



SEIR-LT HYBRID ASYNCHRONOUS CELLULAR AUTOMATA MODEL FOR INFORMATION DIFFUSION IN SOCIAL NETWORKS

A hybrid asynchronous cellular automata model is proposed for simulating information diffusion processes in social networks, combining the functionality of classical compartmental structures (Susceptible-Exposed-Infected-Recovered) with linear threshold theory. An individualized mechanism for updating user states is implemented to more accurately capture temporal heterogeneity: each network participant is assigned unique activity intervals instead of simultaneous state transitions across the entire population. It is demonstrated that such an asynchronous modeling approach provides a more detailed representation of real human behavior, as individuals respond to external stimuli with varying speeds and levels of attention. A generalized linear threshold (GLT) methodology is introduced to determine the probability of a user transitioning to an active state – that is, the moment when social influence from the surrounding network exceeds the individual resistance threshold, prompting information adoption. The development patterns of a heterogeneous population are characterized, with specific identification of influential and resistant classes of network participants. It is established that abandoning global synchronization of users completely eliminates unrealistic wave-like artifacts inherent in traditional computational approaches. The functionality of the proposed asynchronous cellular automata architecture is tested using Monte Carlo methodology on a two-dimensional lattice. The impact of temporal heterogeneity on the average cascade size is evaluated, revealing an increase from 5.93 % under full synchronization to 6.17 % in localized heterogeneous configurations. A significant increase in the overall probability of large-scale information diffusion is observed: the proposed mechanism achieves successful propagation in 96 out of 100 macroscopic iterations, compared to only 88 % under conventional constraints. A natural smoothing of process intensity is recorded, confirmed by a reduction in peak prevalence (the maximum proportion of the population in the infected state actively disseminating information) from 0.99 % to 0.92 %, along with a more efficient redistribution of load over a slightly extended sequence of simulation stages. The influence of model parameters is analyzed, and a phase transition of the baseline probability is identified, with a critical point near 0.25. A fundamental asymmetry in information spread within the network is revealed: initiating large-scale cascades using a small number of influential users is significantly easier than suppressing them by increasing the number of resistant participants. The developed asynchronous cellular automata model is proposed as a tool for forecasting and testing strategies aimed at mitigating the spread of undesirable content.

Keywords: Generalized Linear Threshold; misinformation containment; epidemiological compartments; social contagion; temporal heterogeneity.

Introduction / Вступ

The study of information diffusion in social networks has become increasingly important in the digital age, where understanding how ideas, behaviors, and information spread through populations has significant implications for marketing, public health, and social policy. Traditional approaches to modeling these processes have drawn from two main theoretical frameworks: epidemiological models that treat information spread as analogous to disease transmission [8], and social influence models that emphasize threshold-based adoption decisions [5].

Cellular automata (CA) provide an ideal computational framework for studying these processes, as they naturally

capture the local interaction patterns that characterize social influence while enabling the emergence of complex global dynamics from simple local rules [15]. The history of cellular automata dates back to the 1940, with significant theoretical developments establishing CA as a mature methodology for studying complex systems. Previous research has demonstrated the utility of CA-based approaches for modeling various diffusion phenomena, from disease spread to opinion dynamics.

However, existing models often suffer from multiple oversimplifications. Basic SIR (Susceptible-Infected-Recovered) models assume homogeneous populations and instantaneous state transitions, while simple threshold mo-

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Грабовенська Ірина Михайлівна, студент, кафедра програмного забезпечення.

Email: iryna.hrabovenska.pz.2023@lpnu.ua; <https://orcid.org/0009-0007-7358-0818>

Рабійчук Ігор Олександрович, асистент, кафедра програмного забезпечення.

Email: ihor.o.rabiichuk@lpnu.ua; <https://orcid.org/0009-0002-0881-0406>

Фечан Андрій Васильович, д-р техн. наук, професор, завідувач кафедри електронних засобів інформаційно-комп'ютерних технологій. Email: andrii.v.fechann@lpnu.ua; <https://orcid.org/0000-0001-9970-5497>

Яценко Роман Олексійович, асистент, кафедра програмного забезпечення.

Email: roman.o.yatsenko@lpnu.ua; <https://orcid.org/0009-0009-4662-7873>

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dels neglect the probabilistic nature of influence. Perhaps most critically, the vast majority of cellular automata models employ synchronous updates, where all cells transition simultaneously based on the previous global state. This assumption produces artificial wave-like propagation patterns and fails to capture the temporal heterogeneity inherent in real social systems, where individuals process and respond to information at vastly different rates [4].

Real-world information diffusion exhibits characteristics that demand more sophisticated modeling [2, 7, 9, 14]:

- Individuals may be exposed to information multiple times before adopting it (latency).
- Adoption decisions involve both threshold-based considerations and stochastic elements.
- Response times vary dramatically across individuals based on personality, context, and attention.
- Information processing and decision-making are inherently asynchronous processes.

Object of research – process of information diffusion in social networks, characterized by complex dynamics of adoption, resistance, and temporal heterogeneity among network participants.

Subject of research – mathematical modeling and computational simulation of information spread using hybrid cellular automata that combine epidemiological compartmental models with social influence threshold mechanisms and asynchronous update dynamics.

The purpose of the research – develop, implement, and analyze a novel SEIR-LT-Async (Susceptible-Exposed-Infected-Recovered with Linear Threshold and Asynchronous Updates) cellular automata model that more realistically captures the temporal heterogeneity of information diffusion in social networks compared to traditional synchronous approaches.

To achieve this *purpose*, the following main *research objectives are identified*:

- to analyze existing approaches to modeling information diffusion, identifying limitations of synchronous cellular automata and classical epidemiological models, which will allow for establishing a concrete theoretical baseline for introducing an asynchronous paradigm;
- to develop a mathematical formulation of the hybrid SEIR-LT model incorporating the Generalized Linear Threshold (GLT) mechanism for activation probability, which will enable the precise calculation of individual state transitions based on complex social reinforcement;
- to design and implement an asynchronous update mechanism with individual update intervals that captures temporal heterogeneity in agent behavior, which will eliminate artificial synchronous waves and closely mimic realistic variations in human response times;
- to integrate dynamic parameter evolution (resistance decay, influence growth) reflecting realistic social learning processes, which will provide a dynamic framework where agents' susceptibility adapts based on accumulated exposure;
- to conduct comprehensive Monte Carlo simulations comparing synchronous and asynchronous update dynamics, which will statistically quantify the impact of temporal heterogeneity on overall cascade size and propagation probability;
- to perform parameter sensitivity analysis identifying critical thresholds and regime transitions, which will pinpoint the key leverage points, such as the minimum transmission probability and the required share of high-impact users;
- to evaluate the model's ability to reproduce qualitative features of real-world information diffusion phenomena, which will confirm its viability as a predictive tool for analyzing misinformation containment strategies.

This paper addresses these objectives by proposing the SEIR-LT-Async hybrid model, which integrates the epidemiological SEIR framework with the Linear Threshold model from social influence theory, enhanced with a novel asynchronous update mechanism.

Analysis of recent research and publications. In the study [8], the authors developed the mathematical theory of rumor spreading in complex networks, establishing the foundational connection between epidemiological dynamics and information propagation. They demonstrated how SIR-type compartmental models can effectively describe the spread of information through social structures, providing a theoretical baseline that justified the broad applicability of disease transmission frameworks to sociology and information science.

In the comprehensive work [4], the authors explored the fundamental differences between synchronous and asynchronous cellular automata, serving as a primary resource for understanding temporal variations. They presented a detailed taxonomy of stochastic asynchronous updating schemes – such as fully asynchronous, α -asynchronous, and random order – and demonstrated that switching from synchronous to asynchronous updates can radically alter the dynamics of the same theoretical model. Ultimately, their findings highlighted that global synchronous updates produce strong simultaneity artifacts, raising critical questions about the realism of such constraints for natural and social systems.

In the research [10], the authors rigorously established the mathematical construction of modern epidemiological extensions, such as SEIR, on cellular automata to encompass latent incubation phases. They provided the necessary theoretical foundation for discretizing continuous compartmental models onto discrete spatial lattice structures while preserving their essential dynamic properties, which is highly critical for simulating complex, multi-stage temporal state transitions across a physical or network topology.

In the article [9], the authors advanced the understanding of social influence by proposing the Generalized Linear Threshold (GLT) model, which significantly extended classical collective behavior theories. They addressed the limitations of traditional discrete-time threshold models by introducing continuous-time stochastic dynamics and enabling seamless integration with epidemiological frameworks through rate-based transitions. This approach allowed for more nuanced representations of inter-event waiting times and individualized activation speeds based on neighbor interaction.

In the paper [13], the authors analyzed the effectiveness of cellular automata for complex optimization tasks in spatially heterogeneous environments, successfully confirming the meta-heuristic flexibility of this approach. They developed a three-dimensional lattice gas cellular automaton that introduced probability-based cell transitions, where variable environmental factors, such as high-risk or minimal-risk zones, dynamically modulated transmission probabilities. This formulation effectively demonstrated how distinct spatial regions can exhibit entirely different diffusion characteristics over time.

In the study [11], the authors investigated contagion processes extended to higher-order network structures, where interactions occur not just in pairwise connections but within larger group environments. They derived a universal nonlinear infection kernel from heterogeneous exposure, showing that simultaneous exposure through group interac-

tions of varying sizes fundamentally alters epidemic thresholds compared to classical pairwise compartmental models, moving the field explicitly toward more complex, non-linear adoption dynamics.

In the work [7], the authors examined temporal heterogeneity in social systems by proposing a higher-order non-Markovian social contagion model on simplicial complexes. They revealed that when inter-event times for infection and recovery follow non-exponential (Weibull) distributions – characteristic of bursty human behavior – the resulting contagion dynamics deviate substantially from memoryless Markovian predictions. Furthermore, they demonstrated that such temporal concentrations profoundly impact information cascade progression, suggesting that synchronized recovery processes can significantly boost overall system resilience against large-scale infections.

Despite the significant contributions of these studies, the integration of multi-stage epidemiological compartmental models with generalized threshold mechanisms under conditions of individual temporal update heterogeneity remains unaddressed. Most existing cellular automata frameworks assume global synchronization, which introduces artificial propagation waves and fails to reflect realistic human behavior. Therefore, there is a clear need to develop a hybrid asynchronous cellular automata model that combines compartmental SEIR structures and generalized thresholds, which has not been previously investigated in the analyzed literature.

Materials and methods of research. The study of information diffusion was conducted using computer simulation methodology based on cellular automata theory [4, 15]. The modeling was performed on a discrete two-dimensional grid, where individual agents were assigned heterogeneous socio-temporal characteristics. To calculate the probabilistic spread of information, a hybrid algorithmic approach combining SEIR epidemiological compartments and the Generalized Linear Threshold (GLT) model was applied [9]. Statistical processing, scenario validation, and comparative analysis of the obtained asynchronous cascade distributions were conducted utilizing the Monte Carlo method across multiple independent simulation runs.

Research results and their discussion / Результати дослідження та їх обговорення

State Space and Cell Properties. The SEIR-LT-Async model operates on a two-dimensional grid G of size $N \times N$. Each cell c_{ij} at position (i, j) represents an individual agent with the following properties:

State Variable: $\sigma_{ij} \in \{S, E, I, R\}$ where:

- S (Susceptible): has not received information.
- E (Exposed): has received information but not yet spreading.
- I (Infected): actively spreading information.
- R (Recovered): no longer spreading.

Influence Weight: $w_{i,j} \in [1, 1]$ representing the cell's ability to influence neighbors. High-impact users have $w_{i,j} \in [0.8, 1.0]$, while regular users have $w_{i,j} \in [0.1, 0.3]$.

Resistance: $r_{i,j} \in [0, 1]$ representing the cell's resistance to adoption. High-resistance users have $r_{i,j} \in [0.8, 1.0]$, while regular users have $r_{i,j} \in [0.1, 0.3]$.

Individual Threshold: $\theta_{i,j} \in [0.1, 0.6]$ representing the individual's adoption threshold in the GLT framework.

Update Interval (Asynchronous Parameter): $\tau_{i,j} \in [\tau_{min}, \tau_{max}]$ representing the cell's individual update period – the number of time steps between potential state transitions.

Neighborhood Definition. The model uses the Moore neighborhood, which includes all eight adjacent cells:

$$\mathcal{N}(i, j) = \{(i + \Delta i, j + \Delta j) : \Delta i, \Delta j \in \{-1, 0, 1\}, (\Delta i, \Delta j) \neq (0, 0)\}. \quad (1)$$

This yields a maximum of 8 neighbors for interior cells, with boundary cells having fewer neighbors.

Asynchronous Update Mechanism. Following the framework established by Fatès [4], each cell maintains:

- Update interval τ_{ij} : Fixed period between updates;
- Step counter $c_{ij}(t)$: Counts steps since initialization;
- Last update step $t_{last}(i, j)$: Records when cell last transitioned.

Update Eligibility: At each global time step t , cell (i, j) is eligible to update if and only if:

$$\text{canUpdate}(i, j, t) = (c_{i,j}(t) \bmod \tau_{i,j} = 0). \quad (2)$$

Interval Assignment: During initialization, each cell is assigned an update interval:

$$\tau_{i,j} \sim \text{Uniform}(\tau_{min}, \tau_{max}). \quad (3)$$

For integer intervals, this is:

$$\tau_{i,j} = \tau_{min} + \lfloor \xi \cdot (\tau_{max} - \tau_{min} + 1) \rfloor, \quad (4)$$

where $\xi \sim U(0,1)$.

Special Cases:

- $\tau_{min} = \tau_{max} = 1$: Synchronous updates (all cells update every step);
- $\tau_{min} = \tau_{max} = k > 1$: Synchronous with period k ;
- $\tau_{min} < \tau_{max}$: True asynchronous behavior.

Duration Accumulation: Critically, even when a cell cannot update (is not eligible), time-dependent quantities continue to accumulate:

$$\begin{aligned} d_E(i, j, t+1) &= d_E(i, j, t) + 1 \quad \text{if } \sigma_{i,j} = E, \\ d_I(i, j, t+1) &= d_I(i, j, t) + 1 \quad \text{if } \sigma_{i,j} = I. \end{aligned} \quad (5)$$

This ensures that latent and infection periods are measured in real time, not update cycles, maintaining biological plausibility.

State Transition Rules. State transitions occur only when a cell is eligible to update.

Susceptible to Exposed ($S \rightarrow E$). A susceptible cell may become exposed based on a threshold mechanism inspired by the Linear Threshold model [5] and its continuous-time generalization [9]. Within this framework, a user's decision to change state and adopt information is driven directly by the behavioral trends of their immediate surroundings: the transition is triggered when the continuous social influence from active neighbors sufficiently overcomes the individual's innate resistance. First, we identify infected neighbors:

$$\mathcal{I}_{i,j} = \{(k, l) \in \mathcal{N}(i, j) : \sigma_{k,l} = I\}$$

e transition occurs if the cell can update AND two conditions are met:

Condition 1 (LT Threshold): The number of infected neighbors must meet the global threshold: $|\mathcal{I}_{i,j}| \geq \theta_{global}$.

Condition 2 (Probabilistic Adoption): The exposure probability is calculated using an independent cascade formula:

$$P_{exp}(i, j) = \left(1 - \prod_{(k,l) \in \mathcal{I}_{i,j}} (1 - \beta \cdot w_{k,l})\right) \cdot (1 - r_{i,j}). \quad (6)$$

Where $\beta \in [0,1]$ is the base transmission probability. The cell transitions to state E if: $\xi < P_{exp}(i, j)$ where $\xi \sim U(0,1)$ is a uniform random variable.

This formula combines:

- Independent cascade element: each infected neighbor contributes independently to infection probability;

- Resistance factor: individual resistance reduces overall adoption probability;
- Weight influence: higher-weight neighbors have greater influence.

By integrating these components, the GLT methodology formally links individual state changes to the behavioral trends of the network. The transition to an active state is not a simple random occurrence, but a calibrated response to the immediate environment, taking place only when contextual social pressure structurally outweighs personal resistance.

Exposure Counting: Regardless of update eligibility, exposure count is tracked:

$$n_{exp}(i, j, t+1) = n_{exp}(i, j, t) + |\mathcal{J}_{i,j}(t)|, \quad (7)$$

Exposed to Infected ($E \rightarrow I$)

Exposed cells transition to the infected state after a latent period TE :

$$\sigma_{i,j}(t+1) = \text{If} \quad \text{canUpdate}(i, j, t) \wedge \sigma_{i,j}(t) = E \wedge d_E(i, j) \geq T_E, \quad (8)$$

where $d_E(i, j)$ is the duration in the exposed state (measured in time steps, not update cycles).

Infected to Recovered ($I \rightarrow R$)

Recovery probability increases with infection duration, following a saturation curve:

$$P_{rec}(d_I) = \gamma_0 + (1 - \gamma_0) \cdot (1 - e^{-\mu \cdot d_I}), \quad (9)$$

where: γ_0 is the base recovery probability; $\mu = 0.2$ is the recovery rate growth parameter; d_I is the infection duration.

Additionally, forced recovery occurs after maximum infection duration:

$$\sigma_{i,j}(t+1) = R \quad \text{if } \text{canUpdate}(i, j, t) \wedge d_I(i, j) \geq T_I. \quad (10)$$

Dynamic Parameter Evolution. Dynamic Resistance

Resistance decreases with repeated exposure, modeling the "wear-down" effect:

$$r_{i,j}(t) = r_{i,j}^{(0)} \cdot e^{-\lambda_r \cdot n_{exp}(i,j)}, \quad (11)$$

where: $r_{i,j}^{(0)}$ is the base resistance; λ_r is the resistance decay rate; $n_{exp}(i, j)$ is the cumulative exposure count.

Dynamic Weight

Influence weight increases with successful transmissions, modeling reputation effects:

$$w_{i,j}(t) = w_{i,j}^{(0)} \cdot \left(1 + \alpha_w \cdot \frac{s_{i,j}(t)}{t}\right), \quad (12)$$

where: $w_{i,j}^{(0)}$ is the base weight; $\alpha_w = 0.1$ is the weight growth parameter; $s_{i,j}(t)$ is the number of successful transmissions.

Global Statistics and Metrics. Current Prevalence:

$$\rho(t) = \frac{|\{c : \sigma_c = I\}|}{|G_{active}|}. \quad (13)$$

Total Informed (Outbreak Size):

$$I_{total}(t) = \frac{|\{c : \sigma_c \in \{E, I, R\}\}|}{|G_{active}|}. \quad (14)$$

Basic Reproduction Number (estimated):

$$R_0 \approx \frac{\beta \cdot \bar{w} \cdot k}{\gamma_0}, \quad (15)$$

where $\bar{w}$ is the average weight and $k = 8$ is the neighborhood size.

Peak Prevalence: $\rho_{max} = \max_t \rho(t)$

Time to Peak: $t_{peak} = \operatorname{argmax}_t \rho(t)$

Effective Update Rate: For asynchronous systems, the effective fraction of cells updating per step:

$$\phi = \frac{1}{N^2} \sum_{i,j} \frac{1}{\tau_{i,j}} \approx \frac{2}{\tau_{min} + \tau_{max}}. \quad (16)$$

Simulation Methodology. Monte Carlo Approach. Due to the stochastic nature of the model, Monte Carlo simulation with multiple independent runs is employed for statistical analysis. For each parameter configuration:

1. Initialize grid with specified population characteristics.
2. Randomly assign cell properties (weights, resistances, thresholds, update intervals).
3. Randomly infect the initial seed population.
4. Run simulation until equilibrium (no E or I cells remain) or maximum steps are reached.
5. Record final metrics.
6. Repeat for the specified number of runs (typically 50-200).

Equilibrium Detection. Simulation reaches equilibrium when:

$$|\{c : \sigma_c = E\}| = 0 \wedge |\{c : \sigma_c = I\}| = 0. \quad (17)$$

This indicates that information diffusion has completed: all individuals have either adopted and recovered, or remained susceptible.

Statistical Metrics. For each simulation configuration, the following statistics are computed across runs:

- Mean and Standard Deviation of outbreak size, peak prevalence, time to peak;
- Outbreak Probability: fraction of runs where outbreak size exceeds 5 %;
- Distribution Analysis: histograms of key metrics.

Experimental Simulation Outcomes. We conducted all simulations using a standardized parameter configuration unless otherwise specified. The following Table 1 presents the primary (default) parameters used throughout the experimental analysis.

Basic Simulation Dynamics. Before examining specific experimental questions, we illustrate the general behavior of the SEIR-LT-Async model during a typical simulation run. For visualization clarity, a reduced grid size of 70×70 is used (compared to the default 150×150 in Table 1), as this scale allows clearer observation of individual cell states and spatial patterns while maintaining qualitatively similar dynamics.

Figure 1 shows the evolution of spatial patterns at six time points selected to capture key phases of the diffusion process: initial seeding ($t = 0$), expansion phase ($t = 15$), peak activity period ($t = 30$), decline phases ($t = 60, t = 90$), and equilibrium state ($t = 121$, when no Exposed or Infected cells remain). The specific equilibrium time varies between runs due to stochastic elements; $t = 121$ represents this particular realization.

The simulation demonstrates the characteristic SEIR dynamics with a visible latent phase. At each time step, some Susceptible cells become Exposed upon contact with Infected neighbors, but they do not immediately begin spreading – they first spend several steps in the Exposed state (shown in yellow). This latent period creates a distinct "incubation zone" around active spreaders, which is especially visible during early and middle stages of the outbreak. After the latent period expires, Exposed cells transition to Infected and begin influencing their own neighbors, continuing the cascade.

Table 1. Simulation Parameters / Параметри симуляції

Parameter	Symbol	Default Value	Description
Grid Size	N	150	Square grid dimension ($150 \times 150 = 22,500$ cells)
Transmission Probability	B	0.3	Base infection probability per contact
LT Threshold	Θ	1	Minimum infected neighbors required
Recovery Probability	γ_0	0.1	Base recovery probability
Latent Period	T_E	3 steps	Duration in Exposed state before becoming Infected
Max Infection Duration	T_I	10 steps	Forced recovery after this duration
Resistance Decay Rate	λ_r	0.05	Rate of resistance decrease with exposure
Weight Growth Rate	α_w	0.1	Rate of influence increase with transmissions
Recovery Growth Rate	M	0.2	Controls recovery probability increase over time
High-Impact Users	P_I	30 %	Percentage of agents with high influence ($w \in [0.8, 1.0]$)
High-Resistance Users	P_R	20 %	Percentage of agents with high resistance ($r \in [0.8, 1.0]$)
Initial Infected	I_0	1 %	Percentage of randomly infected cells at $t = 0$
Async Min Interval	$\tau_{\min}$	1	Minimum update interval
Async Max Interval	$\tau_{\max}$	3	Maximum update interval (default asynchronous mode)
Monte Carlo Runs	M	100	Number of independent runs per configuration
Max Simulation Steps	$T_{\max}$	1000	Maximum steps before forced termination

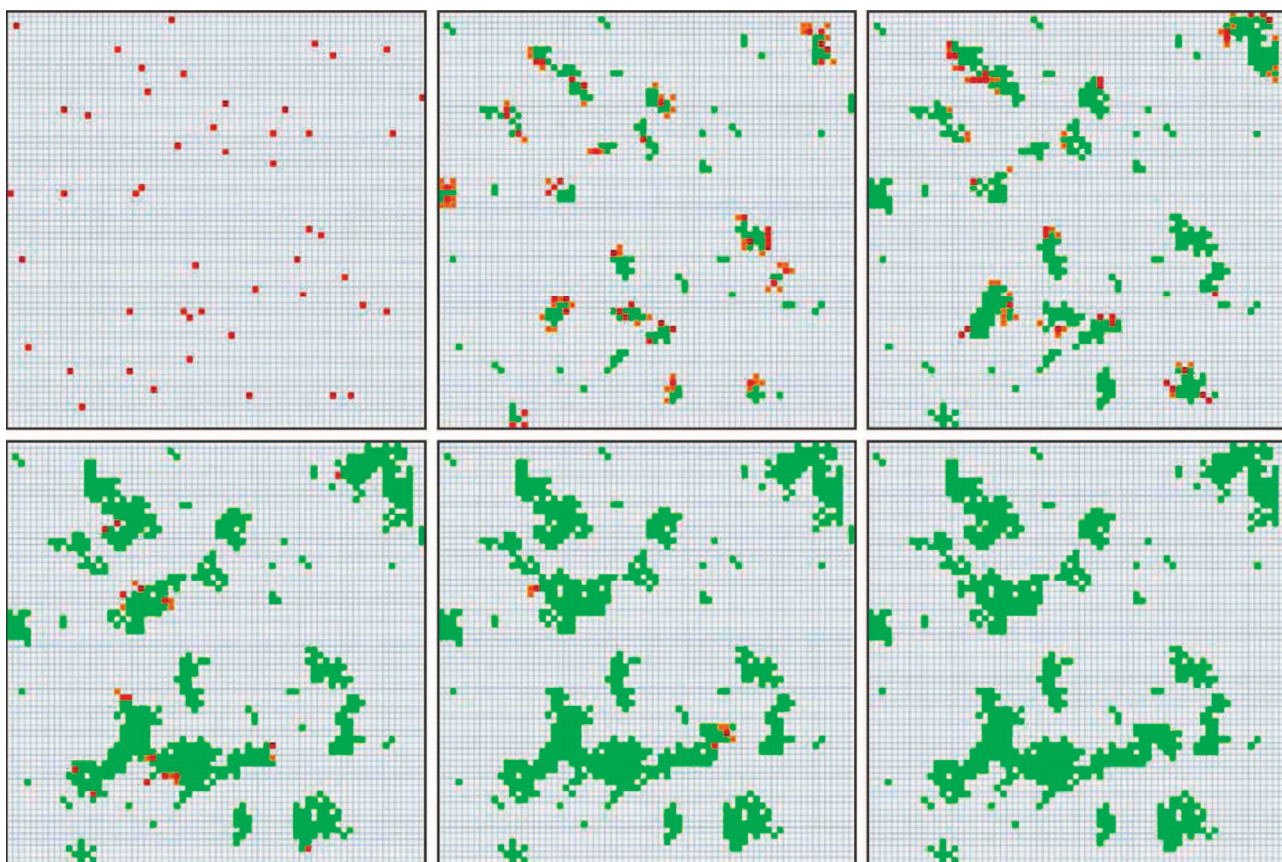


Fig. 1. Grid snapshots at $t = 0, t = 15, t = 30, t = 60, t = 90$ and $t = 121$ (Equilibrium Reached) showing the progression of states / Знімки сітки в процесі часу at $t = 0, t = 15, t = 30, t = 60, t = 90$ and $t = 121$ (Стан рівноваги), що показують прогрес зміни станів

Asynchronous Update Dynamics. In traditional synchronous cellular automata, all cells update simultaneously – an unrealistic assumption for social systems, where individuals differ in response times, attention patterns, and decision-making speeds. Real-world information diffusion is inherently asynchronous: some people check social media every few minutes, others – once a day; processing and deliberation times vary widely. A naive comparison between synchronous $\tau = [1,1]$ and asynchronous $\tau = [1,3]$ conflates two effects: asynchrony itself, and mean update speed. Cells updating every step ($\tau = 1$) naturally have more transition opportunities than cells with mean interval 2.0, making it impossible to attribute differences solely to temporal heterogeneity.

To isolate the pure effect of asynchrony, we designed a controlled experiment in which the mean update interval is held constant while the variance varies. The synchronous

configuration $\tau = [2,2]$ assigns all cells an identical interval of 2 steps. The asynchronous configuration $\tau = [1,3]$ assigns intervals uniformly distributed between 1 and 3 steps, yielding the same mean interval of 2.0 steps: $(1 + 3)/2 = 2.0$. This design ensures that any observed differences arise from temporal heterogeneity alone, not from differences in overall update frequency. The results of 100 Monte Carlo simulations for synchronous and asynchronous update dynamics are given in Table 2.

Asynchrony *increases* both outbreak size (6.17 % vs 5.93 %) and outbreak probability (96 % vs 88 %). The asynchronous configuration produced successful outbreaks in 8 additional runs out of 100. Peak prevalence was slightly lower in async mode (0.92 % vs 0.99 %), suggesting asynchrony "spreads out" infection over time—lower peaks but more sustained transmission.

Table 2. The results of 100 Monte Carlo simulation for synchronous and asynchronous update dynamic / Результат 100 симуляцій за методом Монте-Карло для синхронної та асинхронної динаміки оновлення

Metric	Sync [2,2]	Async [1,3]
τ	[2, 2]	[1, 3]
Mean Interval	2.0	2.0
Mean Outbreak Size, %	5.93	6.17
Std Dev, %	0.70	0.74
Peak Prevalence, %	0.99	0.92
Time to Peak	11 steps	15 steps
Outbreak Prob., %	88.0	96.0
Successful Runs	88/100	96/100

Asynchronous updates provide systemic robustness by leveraging temporal diversity to maintain transmission continuity, effectively eliminating the brittle "blackout windows" inherent in synchronous models. By introducing varied update intervals, the system ensures that fast-responding cells act as continuous bridges, creating constant opportunities for spread even when individual timings are sub-optimal. Ultimately, this variance transforms a fragile, clock-dependent process into a resilient network where the staggered activity of neighbors guarantees that the transmission chain remains unbroken.

Parameter Sensitivity Analysis. Parameter sensitivity analysis identifies which model parameters most strongly influence system behavior. We conducted systematic parameter sweeps, varying each parameter across 10 values while holding others at defaults. For each configuration, 100 Monte Carlo runs on a 150×150 grid provided statistically robust estimates.

Analysis Summary

Parameter: β (Transmission Probability)
Range: 0.100 - 1.000
Steps: 10
Runs per Step: 30

Key Findings

Optimal Value: 1.000 (max outbreak)
Max Outbreak Size: 99.2%
Min Outbreak Size: 1.9%
Sensitivity: High (97.3% range)

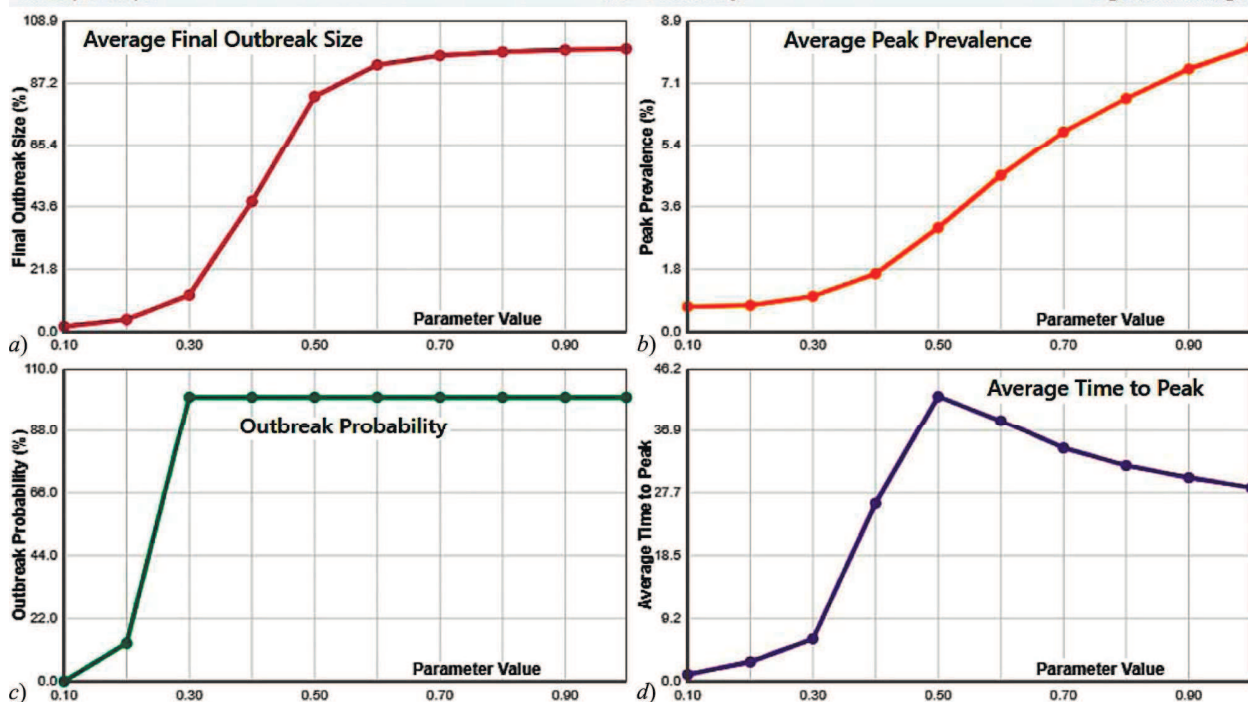


Fig. 2. Parameter Sweep – β / Аналіз чутливості моделі асинхронних клітинних автоматів до зміни параметра β

High-Impact User Percentage. The percentage of high-impact users produced the largest magnitude effects of any parameter tested – a 20x amplification from 5 % outbreak size (at 10 % high-impact users) to 98 % (at 90 % high-impact users). The relationship followed an S-shaped curve:

Measured Metrics:

- **Final Outbreak Size (%)** – the percentage of the population that eventually transitions through the Infected state. This measures the total reach of information diffusion.
- **Peak Prevalence (%)** – the maximum percentage simultaneously infected at any single time step, indicating intensity at peak.
- **Outbreak Probability (%)** – the percentage of runs where final outbreak size exceeds 5 %, indicating reliability of outbreak occurrence.
- **Average Time to Peak (steps)** – the mean number of simulation steps until peak prevalence is reached, indicating how quickly the epidemic develops.

Base Transmission Probability (β). The transmission probability β exhibited the most dramatic influence, revealing a classic epidemiological phase transition. An analysis of the sensitivity of the asynchronous cellular automaton model to changes in the parameter β is shown in Figure 2.

At low values ($\beta = 0.1$), no outbreaks occurred – the infection died in every run. Between $\beta = 0.2$ and $\beta = 0.3$, a sharp transition occurred: outbreak probability jumped from 13 % to 100 % and mean outbreak size tripled from 4 % to 13 %. This marks the critical transmission threshold at approximately $\beta \approx 0.25$. Above this threshold, outbreak size continued growing with diminishing returns, reaching 83 % at $\beta = 0.5$ and 99 % at $\beta = 1.0$.

The existence of this sharp critical threshold provides a clear intervention target – reducing transmission probability below β_{crit} guarantees outbreak prevention, while small changes near the threshold produce disproportionately large effects. This is the most sensitive leverage point in the model.

slow growth below 30 %, explosive growth from 30 % to 65 %, then saturation above 70 %. Outbreak probability showed a critical transition around 15-20 % high-impact users, jumping from 40 % to nearly 100 %. Below this threshold, the high-impact network is fragmented; above it,

high-impact users form a connected backbone that reliably propagates infection. An analysis of the sensitivity of the model of asynchronous cellular automata to changes in the proportion of influencing users is shown in Figure 3.

This quantifies the "influencer effect" – a small minority of highly influential users can dramatically amplify infor-

mation spread. For misinformation containment, targeting high-impact users provides leveraged impact. Conversely, for beneficial campaigns, recruiting even 20 % high-impact participation shifts the system from unreliable to certain outbreak success.

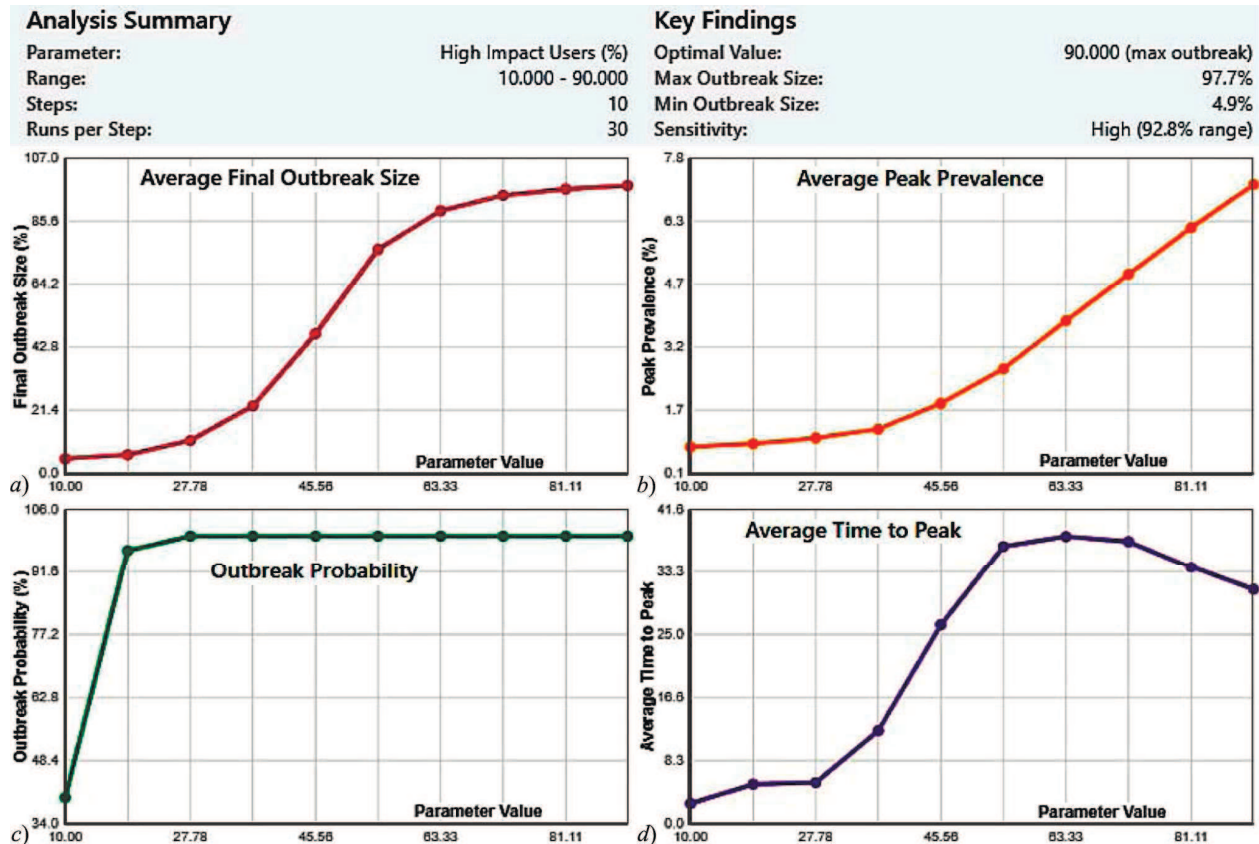


Fig. 3. Parameter Sweep – High-Impact User / Аналіз чутливості моделі асинхронних клітинних автоматів до зміни частки впливових користувачів

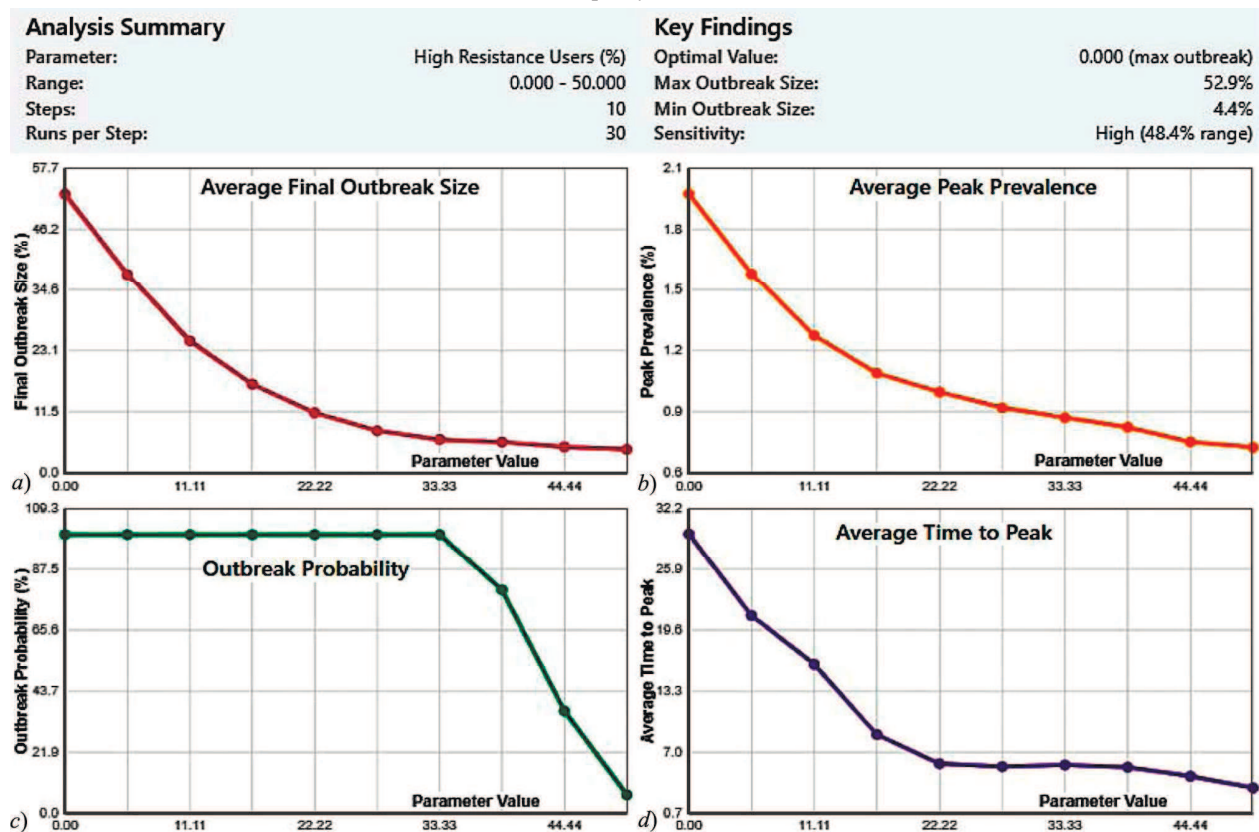


Fig. 4. Parameter Sweep – High-Resistance User / Аналіз чутливості моделі асинхронних клітинних автоматів до зміни частки стійких користувачів

High-Resistance User Percentage. High-resistance users provided powerful outbreak suppression, acting as "firebreaks" in the transmission network. Increasing resistant users from 0 % to 50 % reduced outbreak size from 53 % to 4 % – a 92 % reduction. Outbreak probability remained at 100 % until resistant users reached 35-40 %, then dropped sharply to just 7 % at 50 % resistant. An analysis of the sensitivity of the model of asynchronous cellular automata to changes in the proportion of stable users is shown in Figure 4.

Notably, the suppression effect (12x) was weaker than the high-impact amplification effect (20x), revealing a fundamental asymmetry: facilitating spread is easier than preventing it. Defensive interventions must be more extensive than offensive efforts to achieve comparable effects.

Resistant users function analogously to vaccinated individuals – they provide indirect protection to neighbors by blocking transmission chains. If resistance represents media literacy, these results suggest educating 40-50 % of the population could reduce outbreak probability from 100 % to below 10 %. Even partial coverage provides substantial benefits: 22 % resistant users reduced outbreak size by 79 %.

Parameter Sensitivity Summary. The analysis shows that the composition of the population matters most. Increasing the share of influential users from 10 % to 90 % amplified outbreaks 20-fold, while increasing resistant users from 0 % to 50 % reduced them 12-fold. The transmission probability β has a critical threshold around 0.25: below it, outbreaks die out; above it, they spread reliably.

An important practical finding is that spreading information is easier than stopping it. The amplification effect (20x) is stronger than the suppression effect (12x). This means that to counter misinformation, defensive measures must reach a larger portion of the population than would be needed to spread information using influencers.

Discussion of research results. The developed SEIR-LT-Async model incorporates three fundamental aspects of information diffusion in social networks: epidemiological state transitions (Susceptible, Exposed, Infected, Recovered), threshold-based activation mechanisms inspired by the Linear Threshold model, and individual temporal heterogeneity through asynchronous updates. The controlled experimental comparison between synchronous and asynchronous configurations demonstrates that temporal heterogeneity fundamentally alters diffusion dynamics, increasing both outbreak probability (96 % vs 88 %) and outbreak size (6.17 % vs 5.93 %). Furthermore, parameter sensitivity analysis identifies a notable asymmetry: facilitating information spread remains inherently easier than preventing it.

In the study [1], researchers developed a network clique cover approximation to analyze complex contagions, showing that densely connected subgroups accelerate threshold-based spreading, yet their group interaction framework lacked individual deterministic asynchrony and focused predominantly on homogeneous temporal assumptions.

In the article [6], the authors introduced novel topological measures for identifying the spread of complex contagions, demonstrating that standard shortest-path metrics fail outside of simple contagions; however, their approach remained synchronous and did not incorporate the "wear-down" resistance mechanism presented in our findings.

In the paper [2], researchers examined the temporal properties of higher-order interactions in social networks, confirming that group interactions form bursty clusters rather than uniform temporal distributions, although they omitted

epidemiological compartmental latency structures from their spreading analysis.

In the study [14], the authors explored the competition among memes under limited user attention, establishing that information propagation is constrained by individual cognitive limits, but their model did not integrate the Generalized Linear Threshold mechanism to weigh distinct types of social reinforcement.

In the work [3], researchers analyzed efficient influence maximization in social networks utilizing threshold models, providing scalable methods for identifying initial seeds, yet their focus was primarily computational without addressing how asynchronous staggered updates organically reshape the cascade geometry itself.

Ultimately, the discussion of these research results confirms the profound necessity of the conducted study, because while previous literature has addressed aspects of threshold adoption, epidemiological spreading, or spatial modeling individually, none have successfully combined them within a strictly asynchronous cellular automata paradigm that accurately eliminates the artifacts of global synchronization the way our developed model accomplishes.

So, based on the results of the work performed, it is possible to formulate the following scientific novelty and practical significance of the research results.

Scientific novelty of the obtained research results – further development was made to the hybrid model of asynchronous cellular automata, SEIR-LT-Async, which, unlike existing models, integrates the epidemiological compartmental structure with the Generalized Linear Threshold mechanism and individual temporal update intervals, which makes it possible to eliminate unrealistic global synchronization artefacts and ensure a more accurate reproduction of the distribution of information cascades in heterogeneous populations.

Practical significance of the research results – can be applied to the study of information diffusion and the design of systemic intervention strategies targeting misinformation containment, as the proposed framework effectively enables simulating how adjusting the percentage of high-impact users or increasing population resistance influences the overall epidemic outcomes in digital communities.

Conclusions / Висновки

A novel SEIR-LT-Async (Susceptible-Exposed-Infected-Recovered with Linear Threshold and Asynchronous Updates) cellular automata model has been developed, implemented, and analyzed, which more realistically captures the temporal heterogeneity of information diffusion in social networks compared to traditional synchronous approaches. Based on the results of the research the following main conclusions can be drawn.

1. Formulated a hybrid SEIR-LT model integrating the epidemiological compartmental structure with the Generalized Linear Threshold (GLT) mechanism, providing a rigorous mathematical framework to calculate activation probabilities based on individual resistance and neighbors' influence.
2. Designed and implemented a novel asynchronous update mechanism with individual update intervals, which successfully captures temporal response heterogeneity and eliminates the unrealistic wave-like artifacts of global synchronization.
3. Integrated dynamic parameter evolution (resistance decay and influence growth) into the model, demonstrating how social learning and the "wear-down" effect of repeated exposure reshape agent susceptibility and reputation over time.

4. Conducted comprehensive Monte Carlo simulations comparing synchronous and asynchronous dynamics, which statistically quantified that asynchronous updates increase the mean outbreak size (from 5.93 % to 6.17 %) and elevate the overall outbreak probability (from 88 % to 96 %).
5. Performed parameter sensitivity analysis to identify critical thresholds, establishing that the transmission probability exhibits a sharp phase transition at $\beta \approx 0.25$, and revealing a fundamental asymmetry where spreading information via influencers requires less effort than suppressing it via resistant users.
6. Evaluated the model's ability to reproduce qualitative features of real-world diffusion, proving that temporal heterogeneity naturally spreads peak prevalence (reducing it from 0.99 % to 0.92 %) and maintains transmission chain continuity through fast-responding agents.
7. Outlined future research directions to investigate complex network topologies, multiple information types, and adaptive update intervals, alongside empirical calibration against empirical social media datasets.

References

1. Burgio, G., Arenas, A., Gómez, S., & Matamalas, J. T. (2021). Network clique cover approximation to analyze complex contagions through group interactions. *Communications Physics*, 4, article number 111. <https://doi.org/10.1038/s42005-021-00618-z>
2. Cencetti, G., Battiston, F., Lepri, B., & Karsai, M. (2021). Temporal properties of higher-order interactions in social networks. *Scientific Reports*, 11, article ID 7028. <https://doi.org/10.1038/s41598-021-86469-8>
3. Chen, W., Wang, Y., & Yang, S. (2009). Efficient influence maximization in social networks. In *Proceedings 15th ACM SIGKDD International Conference on Knowledge Discovery and Data Mining* (pp. 199–208). <https://doi.org/10.1145/1557019.1557047>
4. Fatès, N. (2014). A Guided Tour of Asynchronous Cellular Automata. *Journal of Cellular Automata*, 9(5-6), 387–416. URL: <https://inria.hal.science/hal-00908373v5/document>
5. Granovetter, M. (1978). Threshold models of collective behavior. *American Journal of Sociology*, 83(6), 1420–1443. <https://doi.org/10.1086/226707>
6. Guilbeault, D., & Centola, D. (2021). Topological measures for identifying and predicting the spread of complex contagions. *Nature Communications*, 12, article ID 4430. <https://doi.org/10.1038/s41467-021-24704-6>
7. Lin, Z., Han, L., Feng, M., Liu, Y., & Tang, M. (2024). Higher-order non-Markovian social contagions in simplicial complexes. *Communications Physics*, 7, article number 175. <https://doi.org/10.1038/s42005-024-01666-x>
8. Moreno, Y., Nekovee, M., & Pacheco, A. F. (2004). Dynamics of rumor spreading in complex networks. *Physical Review E*, 69(6), article ID 066130. <https://doi.org/10.1103/PhysRevE.69.066130>
9. Ran, Y., Deng, X., Wang, X., & Jia, T. (2020). A generalized linear threshold model for an improved description of the spreading dynamics. *AIP Advances*, 10, article ID 085223. <https://doi.org/10.1063/5.0011658>
10. Sirakoulis, G. C., Karafyllidis, I., & Thanailakis, A. (2000). A cellular automaton model for the effects of population movement and vaccination on epidemic propagation. *Ecological Modelling*, 133(3), 209–223. [https://doi.org/10.1016/S0304-3800\(00\)00294-5](https://doi.org/10.1016/S0304-3800(00)00294-5)
11. St-Onge, G., Sun, H., Allard, A., Hébert-Dufresne, L., & Bianconi, G. (2021). Universal nonlinear infection kernel from heterogeneous exposure on higher-order networks. *Physical Review Letters*, 127(15), article ID 158301. <https://doi.org/10.1103/PhysRevLett.127.158301>
12. Torskyi, O. I., & Hrytsiuk, Y. I. (2025). Application of machine learning to enhance the efficiency of automated software testing. *Scientific Bulletin of UNFU*, 35(4), 142–149. <https://doi.org/10.36930/40350416>
13. Vorobiov, Ye. S., Pavlenko, M. A., Khliebnikov, Ye. Yu., & Hladyshch, M. H. (2018). Using cellular automata in the method of selecting flight route variants for strike aircraft. *Systemy ozbroiennia i viiskova tekhnika*, 1(53), 84–90. <https://doi.org/10.30748/soivt.2018.53.12>
14. Weng, L., Flammini, A., Vespignani, A., & Menczer, F. (2012). Competition among memes in a world with limited attention. *Scientific Reports*, 2, article ID 335. <https://doi.org/10.1038/srep00335>
15. Wolfram, S. (2002). A New Kind of Science. Champaign, IL: Wolfram Media. URL: <https://horizons-2000.org/92.%20Misc%20Files/Reading/Wolfram%20%20A%20New%20Kind%20of%20Science.pdf>

I. M. Грабовенська, I. O. Рабійчук, А. В. Фечан, Р. О. Яценко

Національний університет "Львівська політехніка", м. Львів, Україна

ГІБРИДНА АСИНХРОННА МОДЕЛЬ КЛІТИННИХ АВТОМАТІВ SEIR-LT ДЛЯ ПОШИРЕННЯ ІНФОРМАЦІЇ У СОЦІАЛЬНИХ МЕРЕЖАХ

Наведено гібридну асинхронну модель клітинних автоматів для моделювання процесів поширення інформації у соціальних мережах, де поєднано функціонал класичних компартментних структур (Сприйнятливі-Експоновані-Інфіковані-Видужалі) з теорією лінійного порогу. Застосовано індивідуалізований механізм оновлення станів користувачів для максимально точної фіксації часової неоднорідності: кожному учаснику мережі призначаються унікальні інтервали активності замість одностороннього переходу об'єктів усієї популяції. З'ясовано, що такий асинхронний підхід до моделювання дещо детальніше характеризує реальну поведінку людей, які реагують на зовнішні подразники з різною швидкістю та увагою. Введено методичку узагальненого порогу GLT (англ. *Generalized Threshold Model*) для визначення ймовірності переходу користувача в активний стан – момент, коли соціальний вплив оточення перевищує індивідуальний поріг опору, змушуючи її прийняти інформацію. Охарактеризовано закономірності розвитку гетерогенної популяції користувачів з виділенням класів впливових та стійких учасників мережі. Встановлено, що відмова від глобальної синхронізації користувачів дає змогу повністю усунути нереалістичні хвилюподібні артефакти, притаманні для традиційних обчислень. Протестовано функціонування спроектованої архітектури асинхронних клітинних автоматів із використанням методології Монте-Карло на двовимірній сітці. Оцінено вплив часової гетерогенності на середній обсяг каскаду, який продемонстрував зростання від 5,93 % за умов повного синхронізму до 6,17 % у локалізованих неоднорідних конфігураціях. Виявлено істотне підвищення загальної ймовірності масштабного поширення інформації: запропонований механізм забезпечив успішну передачу імпульсу у 96 % зі 100 макроскопічних ітерацій порівняно з тільки 88 % за традиційних обмежень. Зафіксовано природне згладжування інтенсивності процесу, що підтверджується зниженням пікової поширеності (максимальної частки популяції, що перебуває в інфікованому стані та активно поширює інформацію) від 0,99 до 0,92 % та ефективним перерозподілом навантаження впродовж дещо тривалішого відрізка етапів дослідження. Досліджено вплив параметрів на розроблену модель асинхронних клітинних автоматів та проаналізовано фазовий перехід базової ймовірності з критичною точкою близько рівня 0,25. Виявлено фундаментальну асиметрію поширення інформації в мережі: запустити масштабні інформаційні каскади за допомогою малої кількості впливових користувачів значно легше, ніж придушити їх збільшенням кількості стійких учасників мережі. Розроблену асинхронну модель клітинних автоматів запропоновано використовувати для прогнозування та тестування стратегій стримування небажаного контенту.

Ключові слова: узагальнений лінійний поріг; стримування дезінформації; епідеміологічні компартменти; соціальне зараження; часова гетерогенність.