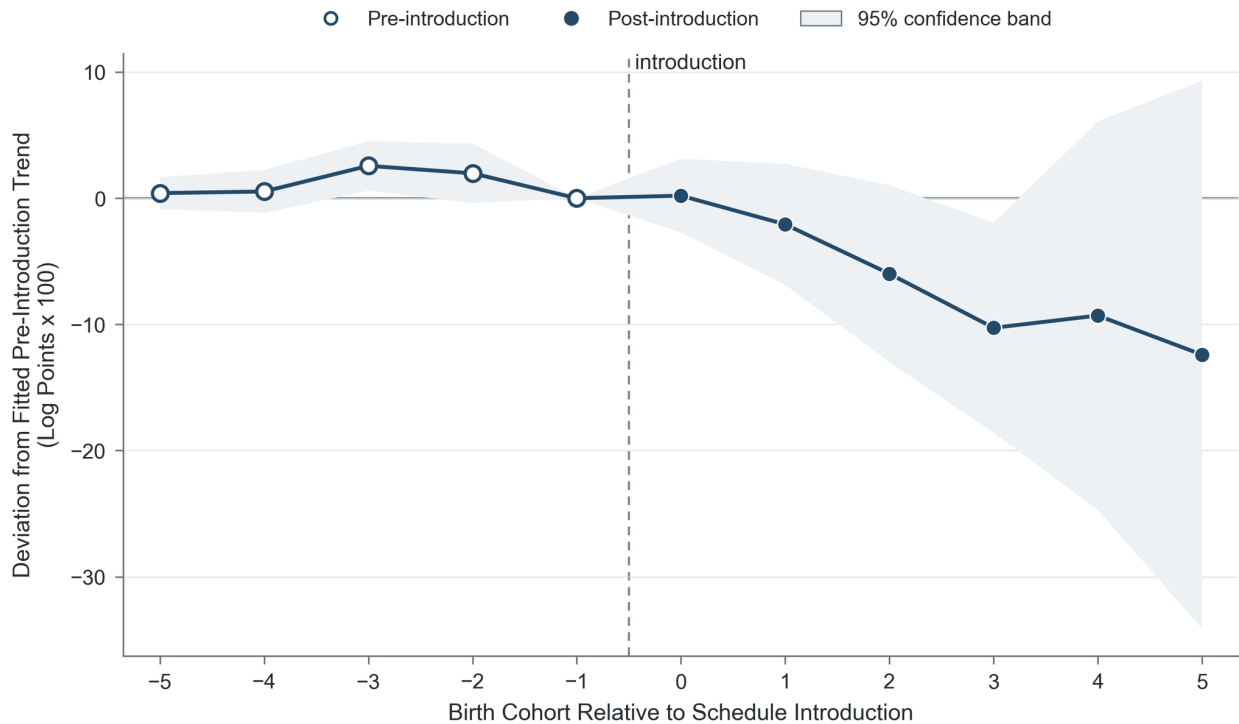


Figure 10. Across 185 series-event instances (138 series), the display does not show a clear positive post-introduction pattern. The display covers 138 cohort series, 47 jurisdictions, and 55 jurisdiction-by-event clusters; the shaded band is the 95 percent confidence band. Heterogeneous products, repeated cohort observations, and a nonzero lead make this a broad diagnostic rather than a causal pooled estimate.

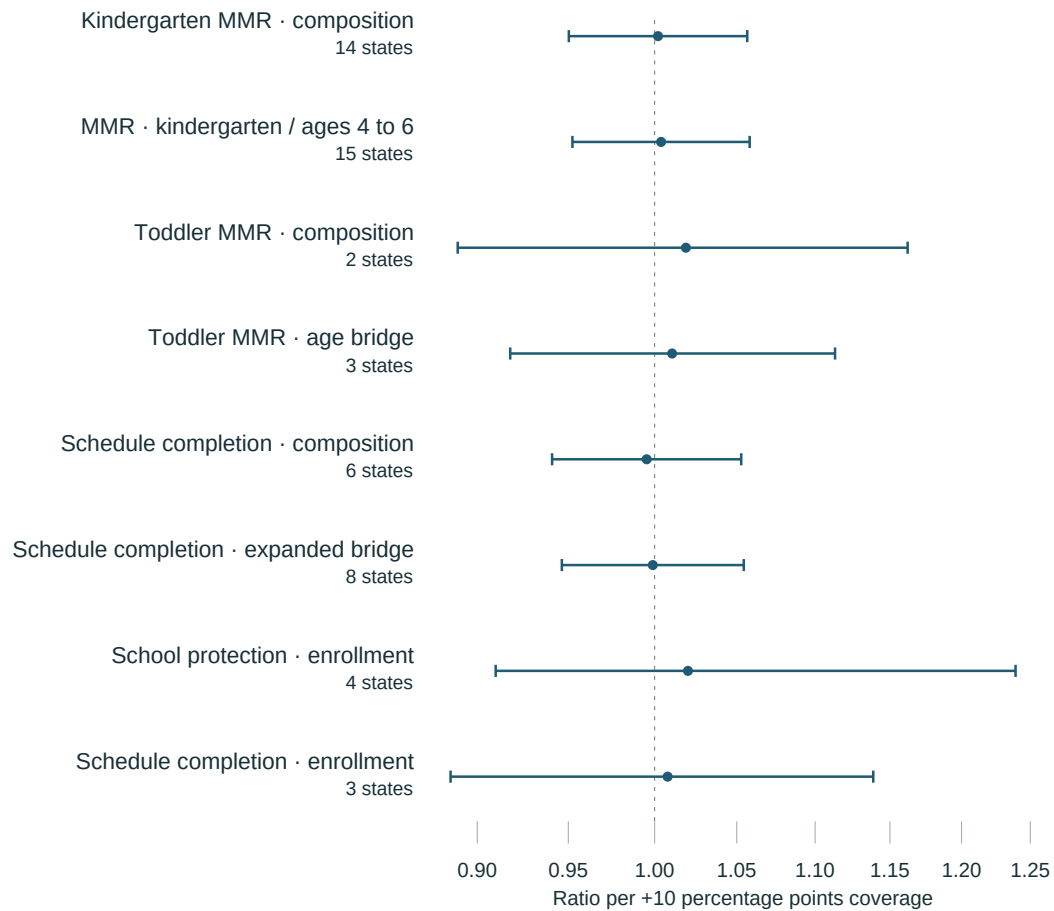
4.8 Local MMR gradients and other outcome systems

Local coverage gradients require separate denominators and exposures. Across fourteen states, the same-year association of kindergarten MMR coverage with autism's share of special education is 1.002 (95 percent credible interval 0.950-1.057) per ten coverage percentage points under the reference bias assumptions. It is not autism prevalence among all children and is not an introduction discontinuity. The complete local layer contains 22 designs in 21 states and separates kindergarten MMR, toddler MMR, school protection proxies and complete-schedule compliance. It retains failed as well as unavailable timing diagnostics, and uses one California source at a time. Coverage gradients with different exposure and outcome denominators cannot be combined into a common MMR population-risk bound; the family definitions and alternative summaries are in Supplement S67 and EA2. The source-matched early-childhood composition analysis combines six states and gives 1.002 (modified Hartung-Knapp nominal 95 percent interval 0.761-1.321, assuming independent member errors) per ten percentage points of contextual kindergarten MMR coverage. This distinct contrast concerns autism's share of disability classifications among children aged three to five in paired reporting districts; it is not the fourteen-state same-year association or autism incidence. Shared reporting shocks and leave-one-state-out sensitivities accompany it. Separate outcomes, denominator identities and dependence sensitivities are reported in Supplement S67.

Table 3. Principal estimates and identification assessment

Setting	Effect unit	Ratio	95% interval	Interpretation
Japan: MMR coverage	RR per +10pp coverage	0.981	0.805-1.196	Nine aggregate cohorts
Spain: varicella convergence	late-vs-early post RR	0.573	0.296-1.112	Failed differential pretrend
U.S. RotaShield pulse	RR per +10pp uptake	0.996	0.945-1.051	One clean lead
U.S. PCV7 rollout	RR per 1-SD early uptake	1.013	0.981-1.046	Ranking contrast; exposure mapping unresolved
U.S. PCV7 diagnostic substitution DDD	autism-specific differential RR	0.995	0.952-1.040	Autism-specific falsification contrast
U.S. SchoolVax MMR dose-response	RR per +10pp kindergarten MMR coverage	0.989	0.930-1.052	Three pandemic-adjacent cohorts
U.S. ADDM PCV7 replication	RR per 1 six-site SD (2.35 pp) early uptake	1.053	0.971-1.143	Six sites; small-cluster inference
England: rotavirus, pooled ages	eligible-vs-pre RR	0.940	0.891-0.993	Age-period-cohort and trend sensitivity
Scotland: rotavirus ramp	full-share RR per 1-SD uptake	1.020	0.567-1.833	Proxy intensity; weak precision
Wales: rotavirus ramp	full-share RR per 1-SD uptake	0.875	0.489-1.566	Failed pretrend and system break
Northern Ireland: rotavirus	fully exposed-vs-pre RR	1.078	0.915-1.271	Failed age-period-cohort/pretrend checks
Sweden: PCV rollout	fully exposed-vs-pre RR	0.886	0.708-1.109	Failed pretrend; outcome mismatch
U.S. kindergarten MMR; 14 states	Autism share of special education / +10 pp	1.002	0.950-1.057	Composition association; not population prevalence
Brazil municipality cohorts	RR / +10 pp	1.000	0.992-1.008	Exploratory ecological cohort approximation
Peru CONADIS autism	RR / +10 pp	1.759	1.093-2.829	Excluded; future exposure and specificity fail
Overlap-safe PCV7 introduction-kink synthesis	RR / cohort-slope kink	0.983	0.928-1.043	Japan dominates weight; alternative ages are less precise
U.S. PCV7 state-specific kink replication	RR / cohort-slope kink	0.991	0.973-1.009	Same underlying federal data; finer geography

Notes: Each row uses the exposure contrast in column 2; pp denotes percentage points and SD denotes a standard deviation. Ratios concern different outcomes and must not be pooled. Intervals retain their source-specific definitions; the 14-state MMR association has a credible interval. Failed identification diagnostics limit interpretation in either direction. Full p-values and pretrend tests are in Supplement EA4.

Local coverage gradients**Distinct exposures and outcome denominators**

95% credible intervals with assumed bias allowances. Age/provenance bridges are sensitivities.
 Composition is autism's share of special education, not population risk.
 Rows overlap and are not pooled with one another.

Figure 11. Local coverage gradients separated by exposure and outcome denominator. Eight overlapping family rows span kindergarten-MMR, toddler-MMR, schedule-completion and school-protection measures with composition, age-bridge and enrollment denominators; their intervals are 95 percent credible intervals under the reference bias assumptions. A special-education composition ratio is not a population autism risk ratio; none of these associations has a validated individual-risk conversion.

Other outcome systems check measurement, not confounding. Eight staggered Canadian provincial rotavirus introductions aligned to national surveillance give positive naive age-six event studies, but their joint lead tests fail overwhelmingly and therefore receive no causal interpretation; with province-specific trends and partial exposure by age, incidence is RR 0.999 per ten percentage points of cohort saturation (0.937-1.065) and prevalence 1.008 (0.996-1.020). ADDM covers six sites whose exposure standard deviation is 2.35 points, against 8.91 in the IDEA state sample; per ten points its PCV7 estimate is 1.247 (0.881-1.766), extrapolated beyond the sites' 7.2-point range, with nominal power of about 8.9 percent against 1.10. NHIS vaccination-history analyses are exploratory, with every autism estimate crossing one and Holm-adjusted p values near one. The most-cited NHIS association between the hepatitis B birth

dose and autism (Gallagher and Goodman 2010) was retracted in 2026 (Journal of Toxicology and Environmental Health, Part A 2026). Figure 12 reports the twenty-three estimates in the international summary.

Two international associations remain positive and unresolved. Brazilian hospitalization models comparing births from 2007 onward with births through 2004 give autism-coded authorization ratios of 0.992 to 2.063 across specifications. They persist when registered births replace authorizations as the denominator, but unrestricted cohort effects absorb the exposure indicator; conversely, the negative cerebral-palsy associations do not establish protection. Australian disability-service composition gives a weighted ratio of 1.092 (0.893-1.306) and unweighted one-year-lag ratios of 1.134-1.162 that do not survive adjustment across 72 fits. These analyses receive no independent likelihood, and exclusion from pooling does not turn their unresolved signals into evidence of no effect (Sections S41.5 and S45.4).

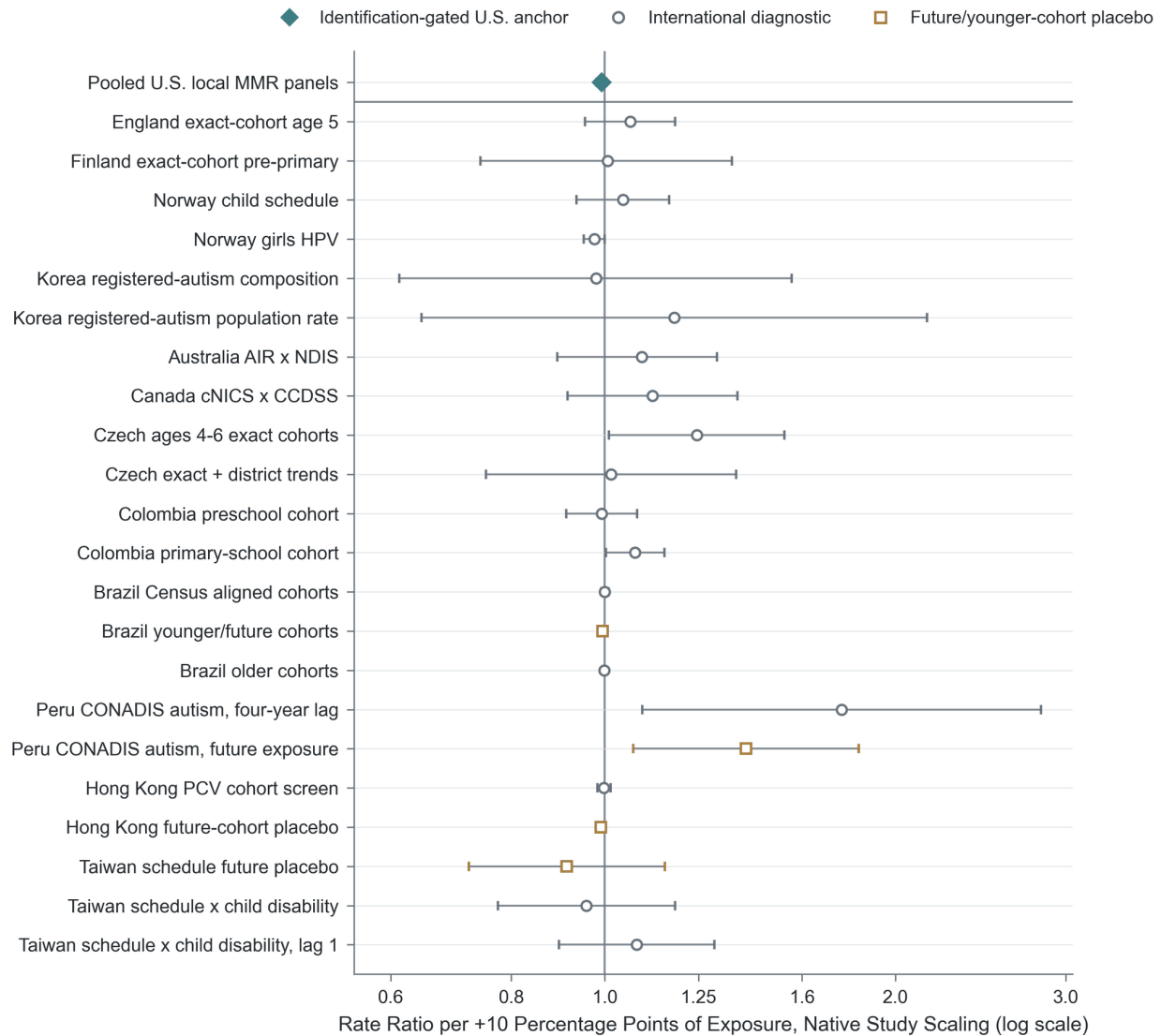


Figure 12. Twenty-three international public-data estimates. No row passes the timing, trend, outcome, and counterfactual gates for a harmful causal interpretation. The display includes positive, inverse, future-exposure, and younger-cohort estimates; outcomes on unlike scales are reported separately rather than pooled. Every row is a rate ratio per +10 percentage points of the study's exposure on its native scaling (log axis).

4.9 Schedule changes and recorded autism

Across 39,753 overlapping comparisons around 404 changes in 40 countries, the uptake-scaled model estimate is 0.973 (95 percent random-effects interval 0.836-1.131; Figure 13A). The pooled estimate is close to no change. With a shared-bias standard deviation of 0.15, its interval is 0.699-1.354 and includes a 25 percent increase. The placebo-calibrated sensitivity gives 0.855-1.107, using a country component of 0.26 and a shared component estimated at zero. That calibration is fitted to the same placebo data and has not established out-of-sample coverage. Table 5 compares the uncertainty assumptions on the same pooled estimate.

Pooling within countries limits the influence of repeated analyses of the same records. Brazil supplies 83 percent of comparisons but 5.4 percent of the random-effects weight; the effective

number of countries is 26.0. Alternative information weights give 0.967 with a wider placebo-calibrated interval of 0.739-1.265. The unscaled population step is +1.7 percent (-7.0 to +11.3); its sign can differ from the uptake-scaled result because exposure changes and weights vary. Full weighting results are in Supplement EA5.

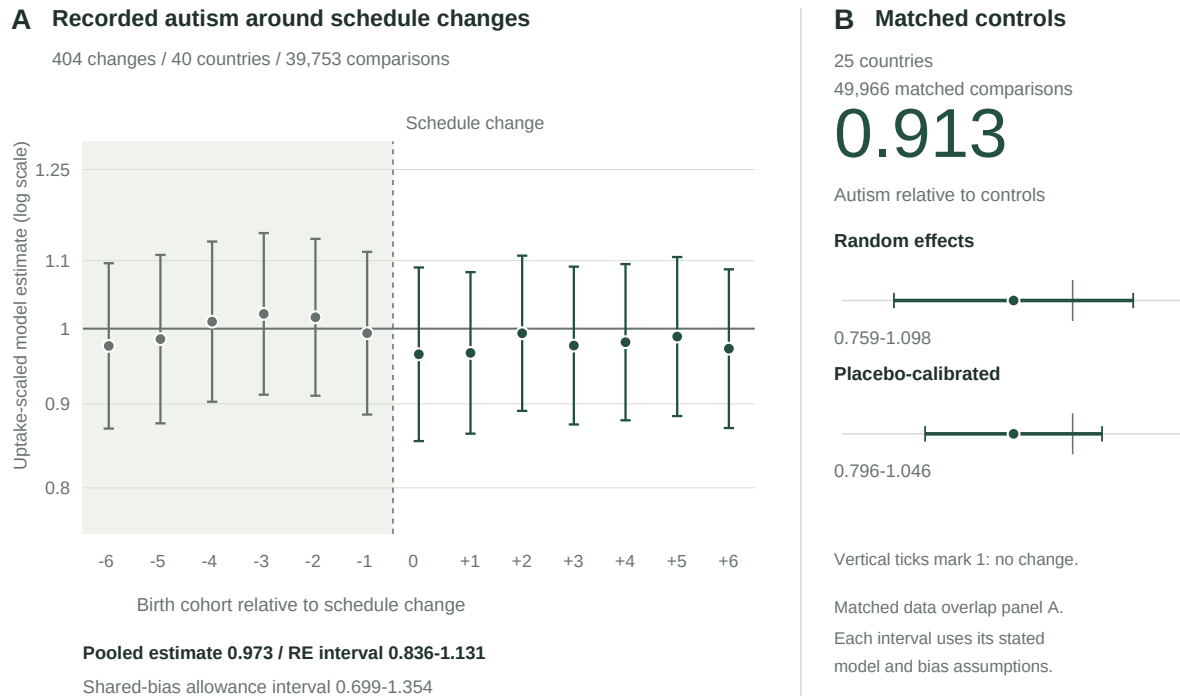


Figure 13. Recorded autism around vaccine schedule changes and the matched-control comparison. (A) Uptake-scaled estimates by birth cohort relative to each change; whiskers show 95 percent random-effects intervals. Country composition varies across cohorts. The separate pooled estimate is 0.973 (0.836-1.131), with a shared-bias allowance interval of 0.699-1.354. (B) Pooled autism-versus-control contrast on 49,966 matched comparisons in 25 countries, with random-effects and placebo-calibrated intervals shown separately. The matched sample overlaps the headline data. Both panels show conditional working-model summaries, not directly observed individual causal risk ratios. The full recovery and matched event-time displays are in Supplement EA1 and EA3.

Specified deterministic perturbations with target ratios of 1.25 and 1.50 return 1.23 and 1.46. Recovery is poorer in some source strata. All six pooled fake-date intervals include zero, while sampling-only country intervals include zero for 80 percent of 187 placebo estimates. These checks support particular features of the estimator but do not validate receipt measurement or general interval coverage (Supplement S78, EA1 and EA4).

The matched autism-versus-control contrast is 0.913 (random-effects interval 0.759-1.098; placebo-calibrated interval 0.796-1.046; Figure 13B), using 49,966 comparisons in 25 countries. It subtracts the control-outcome step from the autism step on the log scale. The data overlap the primary analysis, and matching can remove only biases shared by autism and the selected controls. Separate-outcome and control-set sensitivities are in Supplement EA3 and EA5.

Point estimates remain close to one across alternative trend shapes, windows and uptake-path strata (Table 4). All 40 single-country and 780 country-pair deletions retain random-effects

intervals that include one. These results show stability of the pooled working-model summary across the specified alternatives; they do not resolve shared bias or validate the individual-risk conversion.

Table 4. Sensitivity of the schedule-change estimate to model and sample choices

Specification	Ratio	95% random-effects interval	Countries	Comparisons
Linear trend: primary model	0.973	0.836-1.131	40	39,753
Quadratic trend	0.960	0.828-1.112	39	34,775
Spline trend	0.980	0.848-1.133	37	28,296
End follow-up four cohorts after the change	0.963	0.829-1.118	39	36,108
Measured vaccination uptake	0.934	0.717-1.217	14	8,355
Modeled vaccination uptake	0.967	0.807-1.159	37	15,055
Mixed measured and modeled uptake	0.963	0.779-1.190	16	16,343
Large increases in uptake	0.984	0.843-1.148	38	20,514
Countries with subnational data	0.993	0.818-1.205	18	39,532
Exclude vaccine mandates	0.970	0.834-1.129	40	37,386

Notes: Each specification uses its own retained observations. Countries and records recur across rows, including uptake-path strata. Intervals shown here do not add the shared log-bias SD of 0.15. Across all 40 single-country deletions, point estimates range from 0.965 to 0.985; across all 780 country-pair deletions they range from 0.957 to 0.993. Every deletion interval includes one. These checks assess model stability, not causal identification. Full recovery, placebo and dependence results are in Supplement EA4.

An additional cohort-slope model estimates -1.73 percent per cohort (placebo-calibrated interval -8.05 to +5.02) on 35,365 comparisons. Its specified trajectory allows a change across successive birth cohorts, including a 25 percent rise by the sixth cohort. This is not a prediction of within-person accumulation following an abrupt uptake change. Four of six ramp placebo random-effects intervals exclude zero from below, limiting interpretation of this sensitivity analysis (Supplement EA4).

Omitting Brazil outpatient records, excluding mandates and changing selected exposure or case definitions leaves point estimates close to one (Table 4; Supplement Figure S117). School-entry-only exemption laws fail the recovery check and do not validate the uptake-scaled design. Detailed source and eligibility sensitivities are in Supplement S74 and EA5.

The receipt-conversion battery passes six of seven specified criteria but fails the zero-intercept dose-response check. Effect recovery also attenuates more strongly with measured uptake than with modeled uptake. The conversion to an individual risk ratio is therefore conditional; the failed check and its implications are discussed in Section 8, with full results in Supplement S78 and EA4.

5. Could the analysis have missed a harmful effect?

5.1 Every harm-direction signal and its diagnostics

The harm-direction register lists every stored jump-program estimate whose interval lies entirely above the null, and every posterior (hierarchy context, U.S.-alone diagnostic or unpooled block) whose lower 95 percent limit exceeds 0.95 or whose probability of exceeding one is at least 0.9, with its placebo, pretrend, multiplicity and first-stage diagnostics. It holds 154 rows: 89 jump-program signals, one hierarchy context, seven U.S.-alone diagnostics, nine unpooled block posteriors, 39 project estimates for chronic and disability outcomes and nine published chronic estimates. The jump-program signals come in three tiers: 3 in a row's own headline interval, 55 in another calibrated or otherwise final specification or variant, and 31 in an uncalibrated interval where no calibrated one exists. The chronic and disability rows appear with their diagnostics in Section 6 and Supplement S48.1.

Tier 1. The influenza-by-Hib-deferral interaction is 1.373 (1.085-1.737), or 1.50 (1.11-2.03) after errors-in-variables correction. It does not survive multiplicity across 38 primary rows (Benjamini-Hochberg $q = 0.354$); its excess sits in a "protective" Hib-booster main effect among children without influenza vaccine (0.76, 0.63-0.92); it coincides with the 2009 H1N1 season and the Hib shortage; and its time-shift placebos do not reproduce it. The other two tier-1 signals are Swedish synthetic controls the design audit classified as invalid: a municipal rotavirus design (4.70, 1.13-9.03) lies outside its donor hull and shows its gap before exposure, and an early-county PCV gap (log gap 0.73, 0.59-0.87) exceeds the ceiling attainable for any r and reappears under equal exposure.

Tiers 2 and 3 are listed one by one, with their diagnostics, in Supplement S60.5 (Table S60-B): non-headline specifications of England's hexavalent hepatitis B step and of U.S. influenza vaccination, U.S. targeted hepatitis A, Canadian and Swedish synthetic controls, uncalibrated ADDM fits, a sensitivity-tier interaction with imputed joint receipt, and single rows whose own placebos or headlines undercut them.

The unpooled block posteriors that qualify are listed with their diagnostics in Supplement S76.9.

The U.S. PCV context is the one pooled context that meets the register's rule. Its other U.S.-alone specifications run from 1.141 to 1.212, with the 1997-bound exposure giving 1.212 (1.004-1.468), and the stated infant-exposure sensitivity gives 1.456 (1.169-1.863). Its future-exposure placebo includes one under all four kernels (reference 0.99, 0.68-1.56). Against that, its negative controls lean at least as far or show bias directly (Section 4.4), direct-mixture estimates run from 0.976 to 1.727 across trend specifications, and leaving U.S. PCV out moves the shared-mean upper limit by only -0.006. The lean is unresolved: the data establish neither that it is drift nor that it is specific to autism.

Every signal outside Section 6 therefore falls into one of five classes: a non-headline or uncalibrated specification of a jump whose checked interval includes one; a synthetic control

discarded as invalid or kept for convergence only; a pair that fails multiplicity and coincides with a calendar confound; an estimate whose own placebo or negative controls return the same kind of "effect"; or an unpooled posterior whose interval includes one. The U.S. PCV lean belongs to the fourth class but, as above, remains unresolved.

5.2 Claimed conditions, mechanisms and untested theories

Other conditions are not automatically negative controls (Supplement S76.13).

The substantive register covers twenty-two theories. The policy analyses do not establish the tested generalized patterns: there is no multiplicity-stable schedule synergy, no monotone cross-product administration-age gradient and no saturation of autism growth with schedule stabilization; the load meta-regression is uninformative rather than negative. The Brazil maternal-Tdap screen gives a weighted total-autism RR of 1.000 per ten points (0.988-1.012). Several theories remain outside direct resolution: microbiome mediation beyond persistent measles, maternal influenza and trimester-specific immune activation, epigenetic or transgenerational effects, rare lot or contaminant defects, exact simultaneous-versus-separated visits, acute regression windows, mortality masking, and biomarker-defined susceptibility. These panels do not measure those proposed mechanisms directly. This scope limit is not positive evidence for them or a claim that the wider literature has not examined them.

6. Chronic disease and disability outcomes

6.1 The evidence standard

The project admits a chronic-outcome comparison to its causal synthesis only under its stated randomization or quasi-experimental design, measurement and diagnostic criteria. These are admission rules, not a guarantee that a passing result is causal. Power limits the strength of exclusions; low power alone does not invalidate an otherwise identified estimate. No project comparison below is admitted under those rules, so its assigned causal-synthesis weight remains zero; ecological ratios retain their own exposure scales.

No design here estimates the contrast behind headline chronic-harm claims; utilization contrasts that claim it receive zero weight (Supplement S76.14).

6.2 Type 1 diabetes

Three comparisons that allocated vaccination by randomization or birth date, the Finnish Hib and rotavirus trials and the Northern California HbOC trial, support neither a precision nor an exclusion claim (Supplement S76.2).

In the Norwegian rotavirus natural experiment (740,744 children), full vaccination rose from 0.3 percent before the September 2014 cutoff to 92.8 percent after it. It estimated a harmful-direction change in the birth-cohort trend of type 1 diabetes, relative HR 1.11 (1.03-1.20), or 1.05 (0.93-1.18) excluding the 2013-2015 cohorts (Östman et al. 2026b). This is

a slope change, not a level risk ratio, and it is not rotavirus-specific: post hoc cutoffs at the hepatitis B introduction and the PCV7-to-PCV13 switch give breaks of nearly the same size, 1.10 (0.96-1.28) and 1.10 (0.97-1.24), whose intervals include one, and no pretrend test was reported. Its observational HR for fully vaccinated versus unvaccinated children is 1.17 (0.78-1.77). The same cohort shows no celiac trend break (Östman et al. 2026a; Supplement S49.1). The design receives zero causal weight.

The project's own diabetes analyses (EURODIAB introduction dates, age gradients, Swedish annual contrasts and the Finnish decline) are weak or undercut by their own pretrend and placebo checks; they are set out in Supplement S76.2.

6.3 Obesity, asthma, epilepsy and aluminum

Youth obesity modeled from YRBS fails every pretrend and placebo test, so its associations do not identify a causal vaccine effect; the RotaShield, asthma and WIC rows are in Supplement S76.3.

Andersson et al. (2025), which related cumulative vaccine aluminum by age two to 50 outcomes in Danish children, its correction, and the Vaccine Safety Datalink asthma cohort (Daley et al. 2023) are reviewed in Supplement S76.3.

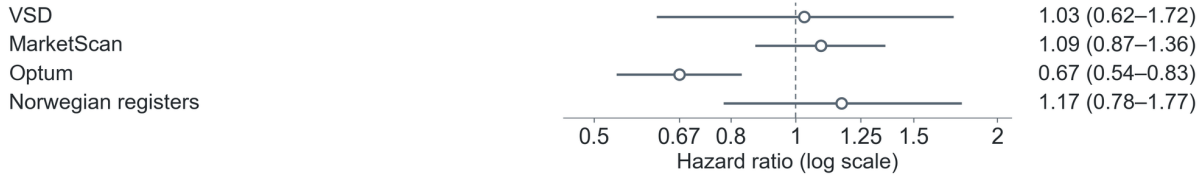
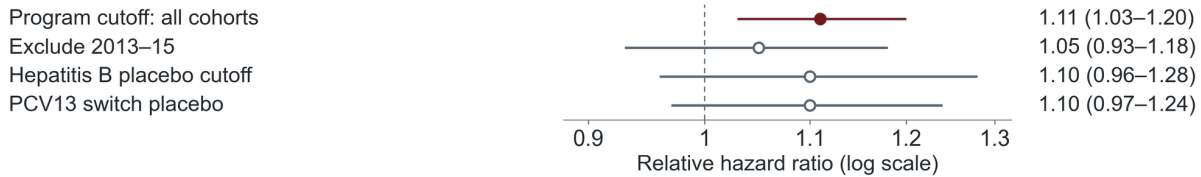
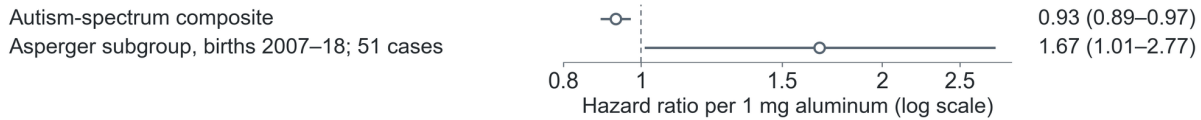
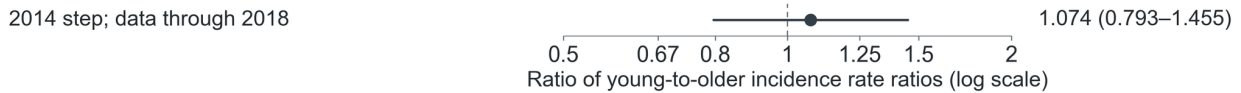
The Danish categorical comparisons also depend on the estimand and estimator. For autism spectrum disorder, the middle-aluminum group (>1.5-3 mg) versus the high group (>3-4.5 mg) has a Cox HR of 0.97 (0.90-1.05), whereas its inverse-probability-weighted cumulative-incidence difference at age five is -8.68 (-12.38 to -4.98) cases per 10,000. Reversing the latter comparison gives 8.68 additional cases (4.98-12.38) per 10,000 in the high group. This positive association is retained, not called a null. The lower-dose group (>0-1.5 mg) versus the high group instead has HR 1.29 (1.11-1.50) and risk difference 13.09 (-2.20 to 28.39) per 10,000. These are different adjusted targets, not interchangeable measures or a coherent monotone dose response. Supplement S72 preserves every effect cell in the corrected supplementary figures. The observational contrasts, correlated multiple tests and unavailable individual records do not establish which pattern is causal, and neither their positive nor inverse values are discarded.

6.4 Disability and psychiatric panels

Disability and psychiatric panels from Hong Kong, the United States, Peru, Brazil, New Jersey, Norway and France give scattered associations, a few surviving false-discovery-rate control, but none has a validated policy first stage and many of their placebos also fire; none carries causal weight, and Supplement S76.4 lists them with their diagnostics.

6.5 Childhood cancer

Childhood cancer was added as an outcome family, with an internal rule file documenting its analysis, because a broad injury theory predicts it and registries measure it independently. A positive control was recovered first: the expected HPV-programme reduction in cervical carcinoma in situ in Czechia, 0.42 (95 percent credible interval 0.18-0.83), and in two Japanese registries; other positive-control fits did not recover. Across 13 gated contexts, all malignant neoplasms give 1.00 (0.78-1.28), with posterior probability 0.040 above 1.25; leukaemia gives 1.07 (0.79-1.43), and lymphoid leukaemia leans above one, 1.16 (0.83-1.62). The designs are weak: with no effect, whole-pipeline simulations exclude 1.25 for all malignant neoplasms with probability 0.43. The evidence is compatible with no effect and does not exclude moderate effects on leukaemia (Supplement S75). These individual-scale rate ratios (with 95 percent credible intervals) are model-implied from population data and, under the standard of Section 6.1, carry no causal-synthesis weight.

Rotavirus series vs no vaccination | Type 1 diabetes*Hazard ratio; observational cohorts***Norway rotavirus program and placebo cutoffs | Type 1 diabetes***Relative hazard ratio for cohort-trend change***Danish aluminum exposure | neurodevelopment***Hazard ratio per 1 mg by age 2; outcome ages 2–5***Swedish rotavirus-era rollout | type 1 diabetes***Triple contrast; low calibrated power*

Selected signals only; panels use distinct native scales and are not pooled. Full 57-row detail: supplement. Open markers: published observational estimates (zero causal weight). Filled slate marker: Sweden, this project's estimate (low calibrated power). Red: Norway's program cutoff; placebo cutoffs at other vaccine changes give breaks of nearly the same size (1.10), so the break is not rotavirus-specific; no pretrend test was reported.

Figure 14. Selected chronic-outcome findings on their native scales, not a pooled estimate. They include the Norwegian type 1 diabetes trend ratio at the rotavirus cutoff, which placebo cutoffs at other vaccine changes match (Section 6.2), and the Danish Asperger hazard ratio from 51 cases. None is assigned causal weight. Open markers are published observational estimates, the filled slate marker is this project's Swedish estimate, and the red marker is Norway's program cutoff (all axes are log scales). All 57 rows, their units and diagnostics appear in Supplement Figures S69-S71.

6.6 What the chronic evidence supports

Conclusions are outcome-specific. Childhood cancer estimates are compatible with no effect but underpowered (Section 6.5). For type 1 diabetes, obesity and asthma, no project design passes the diagnostics required for causal weight, and the evidence identifies neither protection nor a precise null. The non-autism panels neither establish nor exclude a generalized chronic-injury pattern: positive associations survive multiplicity only where placebos, fake dates or negative controls also fire, signs vary across products and systems, and no panel has a validated first stage. Unexplained is not disproved. The French intellectual-disability rows, the Norwegian measles-symptom and musculoskeletal rows, the SchoolVaxView row and the aluminum-asthma association remain unresolved. Annual panels without onset dates are not designed to detect harms such as Pandemrix-associated narcolepsy in Finland, with a rate ratio of 12.7 (6.1-30.8), about one vaccine-attributable case per 16,000 vaccinated four- to nineteen-year-olds (Nohynek et al. 2012).

7. Interpretation of the evidence

The population analyses find no detectable abrupt increase in recorded autism at vaccine schedule changes. Their estimates are broadly stable across the specified model and sample choices, but exposure measurement and shared bias limit individual-risk interpretation. The matched-control analysis reaches a similar conclusion on overlapping data; it does not supply an independent replication. The additional cohort-slope analysis is a distinct sensitivity model, rather than a consequence of within-person accumulation.

Vaccines do not cause autism is the wider-evidence conclusion, not proof that public data identify every effect as zero. Cohorts and policy contrasts fail to reproduce a coherent timing, dose and outcome signature. Jump intervals include one, withdrawals and deferrals show no clear response, and identification keeps growing after uptake stabilizes. Unequal power and counterfactual credibility prevent universal context-specific exclusion.

The 23-context shared-mean model leaves a posterior probability of 9.8 percent above 1.25. Control calibration narrows restricted-model intervals only under comparable-bias assumptions; residual autism-specific bias widens them. Neither that calibration nor the pairwise interaction mean bounds total schedule exposure. The load contrast is largely prior-driven. Counts of stored estimates do not resolve dependence, bias or exposure mapping.

The public panels do not directly resolve every product, context, subgroup, spacing, lot or biomarker hypothesis (Section 5.2); being outside their scope is not evidence of harm. U.S. PCV's lean, 1.17 (0.97-1.39), is unresolved because candidate controls also lean harmful; moving it to 0.89 requires shared-drift assumptions. Chronic conclusions remain outcome-specific (Section 6).

The PCV7 harm injections reverse the spline's sign (Figure 3); narrow intervals and Bayes factors cannot repair that invalid individual-risk mapping. The same standard applies to positive

and negative coefficients: neither automatically identifies injury or protection. Valid translation requires the correct exposure contrast, baseline-risk and compliance assumptions; individual cohorts also remain observational.

Vaccine safety is outcome-specific. MMRV seizures, RotaShield intussusception, Pandemrix narcolepsy and positive acute PCV13/coadministration associations identify specific products, phenotypes and risk windows (Supplement S67). The autism contrasts do not reproduce that coherent signature.

8. Limits and next studies

The principal limits are exposure measurement, shared bias, event eligibility and the form of the effect being tested. A retained timing audit reports 42 contributing California configurations whose source outcomes precede the selected policy's recorded date. Their source and membership qualification remains unresolved for the displayed sample (Supplement EA4 and EA6). The uptake-scaled estimate depends on proportionality between uptake changes and outcome changes, but the zero-intercept dose-response check fails: its intercept is +0.180 (95 percent interval +0.096 to +0.264). Deterministic effect recovery also varies by source stratum. These failures limit interpretation as an individual causal risk ratio, even when a pooled estimate is precise. Detailed recovery results, placebo calibration, source-specific diagnostics and numerical verification are in Supplement S78 and EA4.

Under the specified model, independent country-specific heterogeneity can average down across countries; a shared bias does not. Dependence between country errors limits that averaging. The placebo-calibrated interval assumes a negligible shared component, whereas the assumed shared log-bias SD of 0.15 gives an interval of 0.699-1.354 and includes a 25 percent increase (Table 5). Placebo calibration and holdout checks reuse source records and cannot establish general coverage. Matching removes only biases shared by autism and the selected control outcomes; autism-specific bias can remain.

Table 5. Uptake-scaled model intervals under three shared-bias assumptions

Shared bias SD	Basis	Calibrated variance + shared variance	Random-effects variance + shared variance
0.00	Placebo-calibrated shared component	0.855-1.107	0.836-1.131
0.08	Upper end of the step-placebo shared-component interval	0.794-1.192	0.782-1.209
0.15	assumed shared-bias allowance	0.705-1.341	0.699-1.354

Notes: Same pooled model estimate, 0.973, from 39,753 comparisons in 40 countries. Intervals are $\exp(\log \text{ estimate } \pm 1.96 \times \sqrt{\text{variance} + \text{shared SD squared}})$; the calibrated country component is 0.26 and the specified component is 0.33. These are normal-reference working-model intervals, not demonstrated coverage guarantees. A shared component does not average down.

The primary cohort-step model corresponds to an abrupt uptake change under a constant-effect assumption at comparable follow-up ages. A slope across successive birth cohorts requires a separate exposure or effect-variation hypothesis; within-person accumulation alone does not

supply it. Calendar-year records that mix cohorts can smooth a cohort step as their composition changes. Before-introduction cohorts cannot automatically be treated as unvaccinated: some baseline uptake is modeled, and catch-up vaccination can change the contrast. Source-specific conversion checks are in Supplement S74 and EA5. Linked registers with exact receipt histories would improve this measurement.

Outcomes are institutional as well as biological. IDEA measures educational classification, ADDM measures surveillance prevalence, household surveys use parental report, and international systems often measure service use or disability composition. Fixed ages, flexible cohort paths, diagnosis-policy terms and cross-system comparisons address parts of this separation, but state funding rules, provider capacity, migration and waiver generosity remain incompletely observed.

Vaccination coverage is sampled or administratively reported, with incomplete information on brands, lots and correlated products. Multiple designs reuse the same surveys and registers; overlap adjustments prevent double counting but cannot repair exposure measurement. Data-informed selection remains a limitation. Colombia's estimate depends on pandemic school years, and negative controls reveal possible bias or lack precision in several settings. Pairwise interactions and local coverage gradients do not identify the contrast between the entire schedule and nonvaccination. The ecological PCV interaction is limited to the observed range of MMR and varicella coverage.

Linked mother-child and sibling registers with exact product, dose, visit, date and lot histories, validated onset and susceptibility markers would address unresolved age, prenatal, spacing and subgroup hypotheses. Public NHIS and NSCH cannot supply sibling fixed effects; held panels lack the subannual discontinuities needed for fuzzy regression discontinuity. New policy changes are useful when coverage and outcomes are published by birth cohort.

¹ Poul Thorsen, a co-author of Madsen et al. (2002, 2003) and Parner, Schendel, and Thorsen (2008), pleaded guilty on 1 September 2026 to wire fraud in the theft of CDC grant money (U.S. Attorney's Office, Northern District of Georgia 2026). None of the three papers carries a retraction, correction, or expression of concern (PubMed, Europe PMC, and Crossref, accessed 22 September 2026). The native HR family excludes Madsen's incidence-rate ratio by design and gives 0.954 (0.647-1.415); including it through an explicit IRR/HR bridge gives 0.947 (0.694-1.293). Neither comparison resolves shared observational bias.

Data, code, and research ethics

Data definitions, code, source inventories and saved results accompany the paper's research archive and Evidence Explorer. The supplement documents analysis specifications, data-informed choices, overlap, model diagnostics and release provenance. Reproduction from supplied cells and saved likelihoods does not independently reacquire every raw source, establish source accuracy or validate causal identification. Restricted materials require permitted access. This study used public aggregate data, public-use files and published estimates, without recruitment, intervention, participant contact or identifiable private information.

<https://vaccine-autism-evidence.ljlasker.chatgpt.site> (downloads)

AI use declaration

The author originated the ideas and research questions. Anthropic Claude and OpenAI Codex wrote and revised substantial portions of this paper. Generative AI systems, including Claude, Codex, OpenAI ChatGPT, xAI Grok, and Moonshot Kimi, also assisted with source discovery and triage, code drafting and debugging, data extraction, specification development, robustness review, visualization, and reproducibility checks. These systems were not treated as authors or as independent evidentiary sources. Their outputs were evaluated against archived data, executable code, validation ledgers, cited publications, and official records; suggestions that could not be substantiated were excluded or explicitly labeled. The author directed the research and inferential standards, made the final analytical and editorial decisions, and accepts responsibility for the accuracy, integrity, reproducibility, and conclusions of the work.

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