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Research Article

The mathematical modelling of the dual system of tuberculosis control in the USSR: a mathematical analysis of the infectious process

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Abstract

The tuberculosis (TB) epidemic in the Soviet and post-Soviet space represents a unique case in global epidemiology, shaped by the long-term interaction between coercive state structures, distorted surveillance systems, and inherited scientific paradigms. This article develops a mathematical framework for analyzing the dual system of TB control in the USSR—one that simultaneously generated conditions for sustained transmission within carceral institutions while publicly promoting a narrative of eradication. The study integrates historical epidemiology, structural analysis of Soviet penal institutions, and a review of mathematical models of TB and HIV/AIDS to construct a dual-population model incorporating totalitarian fragmentation, differential visibility of disease, and the asymmetric relationship between civilian and incarcerated populations. The article critically assesses limitations of classical compartmental models when applied to the post-Soviet TB context, emphasizing the cumulative effects of unreliable surveillance data, ideological distortions in diagnostic systems, and the inability of phthiriasis paradigms to account for structural determinants of infection. Employing elements of the morphological concept of epidemic diffusion, the proposed model reconstructs key mechanisms underlying epidemic persistence, including reinfection loops, suppression of epidemiological knowledge, and the emergence of a stable pathological equilibrium sustained by political–institutional dynamics. Mathematical analysis demonstrates the existence of a totalitarian fragmentation threshold beyond which TB becomes self-reinforcing, even under conditions of partial policy intervention. The model further explains why conventional elimination strategies fail in settings where penal systems act as chronic infectious reservoirs and where epidemiological visibility is systematically reduced. The

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findings underscore the necessity of incorporating institutional structures, knowledge asymmetries, and political constraints into models of infectious disease in the post-Soviet region. This study provides a theoretical foundation for future empirical research and offers a methodological framework for analyzing epidemics in environments characterized by data scarcity, institutional opacity, and structural violence.

Keywords: tuberculosis; mathematical modeling; Soviet Union; post-Soviet states; dual system of TB control; carceral epidemiology; penal colonies; diffusion processes; morphological analysis; totalitarian fragmentation; epidemiological visibility; infectious dynamics.

Key Points

1. Necessity of a dual-population model. The tuberculosis epidemic in the USSR cannot be explained using classical models because two fundamentally different populations existed: the civilian population (monitored and controlled) and the prison population (largely hidden and poorly controlled). Their interaction creates a unique epidemiological system.
2. Prisons as a core source of infection. The penal system is not just a risk factor but a reservoir of infection. Overcrowding, poor nutrition, and inadequate treatment create ideal conditions for rapid transmission and the emergence of drug-resistant TB strains.
3. Political "blindness" and data distortion. The state system structurally ignores or underreports data from prisons, leading to distorted epidemiological statistics. This results in incorrect assessments of the epidemic and reduces the effectiveness of control measures.
4. Self-sustaining epidemic dynamics. Continuous movement between prisons and civilian society (through arrests and releases) creates a closed loop of transmission. This leads to a stable state where the epidemic persists even when control measures are in place.
5. Limitations of classical mathematical models. Standard epidemiological models fail because they do not include social, political, and institutional factors. Without incorporating elements such as the penal system, data reliability, and governance structures, it is impossible to accurately describe or predict the epidemic.

Introduction

Relevance of Research. The ongoing war in Ukraine has produced numerous humanitarian, political, and economic consequences, but one of its less examined dimensions concerns public health and the large-scale movement of populations from high-burden regions into Europe. Millions of refugees have crossed borders since 2014, fleeing areas in which epidemiological indicators for infectious diseases—particularly tuberculosis (TB)—exceed European averages by an order of magnitude. This creates a complex infectious interface between regions shaped by decades of post-Soviet health system deterioration and countries with comparatively low levels of infectious disease

transmission. In such a context, it becomes crucial to obtain corrected, analytically meaningful estimates of disease burden and to understand the mechanisms underlying these processes. The present article represents an attempt to conceptualize and model these dynamics, recognizing the limitations of available data and the unique structural challenges posed by the post-Soviet infectious landscape.

Object of Research. The object of this study is the long-term dynamics of tuberculosis indicators in the Soviet and post-Soviet spaces. These data are intrinsically problematic: underreported, inconsistent, influenced by administrative incentives, and embedded in a legacy of Soviet diagnostic and classificatory practices. This situation requires the development of methodological strategies for reconstructing infectious processes despite the unreliability of primary sources. Given the minimal likelihood that data quality will improve in the foreseeable future—due to conflict, institutional instability, and the collapse of public health infrastructures—there is a pressing need for research frameworks that permit analytical modeling under uncertainty.

Purpose and Research Tasks. The purpose of this study is to establish a theoretically grounded and empirically informed understanding of infectious dynamics related to tuberculosis in the post-Soviet region. To achieve this, the article pursues two primary tasks:

1. Critical analysis of the scholarly literature on mathematical modeling of infectious processes, with particular attention to modeling approaches applied to TB and HIV/AIDS. These two diseases, though fundamentally different biologically, share structural similarities within the post-Soviet context: long-term endemicity, profound social determinants, and a high degree of institutional distortion in reporting systems.

2. Detailed examination of the Soviet penal system as a major incubator of infectious diseases, particularly tuberculosis. The “correctional labor colonies” (ITUs) of the USSR constituted dense, poorly ventilated, overcrowded environments with minimal access to medical care—conditions that created ideal infectious “amplifiers.” Understanding the historical and contemporary role of carceral institutions is essential for reconstructing transmission dynamics that continue to shape regional epidemics today.

Methodology. The methodological foundation of this study is an initial version of a mathematical model tailored to the Soviet and post-Soviet infectious environment. This model is informed by what may be termed the “Soviet epistemology of disease”—a culturally specific framework that shaped diagnostic criteria, reporting practices, and conceptual interpretations of infection. Recognizing that Soviet medical statistics were often ideological rather than descriptive, the model integrates correction factors derived from structural inconsistencies, demographic discontinuities, and spatial anomalies across administrative territories.

Empirical Data. The empirical basis for the study consists of materials collected and analyzed by the Laboratory of Geomonitoring and Forecasting of Epidemic Processes, which operated in Kyiv between 2007 and 2013 under the Institute of Cartography (Committee for Cartography, Geodesy, and Aerial Surveying of the Cabinet of

Ministers of Ukraine). Although these datasets have been only partially published, they include detailed cartographic representations of TB incidence at multiple administrative levels of Ukraine. Examples include the series of regional maps by Tymoshenko et al. (2014–2020) and Tarkovsky et al. (2014) (Tymoshenko et al [2014-2020], Tarkovsky et al [2014]), which document spatial heterogeneities in TB burden across districts and provide valuable reference points for model calibration.

All available materials from post-Soviet states were utilized in this study, yet the author maintain a critical stance toward these sources in light of known issues of data reliability. Nonetheless, this body of information remains essential for establishing context and providing a necessary foundation for analysis. However, it must be emphasized that the most reliable data can only be obtained through direct field expeditions—specifically, by on-site inspections of AIDS centers and tuberculosis hospitals—which yield firsthand evidence unobtainable from secondary sources.

RESULTS

Mathematical Modeling of Infectious Processes Related to Tuberculosis: A Critical Review

Mathematical modeling of TB epidemics has developed over nearly three decades, beginning with foundational compartmental models that sought to describe the "intrinsic" dynamics of TB in human populations. One of the earliest and most influential works by Blower and colleagues used a deterministic framework to show that TB epidemics can rise and fall over time scales of many decades, even in the absence of treatment, emphasizing the slow, endemic nature of TB and suggesting that part of the historical decline in high-income countries reflected intrinsic epidemic behavior rather than solely chemotherapy or vaccination (Blower et al [1995]). Building on this, Porco et al. applied uncertainty and sensitivity analyses to similar models, demonstrating that a small number of key parameters – notably reactivation rates and transmission intensity – dominate long-term epidemic trajectories (Porco et al [1996]).

A parallel line of work has focused on the natural history of infection and disease. Vynnycky and Fine used an age-structured model calibrated to historical data from England and Wales to disentangle primary disease, reactivation, and reinfection, showing how age-dependent risks of progression strongly shape epidemic patterns (Vynnycky et al [1997]). More recently, Menzies et al. synthesized evidence on latent infection and progression risks, highlighting the wide uncertainty in parameters that underlie most TB transmission models (Menzies et al [2018]). Together, these studies show that many core quantities in TB modeling are only loosely constrained.

At the global and programmatic level, Dye and Williams and their collaborators used mathematical models to evaluate the impact and limitations of the WHO DOTS strategy and to explore prospects for TB control under various scenarios of case detection and treatment (Dye et al [1998], Dye et al. [2010]). These frameworks made clear that even optimally implemented DOTS would be unlikely to eliminate TB in high-burden settings, helping to shift policy thinking toward broader determinants and new tools. Cohen and Murray extended the modeling paradigm to multidrug-resistant TB

(MDR-TB), introducing multi-strain models that explored how the relative fitness of resistant strains influences the likelihood of MDR epidemics (Cohen et al [2004]).

As modeling matured, attention turned from simple homogeneous populations to more realistic structures. Hill et al. developed a model calibrated to US incidence trends in native-born and foreign-born populations, using it to assess the potential for TB elimination and the role of treatment of latent infection (Hill et al [2012]). Trauer et al. constructed a ten-compartment model incorporating BCG vaccination, waning protection, and reinfection to reproduce dynamics in highly endemic Asia-Pacific settings where HIV plays a limited role (Trauer et al [2014]). Dodd and colleagues recently proposed an age-structured model including HIV and antiretroviral therapy, calibrated to data from 12 African countries, using Bayesian methods to quantify uncertainty and estimate age-specific infection risks and the contribution of recent infection to incidence (Dodd et al [2023]).

Several reviews have synthesized the achievements and gaps in TB modeling. Zwerling et al. reviewed transmission models with an emphasis on diagnostics and novel therapies, arguing that models are most useful for comparing interventions and identifying key knowledge gaps rather than for precise prediction (Zwerling et al [2015]). Melsew and co-authors systematically examined how heterogeneous infectiousness is (or is not) captured in TB models, finding that most frameworks still rely on strong simplifying assumptions that underplay individual-level variability in transmission (Melsew et al [2020]). Fuller et al. have recently critiqued models of drug-resistant TB, highlighting how limited data and structural assumptions can bias projections for resistance control (Fuller et al [2024]).

Beyond classical ordinary differential equation (ODE) models, newer approaches include models tailored to informal settlements and townships (Pienaar et al [2010]), models incorporating environmental and household risk factors (Kendall et al [2015]), and agent-based or microsimulation frameworks that attempt to reflect contact networks and spatial structure more explicitly (Bui et al [2024]). Within-host and multiscale models have also been reviewed, particularly for their contributions to understanding treatment and latency (Kirschner et al [2017]).

Overall, the modeling literature on TB has succeeded in clarifying time scales, highlighting the importance of reactivation and reinfection, comparing intervention strategies, and quantifying uncertainty. At the same time, many authors emphasize that strong dependence on latent, poorly measured parameters, profound social and environmental determinants, and extreme heterogeneity of transmission limit the explanatory power of even sophisticated models (Zwerling et al [2015], Melsew et al [2020], Fuller et al [2024], Castillo-Chavez et al [2004], Okuonghae et al [2016], Tomczak et al [1998]). This tension between mathematical tractability and biosocial complexity is central to any critical assessment of TB epidemic modeling.

Why Mathematical Models Explain HIV but Struggle with Tuberculosis: a Critical Review

Mathematical models have become tools in infectious disease epidemiology, yet their impact has not been uniform across pathogens. For HIV, modeling has fundamentally reshaped scientific understanding and public health policy. In contrast, similar efforts in TB have produced more modest, largely descriptive insights. This asymmetry reflects deep structural differences between the infections rather than weaknesses in the modeling techniques themselves.

HIV fits remarkably well into the classical framework of compartmental and network-based models developed by Anderson, May and others. Transmission can be described as a function of relatively well-measured behavioral variables—numbers of partners, types of sexual acts, needle-sharing patterns—and biologically quantifiable parameters such as per-contact transmission probability and viral load–dependent infectiousness (Anderson et al [1991], Diekmann et al [2010], Garnett [2002]).

The relationship between antiretroviral therapy, viral suppression and reduced transmission is robust and monotonic, allowing models to predict the population-level impact of treatment-as-prevention long before trials confirmed it (Fraser et al [2007], Granich et al [2009], Cohen et al [2011], Eaton et al [2012]). These models underpinned the “test and treat” paradigm and informed ambitious targets such as the UNAIDS 90–90–90 goals (UNAIDS. 90–90–90 [2014]). In short, HIV spreads through discrete, countable events mediated by measurable behaviors and a single, time-varying biological state (viral load), which makes it highly amenable to mathematical abstraction.

Tuberculosis presents a fundamentally different challenge. TB is characterized by long and variable latency, with a large fraction of the global population harboring latent infection that may never progress to disease. Age-dependent risks of primary disease, reinfection, and late reactivation, as quantified by Vynnycky and Fine, make simple compartmental descriptions inherently unstable and sensitive to uncertain parameters (Vynnycky et al [1997]). Yet the empirical basis for these parameters remains weak, as systematic reviews of the natural history of untreated and latent TB demonstrate substantial uncertainty (Tiemersma et al [2011]).

Moreover, TB is deeply embedded in social and environmental contexts. Overcrowding, malnutrition, migration, incarceration, and poverty act as powerful determinants of both infection and disease progression (Lönnroth et al [2009]). These determinants are heterogeneous in space and time and cannot be easily collapsed into a single “effective contact rate” without losing critical structure. Models struggle to represent such biosocial complexity, and sensitivity analyses repeatedly show that predictions are highly dependent on poorly constrained social and programmatic parameters (Dowdy et al [2013], Melsew et al [2020]). Heterogeneity in infectiousness—where a minority of patients may drive transmission in poorly ventilated, high-risk environments—further undermines the assumption of homogeneous mixing that underlies most analytic frameworks (Kirschner et al [2017]).

At the same time, TB epidemics unfold slowly. Intervention effects may take 5–20 years to become visible, limiting opportunities for rapid empirical validation of model predictions. In contrast, HIV incidence responds more quickly to changes in prevention and treatment, enabling iterative refinement of models and policy in near-real time. For TB, even sophisticated multiscale and within-host models, though valuable for exploring latency and treatment, have not yet yielded decisive shifts in global strategy.

In summary, mathematical models have been extraordinarily successful for HIV because the infection is behaviorally mediated, biologically quantifiable and relatively fast-moving. TB, by contrast, is slow, socially entangled and driven by latent, hard-to-measure processes. As a result, TB models are most powerful as tools for scenario comparison and resource planning, but they rarely achieve the explanatory or predictive depth seen in HIV modeling.

Why Mathematical Modeling of Tuberculosis Is Especially Critical for the Post-Soviet Space

The question of whether TB can be effectively modeled has long generated debate, given the conceptual and empirical difficulties inherent in the disease. Yet despite these limitations, mathematical modeling remains indispensable—particularly in the post-Soviet space, where infectious, social, political, and data-quality factors converge to create an urgent need for analytical tools capable of reconstructing and forecasting infectious processes. In this context, the value of modeling lies not in perfect prediction but in enabling rational inference under conditions of uncertainty, especially where traditional surveillance systems are inadequate.

The first reason modeling is essential in the post-Soviet space is the exceptional intensity of TB transmission observed across the region. Incidence rates in many former Soviet republics exceed those of Western Europe by an order of magnitude. These persistently elevated burdens stem from structural determinants—poverty, deteriorating health systems, high rates of incarceration, widespread alcoholism, and the legacy of Soviet institutional arrangements—that shape exposure, infection, and disease progression. High-burden settings with long and complex epidemic histories cannot be adequately understood through descriptive statistics alone. Mathematical models provide the means to analyze how transmission dynamics unfold within such structurally unstable environments and to identify the latent parameters driving epidemic persistence.

A second reason is geopolitical. The post-Soviet region has been a generator of recurrent conflicts and large-scale population displacements, with direct implications for European public health. The ongoing war in Ukraine, which began in 2014 and continues today, has produced millions of refugees moving from areas where TB incidence and MDR-TB rates substantially exceed European averages. Migration flows from regions with a significant infectious burden create a spatial linkage between the post-Soviet epidemic and EU countries. Mathematical models are uniquely suited to evaluating how such population movements may reshape transmission networks, alter age and risk structures, and impose new pressures on health systems across borders.

In this sense, modeling TB in the post-Soviet region is not simply a regional concern but an instrument of European health security.

A third factor necessitating modeling is the pervasive unreliability of TB surveillance data across much of the post-Soviet space. Underreporting, inconsistent diagnostic criteria, fragmentation of health information systems, and the continued influence of what may be termed "Soviet epistemology" in disease classification all contribute to systematic distortions in epidemiological indicators. The cultural legacy of the Soviet medical system—where certain diseases, including TB, were ideologically stigmatized or administratively manipulated—continues to shape data quality today. Under such conditions, mathematical modeling provides a corrective framework: it enables the reconstruction of plausible epidemic curves, estimation of true incidence, and identification of spatial heterogeneities even when raw data are incomplete or biased.

One methodological strategy particularly applicable to this region is the "key-point method," widely used in geographical science. Here, well-studied territories serve as reference "keys" for inferring dynamics in poorly documented regions. When calibrated with high-quality local data from selected subpopulations—urban centers, prisons, migrant communities—mathematical models can extrapolate broader patterns across the larger post-Soviet landscape. Although imperfect, this approach yields insights that cannot be obtained by conventional surveillance tools alone and provides decision-makers with the best available estimates under constraints of uncertainty.

Mathematical modeling is further justified by the temporal depth of available historical information. The Soviet and post-Soviet TB epidemic spans more than a century, providing a large numerical basis for analysis. While interpretations of historical events may vary, the volume of infectious, demographic, and archival material is substantial. Learning to structure, parameterize, and critically interpret these data requires formal quantitative frameworks that can link historical processes to contemporary patterns.

Finally, modeling is becoming more powerful due to recent theoretical advances, such as the morphological concept of epidemics developed by Professor Dmitry Nikolaenko (Nikolaenko et al [2009], Nikolaenko [2009], Nikolaenko [2010], Nikolaenko [2011], Nikolaenko et al [2011]). This framework conceptualizes TB and HIV/AIDS epidemics as diffusion processes characterized by identifiable spatial–temporal morphologies. Incorporating these ideas into mathematical models allows for more accurate representation of epidemic propagation fronts, local amplifiers of transmission, and structural constraints inherent to the post-Soviet context. Diffusion-based approaches permit systematic exploration of alternative epidemic trajectories, enabling researchers to investigate how environmental, political, and social shocks modulate transmission dynamics.

In sum, mathematical modeling in the Soviet and post-Soviet TB context plays a necessarily auxiliary but indispensable role. Researchers are acutely aware of the imperfections of available data, yet abstaining from modeling is not a viable alternative. Models provide the only coherent framework through which heterogeneous historical

information, uncertain surveillance data, and complex socio-political processes can be integrated into an interpretable and actionable understanding of TB dynamics. Even if models cannot fully overcome the limitations of the underlying data, they offer structured reasoning where none would otherwise be possible.

THE DUAL-POPULATION EPIDEMIOLOGICAL MODEL

Why tuberculosis in the USSR requires a dual-population model

Standard epidemiological models assume uniform surveillance, uniform policy response, and symmetric visibility across all segments of society. In the Soviet Union, none of these conditions held. Two populations existed simultaneously:

1. Civilian population

- monitored,
- counted,
- studied,
- publicly discussed,
- used to measure success of the socialist healthcare project.

2. Prison population

- unmonitored,
- uncounted,
- scientifically inaccessible,
- ideologically inconvenient,
- excluded from official statistics.

Yet these two populations were infectious coupled through:

- arrest flow: civilians → prisons
- release flow: prisons → civilians
- family visits, informal contacts, prison guards

Thus, prisons acted as infectious sources, while civilians acted as infectious sinks. It is a type of "fireplace" that maintains a continuous flame. The Soviet state has consistently maintained a controlled environment for the cultivation of tuberculosis and various other infectious diseases.

Central asymmetries

Epidemiological factor	Civilian population	Prison population
Crowding	Low/Moderate	Extreme
Nutrition	Moderate	Poor
Medical access	Good	Minimal
Treatment continuity	High	Rare

Surveillance	Present	Absent
TB intensity	Medium	Very High
Visibility	Full	Zero
Policy responsiveness	Medium	Near Zero
Treatment	Compulsory, forced	Refusal of treatment
Deliberately exposing oneself to tuberculosis	Entirely unprecedented	This is a standard procedure

This asymmetry forces the model to treat the penal subsystem as a structurally different compartment, not a small correction.

Transmission flows

1. Flow from civilians into prisons

Healthy civilians can acquire TB in prison. Infected civilians can become infectious faster due to deprivation.

This is the term:

$$\frac{dE_p}{dt} = r_p E_p \left(1 - \frac{E_p}{C_p}\right) + aE_c - \delta_p E_p - \alpha_p (1 - B) E_p$$

Formula 1.

2. Flow from prisons back to civilians

Released prisoners often carry:

- untreated TB
- partially treated TB
- MDR-TB
- latent infections

This appears as:

$$\frac{dE_c}{dt} = r_c E_c \left(1 - \frac{E_c}{C_c}\right) + rE_p - \delta_c E_c - \alpha_c (1 - B) E_c$$

Formula 2.

Political asymmetry embedded in the model

Political visibility affects epidemiology.

- Civilian TB triggers policy responses.
- Prison TB triggers political denial.

Thus, political structure alters the dynamics:

Totalitarian fragmentation reduces the effective strength of health policy

$$\frac{dP_p}{dt} = p_{1p}K_p - p_{2p}BP_p$$

Formula 6

$$\frac{dP_c}{dt} = p_{1c}K_c - p_{2c}BP_c$$

Formula 7

Totalitarian fragmentation inhibits knowledge formation

$$\frac{dK_p}{dt} = s_p(1 - \sigma B)E_p - d_pK_p$$

Formula 4

$$\frac{dK_c}{dt} = s_c(1 - \kappa B)E_c - d_cK_c$$

Formula 5

Totalitarian fragmentation grows when epidemics worsen

$$\frac{dB}{dt} = \lambda_pE_p + \lambda_cE_c + \mu - \gamma B$$

Formula 3

This makes the penal epidemic *self-reinforcing*.

Emergence of the pathological equilibrium

If:

- penal TB is high,
- totalitarian fragmentation increases,
- knowledge declines,
- policy collapses,
- reinfection flows persist,

then the system converges to:

- high E_p^*,
- moderate E_c^*,
- high B^*,
- persistent reinfection.

This is the historical Soviet pattern.

MATHEMATICAL FORMULATION

This section formalizes all mechanisms introduced above. We present the full dynamic system:

- two epidemic compartments,
- two knowledge compartments,
- two policy compartments,
- one blindness variable.

Then we reduce the system for analytical tractability.

Full epidemic system

1. Prison TB equation

$$\frac{dE_p}{dt} = r_p E_p \left(1 - \frac{E_p}{C_p}\right) + aE_c - \delta_p E_p - \alpha_p (1 - B) E_p$$

Formula 1

This includes:

- logistic growth
- civilian inflow
- natural removal
- policy removal scaled by visibility (1–B)

2. Civilian TB equation

$$\frac{dE_c}{dt} = r_c E_c \left(1 - \frac{E_c}{C_c}\right) + rE_p - \delta_c E_c - \alpha_c (1 - B) E_c$$

Formula 2

This includes:

- logistic growth
- reinfection from prisons
- civilian policy effort scaled by visibility

Totalitarian fragmentation dynamics

Totalitarian fragmentation increases when:

- prison epidemic increases,
- civilian epidemic increases,
- ideological pressure increases.

Totalitarian fragmentation decreases through:

- knowledge accumulation,
- information leakage,
- elite turnover.

$$\frac{dB}{dt} = \lambda_p E_p + \lambda_c E_c + \mu - \gamma B$$

Formula 3

Knowledge dynamics

Prison knowledge

$$\frac{dK_p}{dt} = s_p (1 - \sigma B) E_p - d_p K_p$$

Formula 4

Civilian knowledge

$$\frac{dK_c}{dt} = s_c(1 - \kappa B)E_c - d_cK_c$$

Formula 5

Totalitarian fragmentation suppresses knowledge by coefficients σ and κ.

Policy dynamics

Prison policy

$$\frac{dP_p}{dt} = p_{1p}K_p - p_{2p}BP_p$$

Formula 6.

Civilian policy

$$\frac{dP_c}{dt} = p_{1c}K_c - p_{2c}BP_c$$

Formula 7.

Totalitarian fragmentation weakens policy directly.

Reduced model

For formal analysis, we reduce the system:

- combine knowledge + policy into effective removal rates,
- maintain totalitarian fragmentation as explicit variable,
- keep dual epidemic structure.

$$\frac{dE_p}{dt} = r_pE_p\left(1 - \frac{E_p}{C_p}\right) + aE_c - (\delta_p + \alpha_p(1 - B))E_p$$

Formula 8.

$$\frac{dE_c}{dt} = r_cE_c\left(1 - \frac{E_c}{C_c}\right) + rE_p - (\delta_c + \alpha_c(1 - B))E_c$$

Formula 9.

$$\frac{dB}{dt} = \lambda_pE_p + \lambda_cE_c + \mu - \gamma B$$

Formula 10.

This reduced 3-variable system is sufficient to capture:

- epidemic persistence
- totalitarian fragmentation threshold
- pathological equilibrium
- hysteresis

Jacobian analysis

The Jacobian matrix of the reduced epidemic subsystem is:

$$J(B) = \begin{pmatrix} r_p - \delta_p - \alpha_p(1-B) & a \\ r & r_c - \delta_c - \alpha_c(1-B) \end{pmatrix}$$

Formula 11.

From this, we derive:

- Trace(J): $\rightarrow \mathrm{Tr}(J) = r_p - \delta_p - \alpha_p(1-B) + r_c - \delta_c - \alpha_c(1-B)$ Formula 12.
- Det(J): $\rightarrow \det(J) = (r_p - \delta_p - \alpha_p(1-B))\,(r_c - \delta_c - \alpha_c(1-B)) - ar$ Formula 13.

The dominant eigenvalue:

$$\lambda_{\max}(B) = \frac{\mathrm{Tr}(J)}{2} + \sqrt{\left(\frac{\mathrm{Tr}(J)}{2}\right)^2 - \det(J)}$$

Formula 14.

is crucial for stability.

Blindness threshold

The totalitarian fragmentation threshold is defined by:

$$\lambda_{\max}(B_{\mathrm{crit}}) = 0$$

Formula 15.

If:

- $\lambda_{\max}(B_{\mathrm{crit}}) < 0 \rightarrow$ epidemic dies out
- $\lambda_{\max}(B_{\mathrm{crit}}) > 0 \rightarrow$ epidemic persists

Stability conditions:

$$\lambda_{\max}(B) < 0$$

Formula 16.

$$\lambda_{\max}(B) > 0$$

Formula 17.

Equilibrium structure

Totalitarian fragmentation at equilibrium:

$$B^* = \frac{\lambda_p E_p^* + \lambda_c E_c^* + \mu}{\gamma}$$

Formula 18.

Epidemic equilibrium conditions:

$$0 = r_p E_p^* \left(1 - \frac{E_p^*}{C_p}\right) + a E_c^* - (\delta_p + \alpha_p(1-B^*))E_p^*$$

Formula 19.

$$0 = r_c E_c^* \left(1 - \frac{E_c^*}{C_c}\right) + r E_p^* - (\delta_c + \alpha_c(1 - B^*))E_c^*$$

Formula 20.

The penal equilibrium must satisfy:

- $E_p^* > 0$
- reinfection from prisons remains active
- totalitarian fragmentation stays high

The reinfection loop:

$$E_c \longrightarrow E_p \longrightarrow E_c$$

Formula 21.

prevents collapse of the epidemic.

FORMAL PROPERTIES OF THE SYSTEM

1. Positivity and invariance

$$\rightarrow [\ E_p(0),\ E_c(0),\ B(0) \geq 0 \quad \Longrightarrow \quad E_p(t),\ E_c(t),\ B(t) \geq 0\ ]$$

Formula 22

through

$$[\ \left.\frac{dB}{dt}\right|_{B=0} = \lambda_p E_p + \lambda_c E_c + \mu > 0\ ]$$

Formula 25

show that:

- the system remains biologically meaningful,
- variables never become negative.

2. Existence of equilibria

Using Brouwer:

$$F(E_p, E_c, B) = \begin{pmatrix} \dot{E}_p \\ \dot{E}_c \\ \dot{B} \end{pmatrix}$$

$$F(\partial\Omega_M) \text{ points inward}$$

$$\exists (E_p^*, E_c^*, B^*) \in \Omega_M : F(E_p^*, E_c^*, B^*) = 0$$

Formula 26–28.

there must exist at least one equilibrium in the positive orthant.

3. Stability of the disease-free equilibrium

The system is disease-free only when:

- totalitarian fragmentation is low,
- removal rates exceed transmission rates.

Linearization:

$$DF(E_p^*, E_c^*, B^*)x = J(B^*)\,x$$

Formula 29.

$$\chi(\lambda) = \lambda^2 - \mathrm{Tr}(J)\,\lambda + \det(J)$$

Formula 30.

Stability criterion:

$$\mathrm{Tr}(J) < 0, \qquad \det(J) > 0$$

Formula 31.

4. Totalitarian fragmentation threshold

The critical totalitarian fragmentation value:

$$\lambda_{\max}(B_{\mathrm{crit}}) = 0$$

Formula 32.

marks the transition from:

- controlled epidemics

→ to

- persistent epidemics.

5. Conditions for pathological equilibrium

If:

- λ_p large,
- γ small,
- α_p small,
- reinfection flows strong,

then:

$$B^* > B_{\mathrm{crit}}$$

Formula 33.

and the system enters a stable endemic equilibrium:

$$(E_p^*, E_c^*, B^*) \in \Omega, \quad E_p^* > 0,\ E_c^* > 0$$

Formula 35.

Stability inequality:

$$\lambda_{\max}(B^*) > 0 \quad \Longrightarrow \quad \text{endemic equilibrium stable}$$

Formula 36.

Thus the Soviet TB regime is mathematically predictable.

APPENDIX A: FORMAL PROOFS AND MATHEMATICAL FOUNDATIONS

This appendix provides the formal mathematical treatment of the dual-population model with totalitarian blindness. We work with the reduced 3-equation system introduced in the main text:

$$\frac{dE_p}{dt} = r_p E_p \left(1 - \frac{E_p}{C_p}\right) + aE_c - (\delta_p + \alpha_p(1 - B))E_p$$

Formula 8.

$$\frac{dE_c}{dt} = r_c E_c \left(1 - \frac{E_c}{C_c}\right) + rE_p - (\delta_c + \alpha_c(1 - B))E_c$$

Formula 9.

$$\frac{dB}{dt} = \lambda_p E_p + \lambda_c E_c + \mu - \gamma B$$

Formula 10.

These are reproduced below for readability:

- Prison epidemic equation
- Civilian epidemic equation
- Totalitarian fragmentation dynamics equation

Definitions and notation follow the main article.

A.1. Positivity and Forward Invariance

Theorem A.1 (Positivity).

If

$$E_p(0) \geq 0, E_c(0) \geq 0, B(0) \geq 0,$$

then

$$E_p(t),\ E_c(t),\ B(t) \geq 0 \forall t \geq 0.$$

Proof.

1. At the boundary $E_p = 0$:

$$\frac{dE_p}{dt} \mid_{E_p=0} = aE_c \geq 0,$$

which implies trajectories cannot cross into $E_p < 0$.

$$\left.\frac{dE_p}{dt}\right|_{E_p=0} = aE_c \geq 0$$

Formula 23.

2. At the boundary $E_c = 0$:

$$\frac{dE_c}{dt} \mid_{E_c=0} = rE_p \geq 0.$$

Formula 24.

3. At the boundary $B = 0$:

$$\frac{dB}{dt}|_{B=0} = \lambda_p E_p + \lambda_c E_c + \mu > 0.$$

Formula 25.

Thus the vector field always points inward on the boundary of the positive orthant.
Therefore the domain

$$\Omega = \{(E_p, E_c, B) \mid E_p, E_c, B \geq 0\}$$

is forward-invariant. ■

A.2. Existence of Equilibria

We rewrite the reduced system compactly as:

$$F(E_p, E_c, B) = \begin{pmatrix} \dot{E}_p \\ \dot{E}_c \\ \dot{B} \end{pmatrix}$$

Formula 26.

Let Ω be a sufficiently large closed box in $\mathbb{R}^3_+$. The vector field satisfies:

$$F(\partial\Omega_M) \text{ points inward}$$

Formula 27.

Theorem A.2.

The system possesses at least one equilibrium in Ω.

Proof.

- The vector field is continuous in Ω.
- Logistic saturation ensures boundedness.
- Totalitarian fragmentation dynamics contain a linear decay term −γB.
- The vector field points inward on ∂Ω.

By **Brouwer's Fixed Point Theorem**:

$$\exists (E_p^*, E_c^*, B^*) \in \Omega_M : F(E_p^*, E_c^*, B^*) = 0$$

Formula 28.

Thus an equilibrium exists. ■

A.3. Characterization of the Disease-Free Equilibrium

The disease-free equilibrium is:

$$(E_p, E_c, B) = (0,\ 0,\ \frac{\mu}{\gamma}).$$

Linearizing the epidemic subsystem gives the Jacobian:

$$J(B) = \begin{pmatrix} r_p - \delta_p - \alpha_p(1-B) & a \\ r & r_c - \delta_c - \alpha_c(1-B) \end{pmatrix}$$

Formula 11.

Associated objects:

- Trace(J): → $\mathrm{Tr}(J) = r_p - \delta_p - \alpha_p(1-B) + r_c - \delta_c - \alpha_c(1-B)$ Formula 12.
- Det(J): → $\det(J) = (r_p - \delta_p - \alpha_p(1-B))\,(r_c - \delta_c - \alpha_c(1-B)) - ar$ Formula 13.

The dominant eigenvalue:

$$\lambda_{\max}(B) = \frac{\mathrm{Tr}(J)}{2} + \sqrt{\left(\frac{\mathrm{Tr}(J)}{2}\right)^2 - \det(J)}$$

Formula 14.

governs stability.

Theorem A.3.

The disease-free equilibrium is locally asymptotically stable if and only if:

$$\lambda_{\max}(B) < 0$$

Formula 16.

and unstable if:

$$\lambda_{\max}(B) > 0$$

Formula 17.

Proof.

- The linearized system has characteristic polynomial:

$$\chi(\lambda) = \lambda^2 - \mathrm{Tr}(J)\,\lambda + \det(J)$$

Formula 30.

  - The equilibrium is stable if both eigenvalues have negative real parts.
  - This requires:

$$\mathrm{Tr}(J) < 0, \qquad \det(J) > 0$$

Formula 31.

But since parameters enter monotonically through totalitarian fragmentation, the sign of $\lambda_{\max}(B)$ alone determines stability (Perron–Frobenius).

Thus the stated conditions follow. ■

A.4. Totalitarian fragmentation Threshold

Define the totalitarian fragmentation threshold B_{crit} by:

$$\lambda_{\max}(B_{\mathrm{crit}}) = 0$$

Formula 15.

Proposition A.4.

The disease-free equilibrium is stable for $B < B_{\text{crit}}$ and unstable for $B > B_{\text{crit}}$.

Proof.

- Eigenvalues of a cooperative matrix increase in response to increases in its entries.
- Increasing B reduces effective removal rates.
- This increases the diagonal of J(B), which increases the spectral radius.
- By continuity of the dominant eigenvalue, the equation:

$$\lambda_{\max}(B_{\text{crit}}) = 0$$

Formula 32.

has a unique solution. ■

A.5. Existence of a Pathological Endemic Equilibrium

We show that high totalitarian fragmentation forces the system into a persistent epidemic.

Theorem A.5 (Existence of pathological equilibrium).

Assume:

- λ_p large (prison epidemic strongly increases totalitarian fragmentation),
- γ small (totalitarian fragmentation decays slowly),
- α_p small (prison policy weak),
- a and r strictly positive (bidirectional flows).

Then the system admits an equilibrium:

$$(E_p^*, E_c^*, B^*) \in \Omega, \quad E_p^* > 0,\ E_c^* > 0$$

Formula 35.

with:

$$B^* > B_{\text{crit}}$$

Formula 33.

(i.e., totalitarian fragmentation at equilibrium exceeds the critical threshold).

Proof.

1. If $B^* > B_{\text{crit}}$, the disease-free equilibrium is unstable.
2. By A.2, at least one equilibrium exists.
3. Since the disease-free equilibrium is unstable, the remaining equilibrium must satisfy:

- $E_p^* > 0$
- $E_c^* > 0$

4. Totalitarian fragmentation equilibrium condition:

$$B^* = \frac{\lambda_p E_p^* + \lambda_c E_c^* + \mu}{\gamma}$$

Formula 18.

Since λ_p dominates, small E_p produces large B*.

5. Therefore B*>B_crit.

Thus a positive endemic equilibrium necessarily exists. ■

A.6. Stability of the Pathological Equilibrium

Theorem A.6.

The pathological equilibrium described above is locally asymptotically stable.

Proof (sketch).

- For large totalitarian fragmentation, policy terms collapse, producing nearly unregulated epidemic growth.
- Logistic terms prevent divergence.
- Totalitarian fragmentation grows with epidemic intensity, reinforcing high B.
- Thus eigenvalues at the equilibrium satisfy:

$$\lambda_{\max}(B^*) > 0 \quad \Longrightarrow \quad \text{endemic equilibrium stable}$$

Formula 36.

Hence the equilibrium is stable. ■

A.7. Hysteresis and Non-Ergodicity

Theorem A.7 (Hysteresis).

If

$$B(t_0) > B_{\text{crit}},$$

then even if B is later reduced to near Bcrit, epidemic levels E_p and E_c may not return to zero.

Proof.

- High B induces high E.
- High E regenerates high B through:

$$\frac{dB}{dt} = \lambda_p E_p + \lambda_c E_c + \mu - \gamma B$$

Formula 10.

- The system enters a loop:

$$B(t_0) > B_{\text{crit}} \quad \Longrightarrow \quad E_p(t), E_c(t) \not\to 0$$

Formula 34.

Thus, the system is **path-dependent**, not ergodic.
Simply reducing totalitarian fragmentation is insufficient once the epidemic reservoir has been formed. ■

A.8. Summary

Appendix A proves that:

1. The system remains in the biologically meaningful space.
2. An equilibrium always exists.
3. Stability of the disease-free equilibrium is controlled by totalitarian fragmentation.
4. Above the totalitarian fragmentation threshold, the system converges to a pathological equilibrium.
5. This equilibrium is stable, persistent, and self-reinforcing.
6. Hysteresis explains the post-Soviet TB and MDR-TB explosion.

Conclusions

1. Mathematical modeling of infectious processes in tuberculosis serves a function that is not limited to strict scientific explanation, and even less to the precise forecasting of the epidemic process. Its primary role lies in the correction and reconstruction of existing data arrays. These datasets are persistently problematic, and mathematical modeling provides one of the few mechanisms through which at least part of these deficiencies can be addressed.

2. Depending on the socio-cultural environment and the specific characteristics of a given state, the functions of mathematical modeling related to tuberculosis infectious processes will differ substantially. The quality of epidemiological data varies dramatically across the world. There is, and cannot be, any universal framework applicable to all contexts.

3. Introducing a theoretical framework for this dual system of tuberculosis control—one involving A. the cultivation of tuberculosis and B. the heroic fight against tuberculosis—provides substantial benefits for mathematical modeling of the infectious process. It allows for the development of far more realistic representations of what has transpired over more than a century across the Russian Empire, the USSR, and the post-Soviet states.

4. In mathematical modeling of the tuberculosis infectious process, and in the many failures associated with it, an exceptionally important role is played by the initial conceptualization of what tuberculosis is and how the infectious process develops. It can be stated that the theoretical foundations of tuberculosis epidemiology are fundamentally flawed. The issue lies not only— and perhaps not even primarily— in the poor quality of empirical baseline data. The deeper problem is that the dominant understanding of the epidemic is shaped almost exclusively by the perspective of phthisiatricians, whose conceptual framework captures only a limited segment of the infectious process. These dominant representations categorically fail to reflect reality.

This may be one of the principal reasons for the persistent failures in mathematical modeling of the tuberculosis epidemic.

5. Our approach is based on the morphological concept of the HIV/AIDS and tuberculosis pandemic developed by Dmitry Nikolaenko. This concept was first articulated around 2005. It draws not only on medical knowledge about infectious diseases, but— fundamentally— on spatio-temporal analysis and long-term trends in the structural transformation of population morbidity. The terms "morphology of the spatio-temporal process" and "diffusion process" are critically important here. This constitutes a fundamentally new explanatory approach. As a result, mathematical modeling is grounded in a far more coherent foundation than the traditional phthisiatric perspective and the endless references to poverty, alcoholism, and other reductive explanatory tropes.

6. Recognizing that the problem lies not only in low-quality data but also in the inadequacy of the theoretical framework underpinning the understanding of the infectious process is highly significant. Specialists in mathematical modeling often suffer from criticism by physicians and from their categorical prescriptions. Modelers are treated merely as "assistants"— at least this has been the case in Ukraine. Throughout our work, the staff of the Laboratory for Geomonitoring and Forecasting of Epidemic Processes were repeatedly told that "your role is to help us." No criticism and no theory were welcome. Thus, the failure of tuberculosis modeling is largely attributable to the conservatism of the phthisiatric community and its unwillingness to hear well-founded critique or accept theoretical extensions. Mathematical modeling of this infectious process is not a "practical aid" to phthisiatry. It is an attempt to explain the unfolding infectious process from a new scientific perspective.

7. It is essential that work on mathematical modeling of the infectious process remain independent from the phthisiatric expert community. A long experience of attempted collaboration demonstrated that genuine cooperation does not materialize. Numerous constraints are imposed on theoretical analysis and modeling. Modeling is viewed solely as an auxiliary tool for practical phthisiatric work. Nothing else is considered necessary by physicians. However, phthisiatrists are not the only experts concerned with this diffusion process.

Conflict of Interest

The authors declare no conflict of interest.

Acknowledgments

This article was written under difficult circumstances. There is a war going on in Ukraine. People talk about it a lot, yet very few truly understand what it looks like in reality. Why so few understand? Probably because many simply choose not to understand — it is more convenient that way, including for the scientific community.

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A new scientific journal is now being created. I am confident that Pollution and Diseases will become an important event in the scientific world. It is a journal for those scientists who cannot think only about their citation metrics or financial stability. There are many long-standing problems in the fields of pollution and disease that urgently need a fresh scientific perspective.

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I wish you great success!

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