

You Can Only Die of One Thing: Competing Causes and the Economics of Recording Liver Mortality in Russia, 2012–2019

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Research Article

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Highlights

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- A death certificate holds one cause; chronic disease does not respect that
- Within regions, rising alcohol use predicts fewer registered hepatitis deaths
- Regions drinking persistently more record more: a competing cause of death
- Widening the liver envelope makes the effect vanish: only the label moved
- Ranking regions by hepatitis mortality ranks coding practice as much as disease

You Can Only Die of One Thing: Competing Causes and the Economics of Recording Liver Mortality in Russia, 2012–2019

Abstract

A death certificate holds one cause. A liver can fail from a virus and from drink at the same time, and only one of them gets written down. Which one wins is a recording decision, not a medical fact. Alcohol damages the liver. Heavy-drinking regions should therefore record the most hepatitis deaths. In Russia they do not.

We use a panel of all 85 Russian regions for 2012–2019 and estimate spatial panel models under a correlated random effects specification, which separates change within regions from differences between them.

Within regions, rising alcohol consumption predicts *fewer* registered hepatitis deaths (urban $\beta = -0.126$, $p < 0.001$; rural $\beta = -0.081$, $p = 0.004$), while regions drinking persistently more record more of them. This is the signature of a competing cause of death. Widening the outcome to cover the neighbouring liver codes makes the effect vanish in urban areas: the deaths are still recorded, but no longer as hepatitis. Fixed-effects designs cannot see this: they discard the between-region margin and report the temporal coefficient alone.

The signature recurs earlier in the process: tuberculosis reverses sign across the margins too, competing for diagnostic attention rather than for

the cause itself.

Rank Russian regions by hepatitis mortality and you rank certifying practice as much as disease. The mechanism is not about hepatitis or alcohol or Russia. Any two conditions that can kill the same organ compete for the same line, and the numbers used to estimate burden and allocate health spending are biased accordingly.

Keywords: Viral hepatitis, Spatial panel data, Correlated random effects, Compositional and contextual effects, Disease surveillance, Health geography, Russia

1. Introduction

Every death gets one underlying cause. Chronic disease rarely obliges. A liver fails after years of infection and years of drinking, and the certificate takes only one. A physician decides, under time pressure and with what the local system can establish. That decision has a cost, and the cost differs across places. So does the bias it leaves behind.

Russia faces a substantial health burden related to viral hepatitis, with hepatitis C remaining the dominant contributor to chronic infectious morbidity. According to national surveillance data, more than 45 thousand new cases of chronic hepatitis C were registered in 2019, corresponding to an incidence of approximately 31 cases per 100,000 population, while the officially reported incidence of acute hepatitis B and C remained below 1 case per 100,000, reflecting the long asymptomatic course and delayed diagnosis of chronic infections (Akimkin and Totolyan, 2024). Over the period 2012–2019, registered incidence rates of viral hepatitis demonstrate relative

stabilization rather than a sustained decline, indicating persistent structural challenges in prevention, early detection, and linkage to care across Russian regions (Akimkin and Totolyan, 2024). Nevertheless, the Russian Federation remains far from the targets set by the World Health Organization, which call for a 90% reduction in new infections and a 65% reduction in hepatitis-related mortality by 2030, emphasizing the importance of improving treatment coverage and regional health system performance (World Health Organization, 2017, 2024).

Clinically, hepatitis burden is driven by parenteral and iatrogenic transmission and by the cofactors governing progression of chronic liver disease: unsafe injection and procedural practices (Tavoschi et al., 2019; Folkema et al., 2021), historical injection campaigns that left large reservoirs of chronic infection (Frank et al., 2000; Miller and Abu-Raddad, 2010), healthcare-associated transmission in procedure-intensive populations (Stuyver et al., 1996; Radcliffe et al.; Kenfack-Momo et al., 2024), and vertical transmission for HBV (Ngui et al., 1998). Donor screening has sharply reduced transfusion-related risk (Donahue et al., 1992), while progression to cirrhosis and hepatocellular carcinoma depends on duration of infection and alcohol consumption (Roudot-Thoraval et al., 1997).

These mechanisms operate on individuals; what varies across regions is the environment that determines whether an infection is ever recorded. Notified cases reflect not only the underlying risk of infection but also progression and diagnosis: an individual must acquire infection, reach a clinically detectable stage, and then be diagnosed—often incidentally, during screening or treatment for HIV or tuberculosis rather than through hepatitis-specific

testing (Chen et al., 2009). Comorbidity is therefore central to observed hepatitis statistics. HIV accelerates hepatitis C progression and raises liver-related mortality, so coinfecting individuals enter clinical care sooner (Hernandez and Sherman, 2011); tuberculosis care involves routine liver function monitoring, and coinfecting patients face elevated hepatotoxicity risk during treatment (Mo et al., 2014). Regional variation in registered hepatitis may thus partly capture the distribution of HIV and TB comorbidity and of healthcare contact, rather than transmission alone (Chang et al., 2018).

Which infections occur, and which are found, is in turn patterned by demographic, behavioural and social conditions—age, migration, drug use, housing instability, incarceration—that shape exposure, timely diagnosis and survival (Galea and Vlahov, 2002; Tosun et al., 2018). These proximal determinants mediate more distal socioeconomic factors operating as “fundamental causes” of health inequalities (Clouston et al., 2016). Risk-environment research shows how structurally disadvantaged places create layered vulnerabilities to HCV (Cloud et al., 2019), while prisons amplify blood-borne infection with spillovers to the community after release (Awofeso, 2010). Even in the treatment era, barriers along the care cascade limit uptake and downstream mortality gains despite highly effective antivirals (Radley et al., 2019; Gonzalez Corro et al., 2024; Lenton et al., 2024). Population-level patterns of burden and liver-related mortality therefore reflect the unequal distribution of diagnosis and access to care as much as transmission (Hofmeister et al., 2018; Hamill et al., 2023), consistent with syndemic framings that integrate social determinants and HIV/TB comorbidity across the disease pathway (Duarte et al., 2018).

At the community level, deprivation is positively associated with hepatitis B and C prevalence net of demographic structure and screening (Omland et al., 2013; Okui and Nakashima, 2022), and socioeconomic differences in cirrhosis mortality persist through macroeconomic shocks (Borrell et al., 2021); at the individual level, low socioeconomic status predicts higher HCV risk and worse prognosis even under direct-acting antivirals (Barré et al., 2024), interacting with incarceration and administrative barriers to care (Chin et al., 2024; Jackson et al., 2024).

Spatial epidemiological approaches to hepatitis remain underdeveloped relative to tuberculosis, with most studies confined to isolated city, regional, or national analyses. Subnational clustering of HCV linked to local deprivation has been shown in Western Europe (Kauhl et al., 2015) and Japan (Okui and Nakashima, 2022); geographically structured hepatitis–HIV coinfection in Spain (Cachafeiro et al., 2011); within-country variation in Brazil (Avanzi et al., 2018; Kuiava et al., 2019); spatial autocorrelation in Chinese HCV incidence tied to urbanisation (Yang et al., 2024); urban co-clustering of hepatitis and HIV mortality (Meyers et al., 2014); and underestimation of hepatitis mortality under underlying-cause coding (Ly et al.). Globally, hepatitis mortality shows strong spatial inequality by income and health-system capacity (Li et al., 2023), yet a recent review confirms spatial analytics in hepatitis remain limited relative to disease burden (Gizamba et al., 2025). Large-scale spatial evidence on hepatitis comorbidity with HIV/TB is scarcer still, most work being non-spatial or single-setting (Harling and Castro, 2014; Mussina et al., 2023; Grigoriev et al., 2025).

Within Russia, viral hepatitis has been studied predominantly within

medical and medico-social frameworks—epidemiological trends, transmission pathways, and occupational or healthcare-associated risks including parenteral exposure among medical workers and vulnerable groups—rather than through explicit spatial or socioeconomic modelling (Kosagovskaya and Volchkova, 2013; Tavošchi et al., 2019; Bykov et al., 2022). Where a spatial and socioeconomic framing has been applied to a Russian infectious disease, it has been to tuberculosis rather than hepatitis (Shartova et al., 2023); indirect evidence on hepatitis emerges mainly through related infections such as HIV, where regional disparities have been linked to mobility, treatment access, and socioeconomic conditions (Nikolaev, 2025). Existing work also relies on simple contiguity-based weights and linear diffusion assumptions poorly suited to institutional and mobility mechanisms—incarceration, prisoner transport, post-release reintegration—that reshape exposure beyond residential proximity (Moran et al., 2012; Averkieva, 2014). No comprehensive nationwide analysis of the socioeconomic and socio-demographic determinants of viral hepatitis in Russia has been conducted within an explicit spatial framework—a gap particularly salient in a large, heterogeneous country with pronounced regional inequalities in economic conditions, institutional exposure, and healthcare access.

This paper asks a single question: what do regional differences in registered infectious disease actually measure? A case enters surveillance only if someone is infected, progresses to a detectable stage, and meets a health system willing to test. The geography such data describe may therefore be the geography of diagnostic capacity as much as the geography of disease. The distinction is not a technical aside. It determines whether a high-burden

region needs treatment resources or needs screening, and it bears directly on the long-standing question in health geography of whether area differences in health reflect the characteristics of the people who live in a place or properties of the place itself (Macintyre et al., 2002; Cummins et al., 2007).

Answering it requires no data beyond what routine surveillance already produces, but it does require using those data in two specific ways. First, we compare two outcomes that are generated at different levels of the health system. Registered morbidity is an output of primary care, which both prevents infection and finds it; registered mortality is an output of specialist and dispensary care, reached only by patients already diagnosed and enrolled in follow-up. The two levels are staffed, financed and administered separately, so regional prosperity need not act on them through the same channel—and a characteristic that predicts the first while leaving the second untouched is a candidate for detection rather than for burden. Second, we estimate spatial panel models under a correlated random effects specification. Conventional fixed-effects models discard cross-sectional variation and are therefore silent on precisely the persistent between-place differences that motivate the geographic question; the specification used here retains both margins at once, and the comparison between them turns out to be diagnostic in its own right.

Applied to an eight-year panel covering all 85 Russian federal subjects, this design returns a consistent answer. Income predicts registered morbidity strongly and mortality not at all. Tuberculosis, alcohol and HIV carry opposite signs within and between regions, so a fixed-effects estimator would not merely miss these relationships but report them with the wrong sign. And the pronounced spatial clustering of hepatitis disappears from the residuals

once regional characteristics are conditioned on, indicating that neighbouring regions resemble one another in composition rather than exchanging disease across administrative borders at this scale. Each result is partial on its own; together they describe a surveillance geography that tracks where disease is found at least as closely as where it occurs.

2. Methods

2.1. Conceptual framework

The empirical strategy rests on a simple institutional observation: the two outcomes analysed here are produced by different parts of the health system, and regional resources reach those parts through different channels.

Registered morbidity originates in primary care. The same first-contact tier that delivers prevention—harm reduction, vaccination, infection control in routine procedures—is also the tier that performs case-finding, since chronic hepatitis is largely asymptomatic and enters the register through screening rather than through presentation. Prevention and detection are thus not merely correlated across regions: they are functions of one organisational level, and they enter the registered count with opposite signs. Better prevention lowers true incidence; better detection raises the recorded share of whatever incidence exists. A regional count is the net of the two.

Registered mortality originates further along the pathway. Death from viral hepatitis follows cirrhosis or hepatocellular carcinoma, conditions managed by specialist hepatology and infectious disease services under dispensary follow-up (*dispanserny uchyot*), the standing register through which chronically ill patients receive scheduled monitoring and treatment. Mortality

therefore describes outcomes within an already selected population: those whose infection was found, who were enrolled, and who remained in contact with specialist care.

In Donabedian’s terms, the two outcomes summarise different structures and different processes (Donabedian, 1988). This matters for interpretation. If registered morbidity and mortality were outputs of a single latent “health-system capacity,” any covariate correlated with that capacity would move both in the same direction, and no contrast between them could be informative. Because they are outputs of separately funded and separately staffed tiers, they are free to move independently, and the pattern of their divergence carries information about which tier a covariate operates on.

The identifying logic follows directly. A regional characteristic operating through *detection* acts on the first tier and should load on morbidity while leaving mortality unaffected. One operating through *burden* should load on mortality, and on morbidity only to the extent that greater true incidence is also found. One operating through *treatment quality* should act on the second tier and attenuate mortality without moving morbidity at all. These are distinct signatures, and the outcome contrast discriminates among them without requiring data on screening volumes that Russian regional statistics do not report.

The framework also disciplines the most serious alternative reading of our central result. Should income predict morbidity but not mortality, one could object that wealthier regions simply detect more *and* treat better, the two effects cancelling to a null mortality coefficient. The objection is coherent, but under the tier structure it requires an exact offset between improvements

at two organisationally distinct levels—first-contact case-finding and specialist management—that are budgeted, staffed and regulated apart from one another. Such a coincidence is possible; it is not a parsimonious default, and the burden of argument lies with it rather than with the detection reading. We return to this in Section 4.

2.2. Study area

The unit of analysis is the constituent entity (federal subject) of the Russian Federation; during the study period Russia comprised 85 such subjects—republics, oblasts, krais, autonomous districts, and federal cities. The analysis uses annual panel data for 2012–2019, capturing a phase of relative epidemiological stability in hepatitis reporting and avoiding structural breaks from the COVID-19 pandemic. Morbidity indicators come from Form No. 12 (newly registered infectious-disease cases) and mortality from Form C-51 (cause-specific deaths), both compiled within the national surveillance system and disseminated by the Central Research Institute for Health Organization and Informatics of the Ministry of Health (CNIIOIZ).

The registered viral hepatitis burden captured by these forms is overwhelmingly chronic. In the national case composition, chronic hepatitis C accounts for roughly three-quarters of all registered viral hepatitis (approximately 74%) and chronic hepatitis B for a further 15%, so that chronic infection constitutes close to 90% of the total; acute forms are marginal, and acute hepatitis A—epidemiologically distinct in its faecal–oral transmission and symptomatic, self-limiting course—amounts to on the order of 0.1% of registered cases. This composition is stable across the study period. The outcome analysed here therefore measures the chronic, largely

asymptomatic hepatitis burden dominated by hepatitis C, precisely the disease process whose registration depends on regional diagnostic and screening capacity rather than on acute symptomatic presentation. This is the population for which the detection mechanism developed below is substantively relevant, and it is why acute, sanitation-driven infection does not confound the socioeconomic gradients we recover.

Age standardization is not feasible in this setting. Form No. 12 does not provide age-disaggregated information on incident cases, and detailed age-specific mortality data for viral hepatitis are not publicly available at the regional level. Consequently, the analysis relies on crude morbidity and mortality rates, with regional demographic and socioeconomic characteristics included as controls to partially account for population structure.

Spatial data use official administrative boundaries for the study period, reflecting Russia’s de facto administrative geography including Crimea and Sevastopol after 2014, based on the State Geodetic Reference System.

2.3. Region-based data collection

Beyond the hepatitis outcomes (Form No. 12 and Form C-51), regional sociodemographic and economic characteristics—population, income, employment, migration—were collected from the Unified Interdepartmental Information and Statistical System (EMISS). To capture institutional exposure, we added convictions by crime severity and seized narcotics (EMISS, reported by the Ministry of Internal Affairs) and penitentiary indicators from the Federal Penitentiary Service (FSIN), cross-checked against public sources where regional counts were incomplete. All variables were harmonised for temporal and spatial consistency.

2.4. Spatial weights

Spatial dependence is modelled through an $N \times N$ weights matrix W with zero diagonal, constructed from first-order contiguity between the administrative boundaries of federal subjects and row-standardised so that $\sum_j w_{ij} = 1$ for every i .

A strict contiguity rule leaves several federal subjects without neighbours: Kaliningrad Oblast is an exclave with no land border with another Russian region, Sakhalin Oblast is an island, and the Republic of Crimea and the federal city of Sevastopol are connected to the mainland only by the Kerch crossing from 2018. Rather than impute artificial adjacencies for these units, we retain them in the sample with an all-zero row in W , so that their spatial lag $\sum_j w_{ij}y_{jt}$ is identically zero: they contribute to estimation through their own covariates but neither receive nor transmit a modelled spatial spillover. This preserves the full set of $N = 85$ subjects while making no unwarranted assumption about the neighbours of geographically isolated regions (the weights list is constructed with a zero-row policy for isolates rather than dropping them). The choice is conservative for our purposes, since assigning these units non-zero weights could only add spatial structure; that the estimated λ and ρ remain indistinguishable from zero is therefore not an artefact of how isolates were treated. We note that a contiguity rule is a strong assumption in this setting: Russian federal subjects differ in area by three orders of magnitude, and adjacency between two Siberian territories the size of Western Europe carries a different epidemiological meaning than adjacency between two Central Federal District oblasts. Section 4 returns to this point.

2.5. *Spatial autoregression methods*

The empirical analysis follows the standard exploratory–confirmatory sequence in spatial data analysis. We detect spatial dependence using global Moran’s I (Moran, 1948), local indicators of spatial association (LISA) for clusters and outliers (Anselin, 1995), and the distance-based Getis–Ord G_i^* statistic for local concentration (Getis and Ord, 1992). Because these ESDA tools cannot accommodate temporal dynamics or unobserved heterogeneity in panels, we complement them with a space–time framework (Rey and Janikas, 2006) and base formal inference on bootstrap Moran’s I tests, which mitigate finite-sample distortion (Ren et al., 2014).

To account for spatial dependence while retaining the ability to estimate the effects of time-invariant regional characteristics, we estimate spatial panel models using a correlated random effects specification in the spirit of Mundlak (1978). The correlated random effects (CRE) approach allows for correlation between unit-specific unobserved heterogeneity and time-varying covariates by augmenting the model with unit-specific time averages of the regressors. This strategy provides an empirically tractable alternative to fixed effects models when time-invariant regressors are of substantive interest (Mundlak, 1978).

In the spatial panel context, this approach has been extended to spatial Durbin panel models by Debarsy (2012), who shows that modelling the correlation between individual effects and regressors through time averages yields consistent estimators while preserving the identification of time-constant variables. More recently, Baltagi (2023) demonstrates that in spatial panel models with spatially correlated disturbances the classical equivalence

between fixed effects and the Mundlak specification no longer holds exactly, but the correlated random effects spatial estimator can still be consistently estimated via maximum likelihood and provides a practical framework for empirical analysis.

Let $i = 1, \dots, N$ index regions and $t = 1, \dots, T$ index time periods. Denote by y_{it} the morbidity (or mortality) rate in region i at time t , by y_{jt} the same rate in a neighbouring region j at time t , by X_{it} a vector of time-varying regional covariates, and by Z_i a vector of time-invariant regional characteristics. Let $W = \{w_{ij}\}$ denote an $N \times N$ spatial weights matrix with zero diagonal elements, whose entry w_{ij} measures the spatial proximity of regions i and j ; the sums $\sum_{j=1}^N w_{ij}y_{jt}$ below therefore run over all regions and, given the zero diagonal, exclude region i itself.

Following the spatial correlated random effects specification, the spatial autoregressive model with autoregressive disturbances (SARAR) in Mundlak form can be written as

$$y_{it} = \lambda \sum_{j=1}^N w_{ij}y_{jt} + X_{it}\beta + \bar{X}_i\delta + Z_i\gamma + \tau_t + u_i + \varepsilon_{it}, \quad (1)$$

with a spatially autoregressive disturbance process

$$\varepsilon_{it} = \rho \sum_{j=1}^N m_{ij} \varepsilon_{jt} + \nu_{it}, \quad \nu_{it} \sim IID(0, \sigma_\nu^2), \quad (2)$$

where $M = \{m_{ij}\}$ is a second $N \times N$ spatial weights matrix, with generic element m_{ij} , governing dependence in the disturbances; throughout we set $M = W$, and where the Mundlak terms

$$\bar{X}_i = \frac{1}{T} \sum_{t=1}^T X_{it}$$

are the region-specific time averages of the time-varying regressors. Their inclusion captures the potential correlation between unobserved regional effects and explanatory variables, thereby relaxing the strict exogeneity assumption required by conventional random effects models (Mundlak, 1978). Every symbol appearing in (1)–(2) is defined in Table 1; the substantive content of the variables entering X_{it} and Z_i is given in Section 2.3 and their descriptive statistics in Appendix Appendix A.

The parameter λ captures endogenous spatial spillovers in the outcome, ρ captures spatial correlation in unobserved shocks, and τ_t represents time fixed effects controlling for common temporal shocks. The region-specific effect is assumed to satisfy $u_i \sim IID(0, \sigma_u^2)$, independent of the innovation $\nu_{it} \sim IID(0, \sigma_\nu^2)$, where σ_u^2 and σ_ν^2 denote the variances of the region effect and of the idiosyncratic innovation respectively.

A point of interpretation deserves emphasis, because it governs every comparison reported below. In the augmented specification (1), β identifies the *within*-region association, while δ identifies the *difference* between the between- and within-region associations; the between-region association is therefore recovered as $\beta + \delta$, not as δ itself (Mundlak, 1978; Wooldridge, 2019). The coefficients on $\bar{X}_i$ reported below are accordingly contrasts, and a significant δ constitutes a direct test of the null that a covariate operates identically within and between regions. Where we describe a covariate as carrying opposite within- and between-region associations, we mean that β and $\beta + \delta$ differ in sign, and both are computed directly from the estimated

Table 1: Notation used in the SARAR–Mundlak specification.

Symbol	Definition
i, j	region indices, $i, j = 1, \dots, N$; $N = 85$ federal subjects
t	year index, $t = 1, \dots, T$; $T = 8$ (2012–2019)
y_{it}	registered outcome rate per 100,000 in region i , year t (morbidity, urban mortality, or rural mortality)
$W = \{w_{ij}\}$	row-standardised first-order contiguity matrix; $w_{ij} > 0$ if i and j share a border, $w_{ii} = 0$
λ	spatial autoregressive parameter of the outcome (endogenous spillover)
X_{it}	vector of seven time-varying covariates (HIV, tuberculosis, unemployment, log income, migration, a
β	vector of within-region coefficients
$\bar{X}_i$	region-specific time means of X_{it} (Mundlak terms)
δ	vector of between–within contrasts; the between-region association is $\beta + \delta$
Z_i	time-invariant covariate (prison population per capita)
γ	coefficient on Z_i
τ_t	year fixed effects (2012 is the reference year)
u_i	region-specific random effect, $u_i \sim IID(0, \sigma_u^2)$
ε_{it}	spatially correlated disturbance
$M = \{m_{ij}\}$	weights matrix of the disturbance process; here $M = W$
ρ	spatial autoregressive parameter of the disturbances
ν_{it}	idiosyncratic innovation, $\nu_{it} \sim IID(0, \sigma_\nu^2)$

model. Throughout the results, the coefficient reported for a Mundlak time-average is δ (the between-within contrast); where we characterise a between-region association, the quantity referred to is $\beta + \delta$.

Estimation is performed by maximum likelihood, under the assumption that the innovations ν_{it} are jointly normally distributed, within the asymptotic framework where the number of cross-sectional units N is large relative to the time dimension T , which is typical in regional panel datasets (Lee and Yu, 2010). With $N = 85$ and $T = 8$ the panel is short, and λ and ρ are identified from partially overlapping variation; power against moderate spatial dependence is correspondingly limited, and we interpret the spatial parameters with that caveat in mind. All exploratory spatial statistics and model estimation were carried out in R, using the `spdep`, `spatialreg`, `splm` and `plm` packages together with purpose-written routines for the Mundlak augmentation and for the bootstrap Moran’s I tests.

Functional form and inference.. Outcomes are modelled as crude rates per 100,000 population in levels, with a linear conditional mean, so that coefficients retain a direct interpretation in cases per 100,000 and the within/between contrasts that motivate the analysis are read off the estimates without transformation. The residual distributions are approximately centred but display heavy and somewhat asymmetric tails, which would render inference based on the nominal Gaussian likelihood fragile. We therefore base all inference on cluster-robust (sandwich) standard errors clustered at the region level, which are robust to the heteroskedasticity and within-region serial dependence implied by these tails; the reported t -statistics and p -values throughout Tables 2–4 use these clustered standard errors. Because each outcome model

reports on the order of fifteen coefficients, we treat individual coefficient p -values as descriptive and rest the substantive conclusions on the coefficients that are significant at the one-per-thousand level and that replicate in sign and pattern across the morbidity and the two mortality specifications, rather than on any isolated marginal result.

The Mundlak augmentation tests for correlation between regressors and region effects through the joint significance of the time-averages $\bar{X}_i$, implementable in spatial panels as a likelihood-ratio complement to the Hausman test (Debarys, 2012). It addresses directly the concern that the region effects u_i may be correlated with the time-varying regressors—that the “random” effects are effectively fixed. The CRE specification does not assume this away; it models it through $\bar{X}_i\delta$, and the joint significance of $\bar{X}_i$ tests its presence. Where the test rejects—decisively in all three models (Section 3)—the within coefficient β is identified under the same strict-exogeneity condition as fixed effects, while the specification additionally recovers the between-region contrast δ and retains the time-invariant regressor Z_i , both discarded by fixed effects. Choosing CRE is therefore not an assumption that the region effects are uncorrelated with the regressors, but a decision to model that correlation rather than difference it away, because the between-region gradient is the object of interest.¹ What CRE cannot address, and neither can fixed effects, is

¹The Mundlak device imposes a single time-invariant projection of the region effect onto the regressor means. A more general alternative is the Chamberlain correlated-random-effects parameterisation, which allows the projection to load on the regressors period by period, and one can further probe non-linearity by adding squares of the Mundlak means. With $N = 85$ and $T = 8$ the fully flexible Chamberlain specification is expensive in degrees

correlation between the regressors and *time-varying* unobservables, to which we return in Section 4. Note also that, under spatial error correlation, the spatial CRE estimator does not reduce exactly to fixed effects even with the Mundlak averages included (Baltagi, 2023).

3. Results

3.1. Spatial distribution and exploratory analysis

Registered hepatitis morbidity across Russian regions over the period 2012–2019 exhibits pronounced and persistent spatial heterogeneity. Global Moran’s I statistics were statistically significant ($p < 0.05$) in every year of the study period, indicating systematic spatial autocorrelation rather than random variation across regions (Figure 1). The magnitude of clustering was substantial throughout, ranging from $I = 0.35$ in 2013 to $I = 0.43$ in 2012 and 2018, suggesting that high- and low-incidence regions tend to cluster geographically and that this pattern was not driven by a single anomalous year.

Local indicators of spatial association (LISA) confirm that this global autocorrelation is driven predominantly by a large and coherent High–High cluster in the eastern part of the country (Figure 2). In 2019, significant High–High clustering encompassed Siberian and Far Eastern federal subjects, including Sakha (Yakutia) and neighbouring regions, while a small but

of freedom, so we adopt the parsimonious Mundlak projection in the main analysis and regard these richer parameterisations as available robustness checks rather than as the baseline.

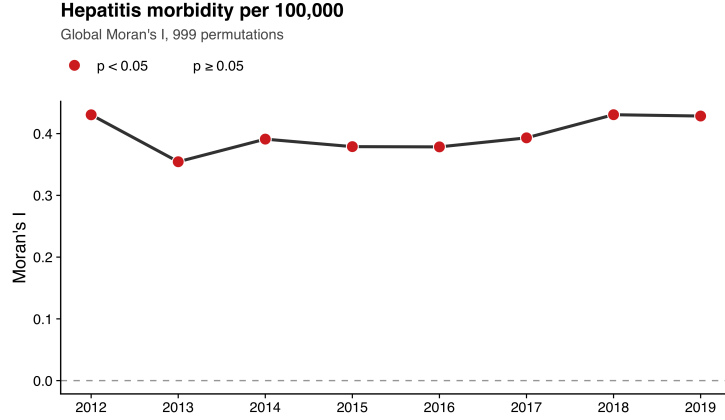


Figure 1: Global Moran’s I for registered hepatitis morbidity per 100,000 population, Russian regions, 2012–2019. All values significant at $p < 0.05$ (bootstrap test).

significant Low–Low cluster was identified in the North Caucasus. The Getis–Ord G_i^* hotspot map for 2019 reinforces this picture: z -scores in the range 4–6 are concentrated in the same eastern macro-region, while strongly negative z -scores (below -3) are observed in a small number of western regions, consistent with the Low–Low LISA pattern (Figure 3).

3.2. *Clustering versus contagion*

The exploratory statistics above establish that hepatitis outcomes are spatially clustered; they do not establish why. Two mechanisms are observationally equivalent on a Moran scatterplot. Neighbouring regions may influence one another, so that burden in one region raises burden in the next—an autoregressive process, captured by λ . Or neighbouring regions may simply resemble one another in the characteristics that generate hepatitis burden and hepatitis registration—a compositional process, captured by the covariates and by their persistent between-region levels. The regression



Figure 2: Local Moran’s I (LISA) cluster map for registered hepatitis morbidity per 100,000 population, 2019. High–High: high-morbidity region surrounded by high-morbidity neighbours; Low–Low: the reverse; High–Low and Low–High are spatial outliers. Regions with no first-order contiguity neighbours (Kaliningrad, Sakhalin, Crimea, Sevastopol) enter the model with a zero-weight row and are shown separately as “no neighbours”; local statistics are undefined for them.

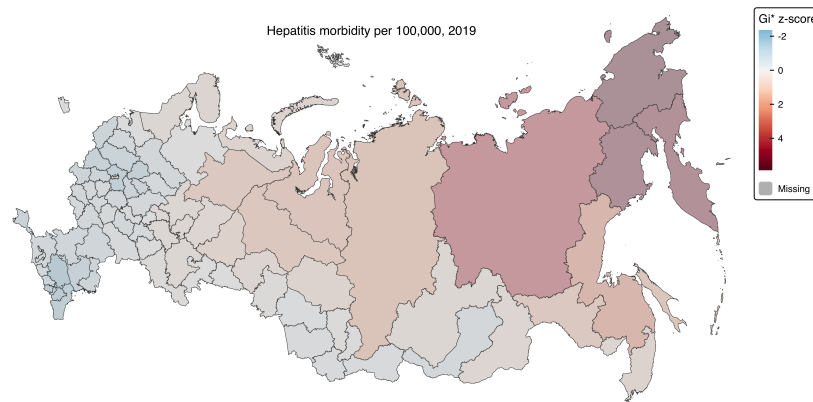


Figure 3: Getis–Ord G_i^* z -scores for registered hepatitis morbidity per 100,000 population, 2019. Values above +2 indicate statistically significant spatial concentration of high morbidity; values below -2 indicate cold spots. Regions with no contiguity neighbours are shown separately.

results below distinguish the two directly. Although the raw outcomes exhibit global Moran’s I between 0.35 and 0.43, once the covariates and their between-region means enter the model the autoregressive parameter λ and the spatial-error parameter ρ are both indistinguishable from zero, and a Moran’s I test on the residuals detects no remaining spatial autocorrelation. The clustering is real; the contagion, at this scale, is not. What looked like interregional diffusion on the Moran scatterplot is absorbed entirely by what kind of place each region is—its income, its comorbidity burden, and the diagnostic capacity that converts infection into a registered case—rather than by which regions it happens to border.

Table 2 reports estimates from the SARAR–Mundlak model for registered morbidity. As anticipated above, the spatial parameters are negligible ($\lambda = -0.024$, $p = 0.72$; $\rho = 0.032$, $p = 0.67$; residual Moran’s $I = -0.047$, $p = 0.68$). The Mundlak likelihood-ratio test strongly rejects uncorrelated unit effects ($\chi^2(7) = 32.53$, $p < 0.001$), validating the CRE specification.

Among the substantive predictors, *log household income* emerges as the dominant positive predictor of hepatitis morbidity. The within-region coefficient is 379.6 (SE = 67.6, $p < 0.001$), and the Mundlak term adds a further 596.3 ($p < 0.001$), so that the between-region association ($\beta + \delta = 975.9$) is larger still, implying that the association between income and registered hepatitis morbidity operates at both temporal and cross-sectional levels and is stronger across regions than within them. This positive income gradient is consistent with a *detection* mechanism: wealthier regions with more developed healthcare infrastructure are better able to identify and register chronic hepatitis cases, rather than reflecting higher underlying biological risk.

HIV incidence is positively associated with registered hepatitis morbidity ($\hat{\beta} = 0.597$, $SE = 0.289$, $p = 0.038$), consistent with co-infection pathways and shared diagnostic contact documented in the literature. *Migration* is negatively associated with morbidity ($\hat{\beta} = -0.375$, $SE = 0.129$, $p = 0.004$), a finding that may reflect compositional effects or differential registration of hepatitis among mobile populations. The Mundlak term for tuberculosis notifications is positive and significant ($\delta_{TB} = 4.389$, $p = 0.009$); since the within-region coefficient is small and insignificant, this places the between-region association ($\beta + \delta = 3.99$) firmly positive, suggesting that between-region variation in comorbidity burden is associated with higher registered hepatitis rates, plausibly through overlapping surveillance and clinical detection pathways. Similarly, the Mundlak term for unemployment is negative and significant ($\delta_{unemp} = -8.957$, $p = 0.018$), and the income Mundlak term is, as noted, strongly positive.

Neither the within-region unemployment rate, tuberculosis notification rate, alcohol consumption, nor narcotic seizure intensity reached conventional significance thresholds in this model. The penitentiary exposure variable (`prison_per_pop`) showed a large positive point estimate (7,982) but wide uncertainty ($SE = 9,016$, $p = 0.38$), precluding firm conclusions from this specification alone.

Year fixed effects indicate a modest downward trend in registered morbidity relative to 2012, with statistically significant reductions observed in 2014 (-42.8 cases per 100,000, $p = 0.024$) and 2015 (-57.8 , $p = 0.019$), consistent with the relative stabilisation of hepatitis reporting documented at the national level over this period.

Spatial impact decomposition confirms that in all three models the estimated direct and indirect (spillover) effects of each covariate are numerically close, a direct consequence of the near-zero spatial lag parameter λ . In the urban mortality model the indirect component is under 1% of the corresponding direct effect throughout; in the rural model it is somewhat larger in magnitude (around 15%) but of *opposite* sign, reflecting its small positive λ combined with a negative spatial-error parameter. In every case the spillover component is too small to alter the substantive interpretation of any covariate.

Table 2: SARAR–Mundlak spatial panel model: registered hepatitis morbidity per 100,000 (outcome: `hep_morb_100k`). Coefficient estimates and standard errors; $N = 85$ regions, $T = 8$ years (2012–2019).

Variable	Estimate	Std. Error	t	p
<i>Spatial parameters</i>				
λ (spatial lag)	−0.024	0.065	−0.36	0.719
ρ (spatial error)	0.032	0.075	0.43	0.665
<i>Within-region (time-varying) covariates</i>				
HIV incidence (per 100k)	0.597	0.289	2.07	0.038*
Tuberculosis notifications	−0.397	0.318	−1.25	0.211
Unemployment rate	0.897	0.789	1.14	0.255
Log household income	379.608	67.622	5.61	< 0.001***
Migration	−0.375	0.129	−2.91	0.004**

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Table 2 continued

Variable	Estimate	Std. Error	<i>t</i>	<i>p</i>
Alcohol consumption	−0.770	3.348	−0.23	0.818
Narcotic seizures (per 100k)	−0.624	1.549	−0.40	0.687
<i>Mundlak time-averages (between-region)</i>				
$\overline{\text{HIV incidence}}$	0.540	1.316	0.41	0.682
$\overline{\text{Tuberculosis}}$	4.389	1.685	2.61	0.009**
$\overline{\text{Unemployment}}$	−8.957	3.793	−2.36	0.018*
$\overline{\text{Log income}}$	596.266	165.530	3.60	< 0.001***
$\overline{\text{Migration}}$	−1.102	0.875	−1.26	0.208
$\overline{\text{Alcohol}}$	−20.639	29.377	−0.70	0.482
$\overline{\text{Narcotics}}$	−9.082	21.513	−0.42	0.673
<i>Time-invariant covariate</i>				
Prison population (per capita)	7,981.6	9,015.9	0.89	0.376
<i>Year fixed effects (reference: 2012)</i>				
2013	−17.330	16.519	−1.05	0.294
2014	−42.782	18.956	−2.26	0.024*
2015	−57.764	24.657	−2.34	0.019*
2016	−40.546	24.567	−1.65	0.099
2017	−46.881	26.386	−1.78	0.076
2018	−53.787	28.511	−1.89	0.059
2019	−45.046	31.352	−1.44	0.151
<i>Model diagnostics</i>				
LR test (Mundlak): $\chi^2(7) = 32.53, p < 0.001$				
Moran's <i>I</i> on residuals: −0.047 ($p = 0.677$)				

Significance codes: *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, $p < 0.1$

3.3. Spatial distribution of hepatitis mortality

Hepatitis-related mortality in urban and rural populations exhibits distinct but overlapping spatial structures. For urban mortality (`hep_urban_100k`), global Moran’s I was positive and significant in six of the eight study years (I between 0.07 and 0.27; 2015 and 2017 non-significant, Figure B.8), with a dominant High–High cluster in Central Siberia and the Krasnoyarsk–Irkutsk corridor visible in both the LISA map and the G_i^* hotspot surface (Figures B.4 and B.5). Notably, Low–High spatial outliers appear in the Far East, indicating regions with comparatively low urban mortality that are geographically embedded within high-mortality neighbours—a pattern consistent with localized variation in healthcare access or registration practices within an otherwise high-burden macro-region.

Rural hepatitis mortality (`hep_rural_100k`) displays a broadly similar geographic footprint, with High–High clustering concentrated in the same Siberian belt and extending into parts of the Far East (Figures B.6 and B.7). Low–High outliers are more numerous in the rural specification, suggesting greater within-region heterogeneity in rural mortality than in urban mortality. Global Moran’s I for rural mortality was statistically significant ($p < 0.05$) in five of the eight study years, with values ranging from approximately 0.03 to 0.20; three years (2013, 2015 and 2017) yielded non-significant statistics, pointing to intermittent rather than uniformly stable spatial clustering (Figure B.9). The autocorrelation in the rural specification is lower and less stable than in the aggregate morbidity series ($I \approx 0.35$ – 0.43 , Figure 1), consistent with greater heterogeneity in rural health system coverage

and reporting completeness. The corresponding cluster and hotspot maps for urban and rural mortality, and the rural Moran’s I series, are reported in Appendix Appendix B (Figures B.4–B.9).

3.4. Regression results: urban and rural mortality

Tables 3 and 4 report estimates from the SARAR–Mundlak models for urban and rural hepatitis mortality respectively. As in the morbidity specification, the spatial lag (λ) and spatial error (ρ) parameters are indistinguishable from zero in both models, and post-estimation Moran’s I tests confirm the absence of residual spatial autocorrelation (urban: $I = -0.183$, $p = 0.99$; rural: $I = -0.113$, $p = 0.92$). The Mundlak LR test is highly significant in both cases (urban: $\chi^2(7) = 32.63$, $p < 0.001$; rural: $\chi^2(7) = 41.65$, $p < 0.001$), confirming that region-specific effects are correlated with the regressors and that the correlated random effects specification is warranted.

The two mortality models return one pattern rather than two, and it is the pattern rather than the individual coefficients that carries the argument; the full estimates are reported in Tables 3 and 4. Tuberculosis notifications are strongly negative within region and strongly positive between regions in both specifications. Alcohol repeats the same divergence—negative within, positive between—more firmly in the rural model than in the urban. HIV incidence is the strongest positive within-region predictor of urban mortality, but its Mundlak term is negative in both models, so that the between-region association reverses sign in each; the reversal is taken up in Section 4. Migration is positively associated with rural mortality only. Time effects are largely uninformative, the one exception being an upward movement in urban mortality in 2019 (+0.647, $p = 0.037$).

Table 3: SARAR–Mundlak spatial panel model: urban hepatitis mortality per 100,000 (**hep_urban_100k**). $N = 85$ regions, $T = 8$ years (2012–2019).

Variable	Estimate	Std. Error	t	p
<i>Spatial parameters</i>				
λ (spatial lag)	−0.057	0.117	−0.48	0.628
ρ (spatial error)	0.008	0.130	0.06	0.949
<i>Within-region covariates</i>				
HIV incidence (per 100k)	0.0110	0.0029	3.81	< 0.001***
Tuberculosis notifications	−0.0114	0.0032	−3.59	< 0.001***
Unemployment rate	0.0025	0.0079	0.31	0.755
Log household income	−0.5776	0.6746	−0.86	0.392
Migration	0.0024	0.0013	1.88	0.060
Alcohol consumption	−0.1263	0.0333	−3.79	< 0.001***
Narcotic seizures (urban)	−0.0174	0.0175	−0.99	0.320
<i>Mundlak time-averages</i>				
$\overline{\text{HIV}}$	−0.0184	0.0048	−3.82	< 0.001***
$\overline{\text{Tuberculosis}}$	0.0286	0.0059	4.88	< 0.001***
$\overline{\text{Unemployment}}$	0.0007	0.0136	0.05	0.958
$\overline{\text{Log income}}$	0.9730	0.8193	1.19	0.235
$\overline{\text{Migration}}$	−0.0016	0.0030	−0.54	0.589
$\overline{\text{Alcohol}}$	0.1644	0.0956	1.72	0.085
$\overline{\text{Narcotics (urban)}}$	0.1298	0.0593	2.19	0.029*
<i>Time-invariant</i>				
Prison population (per capita)	−28.44	30.01	−0.95	0.343

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Table 3 continued

Variable	Estimate	Std. Error	t	p
<i>Year fixed effects (reference: 2012)</i>				
2013	0.273	0.162	1.69	0.092
2014	0.238	0.187	1.28	0.202
2015	0.237	0.236	1.00	0.315
2016	0.217	0.243	0.89	0.371
2017	0.349	0.261	1.34	0.181
2018	0.470	0.282	1.66	0.096
2019	0.647	0.310	2.09	0.037*
<i>Model diagnostics</i>				
LR test (Mundlak): $\chi^2(7) = 32.63$, $p < 0.001$				
Moran's I on residuals: -0.183 ($p = 0.989$)				
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, $p < 0.1$				

Table 4: SARAR–Mundlak spatial panel model: rural hepatitis mortality per 100,000 (hep_rural_100k). $N = 85$ regions, $T = 8$ years (2012–2019).

Variable	Estimate	Std. Error	t	p
<i>Spatial parameters</i>				
λ (spatial lag)	0.087	0.131	0.67	0.504
ρ (spatial error)	-0.150	0.155	-0.97	0.332
<i>Within-region covariates</i>				

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Table 4 continued

Variable	Estimate	Std. Error	<i>t</i>	<i>p</i>
HIV incidence (per 100k)	0.0023	0.0025	0.91	0.362
Tuberculosis notifications	−0.0135	0.0027	−5.06	< 0.001***
Unemployment rate	0.0078	0.0067	1.18	0.239
Log household income	0.2783	0.5671	0.49	0.624
Migration	0.0023	0.0011	2.10	0.036*
Alcohol consumption	−0.0808	0.0282	−2.87	0.004**
Narcotic seizures (rural)	0.0007	0.0088	0.08	0.939
<i>Mundlak time-averages</i>				
$\overline{\text{HIV}}$	−0.0079	0.0039	−2.03	0.042*
$\overline{\text{Tuberculosis}}$	0.0302	0.0049	6.15	< 0.001***
$\overline{\text{Unemployment}}$	0.0009	0.0113	0.08	0.939
$\overline{\text{Log income}}$	−0.4330	0.6831	−0.63	0.526
$\overline{\text{Migration}}$	−0.0023	0.0023	−0.98	0.329
$\overline{\text{Alcohol}}$	0.1769	0.0802	2.20	0.027*
$\overline{\text{Narcotics (rural)}}$	0.0231	0.0680	0.34	0.734
<i>Time-invariant</i>				
Prison population (per capita)	−14.28	24.71	−0.58	0.563
<i>Year fixed effects (reference: 2012)</i>				
2013	0.046	0.123	0.38	0.705
2014	−0.012	0.145	−0.08	0.936
2015	−0.026	0.208	−0.12	0.902
2016	−0.025	0.194	−0.13	0.898
2017	−0.076	0.210	−0.36	0.716
2018	0.228	0.229	1.00	0.319

continued on next page

Table 4 continued

Variable	Estimate	Std. Error	<i>t</i>	<i>p</i>
2019	0.268	0.253	1.06	0.288
<i>Model diagnostics</i>				
LR test (Mundlak): $\chi^2(7) = 41.65, p < 0.001$				
Moran's <i>I</i> on residuals: -0.113 ($p = 0.918$)				
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, $p < 0.1$				

3.5. Widening the mortality outcome: a test of the coding mechanism

If the negative within-region alcohol coefficient reflects deaths moving between codes rather than fewer deaths, then widening the outcome to include the codes that could absorb them should make it disappear. Form C-51 records those codes separately. We re-estimate both mortality models on progressively wider outcomes: viral hepatitis alone (B15–B19), hepatitis plus alcoholic liver disease (K70), and both plus unspecified fibrosis and cirrhosis (K74). All are crude rates per 100,000, so the wider series is the sum of its parts and the coefficients remain directly comparable with those of Tables 3 and 4.

Table 5 reports the result. In urban mortality the effect disappears once the envelope is closed. It does not disappear when alcoholic liver disease is added—only when unspecified cirrhosis is. The between-region association stays positive throughout. The rural series does not follow this pattern: the coefficient attenuates at the first step and returns at the second.

Table 5: Widening the mortality outcome across the liver envelope of Form C-51: alcohol coefficients from the SARAR–Mundlak specification. β_{alc} is the within-region coefficient, δ_{alc} the Mundlak between–within contrast. Outcomes are crude rates per 100,000; $N = 85$ regions, $T = 8$ years.

Dependent variable	Urban β_{alc}	Urban δ_{alc}	Rural β_{alc}	Rural δ_{alc}
Hepatitis (B15–B19)	−0.047**	0.085	−0.028	0.091*
+ alcoholic liver disease (K70)	−0.032**	0.225***	−0.014	0.271***
+ unspecified cirrhosis (K74)	−0.007	0.121***	−0.024**	0.187***

Significance codes: *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, $\cdot p < 0.1$

4. Discussion

We conducted univariate spatial and multivariable panel analyses of viral hepatitis morbidity and mortality across Russian regions over the period 2012–2019. In the exploratory analysis, registered hepatitis morbidity was found to be strongly and persistently spatially clustered, with a dominant High–High corridor concentrated in Siberia and the Far East. Urban and rural mortality shared a broadly similar geographic footprint, although spatial autocorrelation was weaker and less temporally stable in the rural specification. In the multivariable models, hepatitis morbidity was associated with higher household income, HIV incidence, and in-migration, while both mortality outcomes were governed by the same small set of factors—HIV, tuberculosis comorbidity, alcohol, and drug market intensity—entering with

opposite signs within and between regions. Across all three specifications, spatial and error autocorrelation parameters were negligible conditional on the covariates and correlated random effects, and residual spatial dependence was absent in post-estimation diagnostics. That last result is not a null finding to be reported and set aside; it is the paper’s organising claim, and we take it up first.

Spatial clustering and geographic heterogeneity.. The strong and sustained positive spatial autocorrelation in hepatitis morbidity (Moran’s $I \approx 0.35$ – 0.43 across all study years) reflects the well-documented geographic concentration of hepatitis burden in eastern Russia, where historically high rates of parenteral transmission, elevated HIV prevalence, and comparatively weaker healthcare infrastructure converge (Akimkin and Totolyan, 2024). The Siberian High–High cluster identified in LISA and G_i^* maps is consistent with national surveillance data showing persistently above-average hepatitis C incidence in regions such as Krasnoyarsk, Irkutsk, and Sakha (Yakutia), and parallels spatial clustering patterns documented for HIV in the same macro-region (Nikolaev, 2025). The presence of Low–High spatial outliers in the Far East—regions with comparatively low morbidity or mortality embedded within a high-burden neighbourhood—points to within-cluster heterogeneity that may reflect differential healthcare access and registration completeness rather than truly lower disease burden.

Geography as solo composition.. The striking feature of the regression results is what is absent from them. In all three models the spatial lag and error parameters are indistinguishable from zero, and no residual spatial dependence survives post-estimation—even though the raw outcomes display Moran’s I

between 0.35 and 0.43. The clustering is real; the contagion is not. Once regional characteristics and their persistent between-region levels are conditioned on, knowing which regions border a subject adds nothing to knowing what kind of place it is: adjacency, at this scale, is a proxy for similarity, not a channel of transmission.

This reads directly in the compositional–contextual vocabulary of health geography (Macintyre et al., 2002; Cummins et al., 2007). The Siberian corridor is not a corridor because hepatitis moves along it, but because the regions composing it share the income, comorbidity, drug-market and diagnostic attributes that generate registered hepatitis—and because those attributes are themselves geographically sorted. Two qualifications apply. First, the federal subject is a coarse unit: person-to-person transmission operates at the municipal scale, and aggregating it to subjects the size of a mid-sized country averages the local diffusion signal toward zero, so a null λ is what a compositional account predicts rather than a failure to detect structure. Second, with $N = 85$ and $T = 8$ power against moderate λ is limited, and the Mundlak means absorb some of the cross-sectional variation a spatial process would mark; we therefore do not claim to have excluded diffusion, only that none survives conditional on composition—the residual Moran’s I tests, null in all three models, being the strongest statement the design supports.

Detection interpretation of the income gradient in morbidity.. The most prominent morbidity finding is the strong positive association between household income and registered hepatitis, at both within- and between-region levels—the inverse of the poverty gradient typically seen for infectious diseases such as tuberculosis (Gonçalves et al., 2009; Harling and Castro, 2014). We read

it as primarily a *detection* effect: wealthier regions have more developed infrastructure, higher screening rates, and greater capacity to diagnose conditions like hepatitis C that stay asymptomatic for years (Chen et al., 2009), as argued for registered hepatitis gradients in Japan and Western Europe (Omeland et al., 2013; Okui and Nakashima, 2022). This does not preclude a genuine biological component, but it cautions against reading registered morbidity as a direct proxy for true incidence—a caution the next paragraph turns into a test.

Two outcomes, two data-generating processes.. A detection interpretation is easy to assert and, on the morbidity model alone, impossible to refute. The design, however, contains an internal test requiring no additional data. Registered morbidity and mortality are produced by different administrative processes: a chronic case enters the morbidity count only if the system looks for it and finds it, whereas a death brings the individual into contact with the system irrespective of prior screening. Mortality registration is not independent of diagnosis—attributing a death to hepatitis still requires the infection to be known—but it is markedly less sensitive to the intensity of case-finding. Covariates that operate through detection should therefore load on morbidity and fade on mortality; those operating through burden should do the reverse or load on both.

Three covariates occupy three points on that spectrum. *Income* dominates registered morbidity at both margins ($\beta = 379.6$, $\delta = 596.3$, both $p < 0.001$) yet is unrelated to mortality, urban ($\beta = -0.578$, $p = 0.39$) or rural ($\beta = 0.278$, $p = 0.62$): prosperity predicts precisely whether a case is found and nothing about whether a person dies—the signature of a detec-

tion gradient, hard to reconcile with income raising true burden. *Migration* inverts across outcomes, negative on morbidity ($\beta = -0.375$, $p = 0.004$) but positive on rural mortality ($\beta = 0.002$, $p = 0.036$)—the pattern of a group under-diagnosed yet carrying real burden, invisible in either outcome alone. *HIV incidence* loads positively on both morbidity ($p = 0.038$) and urban mortality ($p < 0.001$), operating through genuine comorbidity as well as shared diagnostic contact.

This triad constrains the most damaging alternative reading: that income, HIV, and the between-region tuberculosis term are all proxies for one factor—health-system capacity—so that a single detection mechanism generates every result. But a common capacity factor would move all outcomes in the same direction; ours do not—income moves morbidity not mortality, migration moves the two oppositely, HIV moves both—and three distinct signatures cannot come from one scalar.

A second objection is more specific and more serious: wealthier regions may both detect more and treat better, so that a genuine burden gradient and a treatment gradient offset one another to produce the null mortality coefficient we observe. Regional antiviral coverage is not published, so we cannot exclude this arithmetically. What weighs against it is the institutional structure set out in Section 2.1. Case-finding is a function of primary care; survival among diagnosed patients is a function of specialist and dispensary services. These tiers are budgeted, staffed and administered separately, and regional prosperity reaches them through different channels. The offsetting story therefore requires improvements at two organisationally distinct levels to cancel almost exactly, in every region and in every year of the panel, with

no residual visible at either margin. That is possible, but it is a coincidence rather than a default, and the burden of argument rests with it. The detection reading requires no such coincidence: it requires only that prosperity raise the probability a chronic infection is found.

Taken together, the outcome contrast and the tier structure that generates it constitute the strongest evidence available in these data, and they point consistently in one direction. A direct measure of screening volume, or of registrations disaggregated by acute and chronic presentation, would place the interpretation on firmer ground still.

Comorbidity evidence: HIV and tuberculosis.. HIV incidence positively predicts registered morbidity and urban mortality, consistent with two mechanisms: HIV–HCV co-infection accelerates liver disease and clinically detectable mortality (Hernandez and Sherman, 2011), and HIV-diagnosed individuals enter care more readily and receive incidental hepatitis testing, inflating registered morbidity where HIV burden is higher. The negative between-region HIV term—high-HIV regions showing lower rural hepatitis mortality—fits the concentration of both HIV and hepatitis services in urban centres, leaving rural populations in high-HIV regions underserved, echoing Brazilian evidence linking urban–rural comorbidity gradients to differential healthcare contact (Duarte et al., 2018). Tuberculosis notifications divide along the same two margins but in the opposite direction: *negatively* associated with hepatitis mortality within regions (strongly in both mortality models, $p < 0.001$; same direction but insignificant in morbidity, $\beta = -0.397$, $p = 0.21$) yet *positively* associated between them throughout. The negative within-region effect is unlikely to be biological antagonism; more plausibly,

years of rising TB burden divert diagnostic attention and resources toward respiratory surveillance and away from hepatitis detection, or shared social determinants improving over time lower both series through different channels. The positive between-region term fits the established TB–hepatitis syndemic: regions with chronically higher TB burden also have higher poverty, incarceration, and immune-compromising conditions (Mo et al., 2014; Duarte et al., 2018). In both comorbidities the opposite signs across margins underscore the value of the CRE framework: a fixed-effects estimator would have seen only the negative temporal effect and drawn a misleading conclusion about comorbidity.

Alcohol: the deaths are still there.. Alcohol shows the same within/between divergence in both mortality models, and it is the sharpest case in the paper. Rising consumption within a region predicts *fewer* registered hepatitis deaths, which no account of the liver can explain (Roudot-Thoraval et al., 1997); regions that drink persistently more record more of them, which every account can.

Section 3.5 puts the coding explanation to the test, and in urban Russia the mechanism holds. Close the liver envelope and the negative alcohol effect is gone. The deaths are still there; only the label has moved. And it has not moved where one would expect. Adding alcoholic liver disease does not account for them—unspecified cirrhosis does. Physicians facing a liver death in a heavy-drinking region are not swapping one aetiology for another. They are declining to name one at all, which is exactly the kind of degradation in cause-of-death specificity documented for Russian mortality statistics (Danilova et al., 2016).

This also disposes of the obvious alternative. If drink were simply killing people before hepatitis could, total liver mortality would rise with consumption. It does not move. Deaths are being relabelled, not caused.

Rural mortality is another matter: the envelope does not close there, and we will not pretend to know why. Three categories evidently do not exhaust where a rural liver death can go.

Migration.. Migration behaves differently, positively associated with rural but not urban hepatitis mortality, inverting the usual pattern where urban migrant communities carry blood-borne infection (Jia et al., 2008). In the Russian context, rural inflows may disproportionately include individuals with pre-existing chronic hepatitis—particularly from high-prevalence Central Asian regions—who lack treatment access and face higher liver-related mortality, consistent with evidence on Central Asian labour migration sustaining HCV transmission (Tavoschi et al., 2019), and identifying rural receiving areas as an underrecognised mortality burden zone.

Narcotic seizures and urban hepatitis mortality.. The positive between-region term for urban narcotic seizures—a proxy for drug-market activity absent direct prevalence data (Averkiewa, 2014)—provides indirect evidence for injection drug use as a structural driver of hepatitis mortality, consistent with the established link between injection use, HCV transmission, and liver disease (Galea and Vlahov, 2002; Cloud et al., 2019). The absence of a within-region effect suggests this pathway operates through conditions stable within regions over time rather than year-to-year enforcement fluctuations.

Prison population: an inconclusive but conceptually important predictor..

The prison variable is insignificant in all three models, though large and positive in morbidity. This null should be read cautiously, not as evidence against the incarceration pathway: correctional facilities are documented amplifiers of blood-borne infection, with post-release reintegration a route from institutional to community spread (Awofeso, 2010; Moran et al., 2012). The available indicator captures stock rather than flow and ignores inter-regional transfers and release patterns, and attributing individual institutional exposure to regional aggregates invites ecological fallacy—limitations pointing to a need for finer institutional data rather than a refutation of the mechanism.

4.1. Strengths and limitations

The study is the first systematic spatial panel analysis of viral hepatitis morbidity and mortality across all 85 Russian federal subjects over 2012–2019 (680 region-years). It explicitly models spatial dependence and, through the Mundlak CRE specification, identifies both within- and between-region associations while retaining time-invariant predictors; the urban/rural split shows how the same factors operate differently across demographic and healthcare-access contexts.

Several limitations apply. First, all outcomes are registered cases and deaths, reflecting diagnostic and reporting capacity as well as true burden—most consequentially for the morbidity income coefficient, which likely captures detection. Non-differential under-registration in poorer regions would attenuate the disadvantage–hepatitis association, implying a true poverty gradient larger than observed, as documented for Brazilian TB surveillance (Harling and Castro, 2014). Second, federal subjects are large, internally

heterogeneous units: sub-regional clustering is not captured, and the modifiable areal unit problem means associations here may not hold at finer scales. Third, age-standardised rates could not be built from the administrative sources, so crude rates remain subject to demographic confounding only partially offset by the controls. Fourth, several theoretically relevant covariates—individual injection drug use, healthcare-associated infection, and direct-acting antiviral coverage—are unavailable regionally for the full period, risking residual confounding. Finally, the contiguity weight matrix may not capture transmission through mobility and institutional linkages that transcend administrative adjacency.

5. Conclusions

Registered infectious disease data describe two things at once: where disease occurs, and where a health system is equipped to find it. Using all 85 Russian federal subjects over 2012–2019 and two outcomes generated at different tiers of the health system, this paper has shown that the two can be separated with nothing beyond routine surveillance statistics. Household income predicts registered hepatitis morbidity strongly at both the within- and the between-region margin, yet is unrelated to either urban or rural hepatitis mortality—the signature of a detection gradient rather than of a burden gradient. Tuberculosis, alcohol and HIV reverse sign between the within- and the between-region margin, so that a fixed-effects estimator would report them not merely imprecisely but with the wrong sign; the Mundlak correlated random effects specification recovers both margins and makes the reversal itself interpretable. And although hepatitis is strongly clustered in

a stable Siberian and Far Eastern corridor, no residual spatial dependence survives conditioning on regional characteristics: at the scale of the federal subject, adjacency proxies for similarity of composition rather than for transmission across borders.

Two implications follow. For surveillance, regional rankings of registered morbidity should not be read as rankings of burden, and a region that registers few cases may be one that tests few people; the policy response to a low count is therefore not obviously fewer resources. For spatial analysis of administrative health data more generally, a null autoregressive parameter is an informative result rather than a failed specification, provided the compositional alternative has been given a fair chance to explain the clustering. What the design cannot deliver is a direct measure of screening intensity: regional data on testing volumes, on registrations disaggregated by acute and chronic presentation, or on direct-acting antiviral coverage would allow the detection interpretation advanced here to be tested rather than inferred, and remain the natural next step.

Data availability

All data analysed in this study are drawn from publicly available official sources: Form No. 12 and Form C-51 (CNIIOIZ, Ministry of Health), the Unified Interdepartmental Information and Statistical System (EMISS), the Federal Penitentiary Service (FSIN), and the Ministry of Internal Affairs. The assembled regional panel and the estimation code are available from the author on reasonable request.

Declaration of competing interest

The author declares no competing financial or personal interests.

Appendix A. Descriptive statistics

Appendix B. Spatial maps for hepatitis mortality

This appendix reports the exploratory spatial maps for urban and rural hepatitis mortality discussed in Section 3. The mapping conventions and the treatment of regions without contiguity neighbours follow those of Figures 2 and 3.

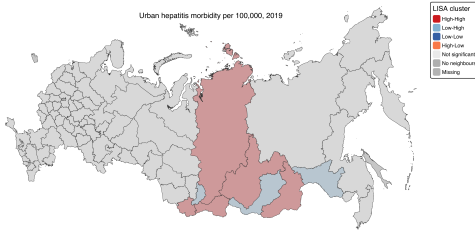


Figure B.4: LISA cluster map, urban hepatitis mortality per 100,000, 2019.

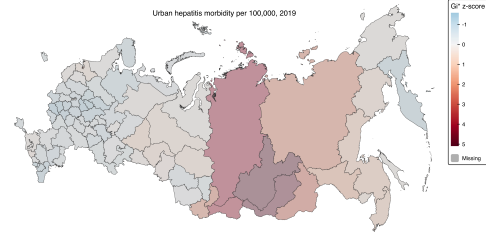


Figure B.5: G_i^* hotspot map, urban hepatitis mortality per 100,000, 2019.

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Table A.6: Descriptive statistics, 85 federal subjects, 2012–2019 ($N = 680$ region-years).

Variable	Mean	St. Dev.	Min	Max
<i>Panel A. Hepatitis outcomes (per 100,000)</i>				
Registered morbidity	661.92	486.39	50.70	3,873.80
Urban mortality	1.121	1.435	0.000	10.758
Rural mortality	0.779	1.215	0.000	10.107
<i>Panel B. Comorbidity and behavioural covariates</i>				
HIV incidence (per 100k)	49.10	38.98	0.00	239.80
Tuberculosis notifications (per 100k)	61.10	28.43	11.40	205.20
Alcohol consumption (l/capita)	6.387	2.328	0.000	13.222
Narcotic seizures, urban (per 100k)	2.887	3.434	0.000	23.469
Narcotic seizures, rural (per 100k)	2.491	5.539	0.000	65.692
<i>Panel C. Socioeconomic and demographic covariates</i>				
Household income (RUB/capita)	27,191.6	11,902.1	10,190.0	83,385.0
Log household income	10.141	0.353	9.229	11.331
Unemployment rate (%)	10.271	11.934	0.300	166.300
Net migration (per 10k)	−5.511	60.000	−223.000	439.000
<i>Panel D. Institutional covariate</i>				
Prison population (per capita)	0.009	0.006	0.000	0.032

Notes. Balanced panel of 85 federal subjects over 2012–2019. Narcotic-seizure variables are defined on 674 region-years. Monetary variables are in current roubles. The Mundlak between-region averages $\bar{X}_i$ are the region-specific time means of the corresponding within-region covariates and are omitted here for brevity.

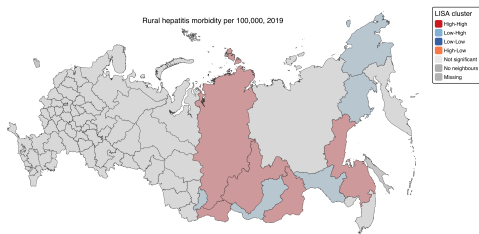


Figure B.6: LISA cluster map, rural hepatitis mortality per 100,000, 2019.

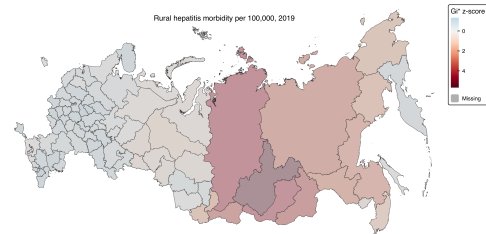


Figure B.7: G_i^* hotspot map, rural hepatitis mortality per 100,000, 2019.

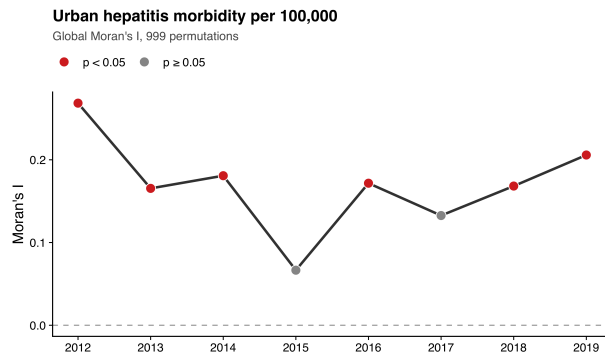


Figure B.8: Global Moran's I for urban hepatitis mortality per 100,000, Russian regions, 2012–2019. Red points indicate years significant at $p < 0.05$; grey points are non-significant.

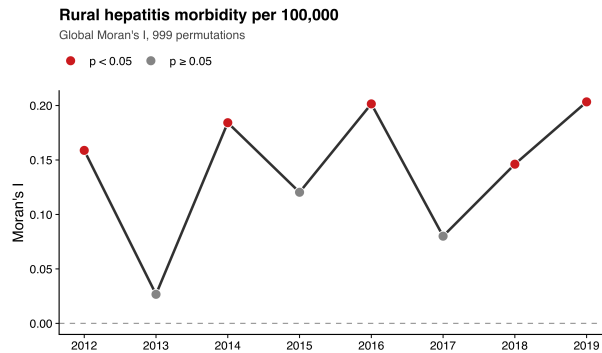


Figure B.9: Global Moran's I for rural hepatitis mortality per 100,000, Russian regions, 2012–2019. Red points indicate years significant at $p < 0.05$; grey points are non-significant.

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