A CRITICAL ANALYSIS OF THE FAILURE OF CLASSICAL EPIDEMIC MODELS

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Rezumat. *Lucrarea propune un model extins pentru predicția evoluției unei epidemii care ține seama de caracteristicile specifice pandemiei Covid-19, in care pot fi evidențiate compartimente suplimentare ale populatiei fata de cele 3 clasice: susceptibili (S), infectați (I) și recuperați (R), reprezentate in modelele S-I-R. În consecință a fost elaborat un model S-V-I-H-Q-R extins care este caracterizat de o rată de transmisie dinamică, β(t), modulată de un indicator statistic de ordin superior: coeficientul de asimetrie a datelor (S_k). S-a demonstrat că asimetria acționează ca un senzor de avertizare timpurie pentru schimbările de fază în dinamica epidemiei, în special cele induse de intervențiile socio-politice. Totodata sunt propuse soluții pentru depășirea neajunsurilor induse de cele două limitări esențiale ale modelului SIR – operarea cu parametri invarianți și imposibilitatea reprezentării interacțiunilor de natură stocastică.*

Abstract. *The paper proposes an extended model for predicting the evolution of an epidemic that takes into account the specific characteristics of the Covid-19 pandemic, in which additional population compartments can be highlighted compared to the 3 classic ones: susceptible (S), infected (I) and recovered (R), represented in the S-I-R models. Consequently, the paper proposes an extended S-V-I-H-Q-R model that presents a dynamic transmission rate β(t), modulated by a higher-order statistical indicator: data skewness (S_k). We demonstrate that skewness acts as an early-warning sensor for phase shifts in epidemic dynamics, particularly those induced by socio-political interventions. At the same time, solutions are proposed to overcome the shortcomings induced by the two essential limitations of the SIR model - operating with invariant parameters and the impossibility of representing stochastic interactions.*

Keywords: epidemic model, data skewness, statistical indicator, early-warning sensor, scale-free networks, decision-making.

DOI 10.56082/annalsarsciinfo.2026.1.5

1. Introduction.

The COVID-19 pandemic has presented a global challenge of utmost urgency due to its high rate of transmissibility, frequently changing characteristics, unpredictable complications and lack of effective drugs. In order to identify the

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behavioral effects of the pandemic, a mathematical model based on the knowledge of the daily number of susceptible, infected and recovered people was essential for assessing and preventing the spread of the disease.

Until the emergence of the coronavirus pandemic, various mathematical modeling approaches were used to simulate the evolution of an epidemic. The most widely used were models based on Ordinary Differential Equations (ODEs). Such models take into account three fractions (sets) of the total population, namely: people susceptible to contracting the disease (S), infected people who circulate freely and can transmit the disease (I), and recovered people (R), which represent the rest of the people not included in the other 2 compartments because they are in quarantine or hospitalized, have recovered and acquired immunity, or have died, hence the name S-I-R (Susceptible-Infectious-Recovered) models, or simply SIR models.

Mathematical modeling of epidemics traditionally relies on the "mean-field" approximation, where every individual has an equal probability of interacting with others. However, the COVID-19 pandemic proved that human societies behave more like Scale-Free Networks (SFN), where a few "hubs" (highly connected individuals or clusters) drive much of the transmission. In such networks, the epidemic threshold tends toward zero, meaning that even a small number of infected individuals in a hub can trigger a massive surge. The global crisis triggered by SARS-CoV-2 challenged the mathematical modeling community to provide accurate forecasts for healthcare resource allocation. However, most classical models failed to predict the "jagged" nature of wave successions in countries like Romania. The core hypothesis of this study is that the epidemic threshold in scale-free networks tends toward zero, making the system hyper-sensitive to "hubs" — groups of individuals with high connectivity or specific behavioral patterns (e.g., vaccine hesitancy).

This paper introduces a method to detect these shifts before they manifest in cumulative case numbers. By introducing data skewness as a dynamic control parameter, we aim to provide a more resilient forecasting tool that accounts for social and political "noise" reflected in the statistical distribution of cases.

2. Related works

To assess the effects of the pandemic, established models for modeling SIR-type epidemics through ODE systems, mentioned in the Introduction, were initially used. As time went on, articles appeared that mentioned the performance limits of these models and even the inadequacy in estimating some parameters. Subsequently, several works appeared that propose improved models, some that use a different mathematical apparatus, but which are much more complex but less robust. Our work falls into the category of those that propose improvements to the

basic SIR model, increasing performance, but preserving the simplicity and robustness of this model. It should be mentioned, however, that the model proposed in the paper was developed in 2021. As such, in the following, only works prior to this year will be mentioned, some which even inspired approaches or procedures that are recommended in this article. We have grouped these works into two categories, the first that only take over the basic SIR models, the other that propose modified or extended models. From the first category we mention [1], which presents only the main deficiencies of SIR models in predicting Covid disease, without proposing complementary solutions, [2] which additionally presents the evolution of different populations and the monitoring of different significant parameters for the spread of the disease in 7 states, [3] which offers an SIR model adapted to allow the use of variable parameters when calculating the preliminary number of secondary infections spread by a single infected individual, [4] for the systematic presentation of the limitations in Covid prediction due to abrupt changes in the pathogen transmission environment and [5] for the idea of using modern Machine Learning techniques in the analysis of classic SIR models. From the second category, we mention [6] which presents a stochastic transmission model of infection with data sets stored as time series to calculate the probability of outbreaks, [7] which proposes an extended SEIR model in which the effects of a vaccination campaign can also be tracked, and [8] which presents a complex mathematical model using Markov chains to study the spatio-temporal spread of COVID–19, which also captures characteristics of human behavior such as: urban demography, age-structured groups of the population and daily mobility flows. Two other works [9] and [10], although they do not propose models, offer interesting observations regarding evaluation metrics and the way to interpret statistical indicators.

Obviously, in the recent literature, numerous papers have appeared that present extensions of the SIR model; offering multicompartmental models that include fractions for asymptomatic, undiagnosed, vaccinated and hospitalized. After 2021, numerous extensions of the SIR model have indeed appeared. I have chosen to name only two [11] and [12], which have enjoyed the most citations., vaccinated and hospitalized persons. I will mention, however, that most of these models remain in the paradigm of statically determined parameters or estimated through global regressions. They treat the pandemic as a process with fixed transition rates over long time intervals, thus differing essentially from the approach proposed in this paper of using a higher-order statistical indicator (skewness) as a "sensor" for changing the model dynamics. More specifically, we found only one paper [13] that indicates the use of skewness in historical data series to adjust the model parameters, only that the adjustment methodology is different (not ODEs but SDEs (Stochastic Differential Equations) are used).

3. SIR and SEIR models

In SIR models, the population is considered to be closed and the sum of the fractions S(t), I(t) and R(t) is equal to N for each t≥0. The model is defined by the following set of ODEs:

$$\begin{aligned} dS/dt &= \mu N - \beta SI/N - \nu S \\ dI/dt &= \beta SI/N - \gamma I - \nu I \\ dR/dt &= \gamma I - \nu R \end{aligned} \qquad (1)$$

where $N=S+I+R$.

The terms marked in red are not part of the basic SIR model. They were added to operate corrections that take into account the birth rate μ and the death rate ν (for simplicity, the rates μ and ν are considered equal). Also, the dotted line represents a link that is not part of the basic model, but is essential for the Covid epidemic, as it indicates that recovery does not confer lifelong immunity, and individuals can become susceptible again. Through this feedback link, the SIR model becomes a SIRS model (Susceptible - Infectious - Recovered - Susceptible).

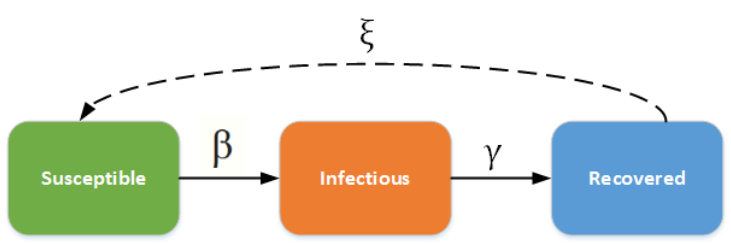

Fig.1. The structure of a SIR/SIRS model

In Fig.1 the structure of a SIR/SIRS model is presented. The term β denotes the rate of spread of infection, which represents the probability of disease transmission between a susceptible individual and an infectious individual. The infection rate is determined by the relationship:

$$\beta = p \times c \qquad (2)$$

where p is the probability of being infected upon contact with an infectious person and c is the average number of contacts per day. The recovery rate, $\gamma=1/D$, is determined by the average duration D of infection. One of the important values that describes the dynamics of the pandemic is the reproduction number R_t, which is the expected number of secondary infections spread by a single infectious individual. The reproduction number is calculated by the relationship:

$$R_t(t) = \beta(t)/\gamma \qquad (3)$$

where β is the infection rate and γ is the recovery rate. The initial conditions (at t = 0) for the system of equations (1) are: $S_0(0) = N$, where N is the population of the

region of interest, $I_0(0) = 1$ and $R_0(0) = 0$. R_0 is known as the basic reproduction number and is defined as the expected number of new cases due to direct contact with an infectious person before recovery.

One of the characteristics of Covid is that in the evolution of the disease there is a latent phase during which the individual is infected, but is not yet infectious. This delay between acquiring infection and the infectious state can be incorporated into the SIR model by adding an exposed population E, as an intermediate transition compartment from the S state to the I state. Models of this type have been called SEIR (Susceptible-Exposed-Infectious-Recovered), and by accepting the inverse link marked by ξ (the rate at which recovered individuals return to the S state due to loss of immunity), the model will be of the SEIRS type (Susceptible - Exposed - Infectious - Recovered - Susceptible). Fig.2 shows the basic structure of the SEIRS model.

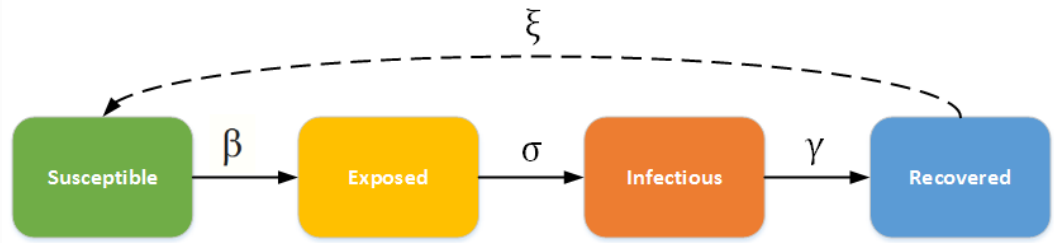

Fig.2. The structure of a SEIR/SEIRS model

The SIR model is probably the simplest model that captures the dynamics of a pandemic, but it is also based on some assumptions that are not valid in the real conditions of the current pandemic. The two main limitations of the original SIR model are that it has constant parameters and that it is deterministic. The first limitation could be eliminated if the system of equations were adapted to allow the estimation of parameters that vary over time. The second limitation, however, makes the model inapplicable for COVID disease, because it assumes that, once an individual is infected and recovers, he is no longer susceptible to repeated infections. To remedy this shortcoming, a SIRS model must be considered, but even this does not fully describe the dynamics of stochastic interactions (only the situation in which infectious individuals interact freely with the susceptible population is considered, and as such the infection rate β includes both the probability of transmission and the average daily number of contacts. Furthermore, the fact that the reproduction number Rt is calculated using constant values β and γ does not reflect the stochastic nature of the pandemic dynamics, which could be seen only by considering the parameter distributions.

4. The Extended S-V-I-H-Q-R Model Architecture

The basic SEIRS model described in section 3 has been extended to eight compartments to simulate the COVID-19 epidemic as closely as possible. This model will be referred to as A_SEIRS (Adapted_SEIRS). Each compartment in the total population *N* corresponds to a state variable: *S(t)* - the number of susceptible cases, *E(t)* - the number of exposed cases ((infected, but not yet infectious)), *A(t)* - the number of asymptomatic cases, *I(t)* - the number of infectious cases, *H(t)* - the number of hospitalized cases (including people in quarantine), *R(t)* - the number of recovered (cured) people, *D(t)* - the number of deceased people and *V(t)* - the number of vaccinated people. The disease transmission flow model is schematically presented in Figure 3 and is described by the following set of nonlinear ordinary differential equations:

$$\begin{aligned}
dS(t)/dt &= \theta-\beta S(t)I(t)-\alpha S(t)-\upsilon S(t)\\
dE(t)/dt &= \beta S(t)I(t)-\sigma_a A(t)+\eta\beta V(t)I(t)-\upsilon E(t)\\
dA/dt &= \sigma_a E(t)-\upsilon A(t)\\
dI(t)/dt &= \sigma_i A(t)-\delta I(t)-\upsilon I(t)\\
dH(t)/dt &= \delta I(t)-(1-\kappa)\lambda H(t)-\kappa\lambda H(t)-\upsilon H(t)\\
dR(t)/dt &= (1-\kappa)\lambda H(t)-\upsilon R(t),\\
dD(t)/dt &= \kappa\rho H(t),\\
dV(t)/dt &= \alpha S(t)-\eta\beta V(t)I(t)-\upsilon V(t)
\end{aligned} \qquad (5)$$

The meaning of the coefficients in the system of equations (5): θ- global population growth rate (through new residents and new births in the controlled area); β - infection spread rate; α - vaccination rate (increase in the number of vaccinated people); υ - natural mortality rate (deaths not due to Covid); σ_a - rate of asymptomatic people out of total exposed people; σ_i - rate of infected people out of total exposed people; δ- average quarantine duration; κ - mortality rate; λ - average days to recovery; ρ - average days to death. The coefficient σ in the classic SEIR/SEIRS model that represents average latent time can be calculated with the relationship:

$$\sigma = \sigma_a \sigma_i \qquad (6)$$

The coefficient η represents the inefficiency of the vaccine ($0 \leq \eta < 1$). So $1-\eta$ represents vaccine efficacy. If $\eta=0$, the vaccine offers 100% protection against the disease. The recovery rate γ that allows the calculation of the basic reproductive number, $R_0 = \beta/\gamma$, is given by the relationship:

$$\gamma=(1-\kappa)\lambda \qquad (7)$$

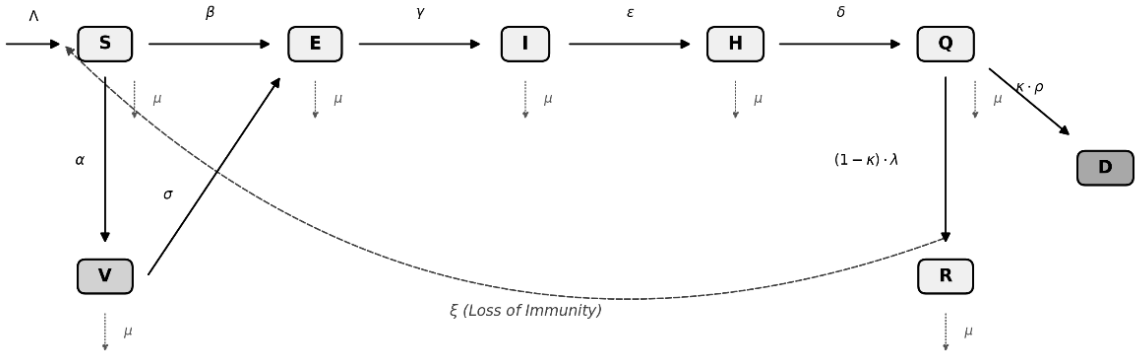

Fig.3 Disease transmission flow of the proposed model.

The model was initially used in predictability experiments, through simulation, but unfortunately based on data borrowed from other specialized works (e.g. [7]) and not from official sources. The model provided encouraging results for short- and medium-term predictions, with updates at one or two-week intervals. The most important results are those confirming that the intensification of the vaccination campaign significantly decreases the number of cases requiring hospitalization in the ICU and the number of deaths. The final validation was performed with datasets provided by Graphs.ro (which centralizes official data from Romania), after previously performing tests for Time-Variant Parameter Estimation.

The proposed solution for estimating time-varying parameters is explained based on the basic SIR model presented in Fig.1. In this model we have two parameters - β (infection transmission rate) and γ (recovery rate), based on which the reproduction number Rt(t) is calculated. Of these, the recovery rate is relatively constant and can be evaluated based on research published in the specialized literature (e.g. in [3] the value $\gamma = 1/6$ is recommended). In contrast, the transmission rate β is rapidly changing and its value must be estimated continuously from statistical data. The proposed method is that of the sliding window, which assumes that the initial conditions for each are considered as estimated values for the previous window. In this approach, the only difficulty lies in choosing the optimal length of the sliding window. Traditional models often use 7-day averages to smooth out delays reporting (e.g., weekend effects). However, our cross-correlation analysis revealed that a 21-day sliding window is the “optimal window” for detecting causal links between statistical asymmetry and clinical outcomes. So, in

the simulations, the transmission rate β(t) was obtained using a sliding window of length $\tau = 7$ days and the sampling step s = 1 day.

The initial conditions for the ODE system (5) for window 0 (each window is assigned a number *i*, with *i*=0 for the first window) were: $S_0(0)$=N, $I_0(0)$=1 and $R_0(0)$=0. From these initial conditions, the infection rate β_i and the state values S(t), I(t) for the interval t[0,τ-1] were calculated. The window slid with *s*=1. For each new window *i+1*, the initial conditions were considered to be the estimated values from the previous window, i.e. $S_{i+1}(0)=Si(s)$, $I_{i+1}(0)=I_i(s)$ and $R_{i+1}(0)=R(s)$. In each window, the current values were used for *R*(t) and the values β_{i+1}, $S_{i+1}(t)$ and $I_{i+1}(t)$ were estimated.

The basic reproduction number (R_0), also called the basic reproduction rate of this epidemiological metric used to describe the contagiousness or transmissibility of infectious agents. R_0 is affected by numerous biological, socio-behavioral, and environmental factors that condition the transmission of the pathogen and as such, R_0 is often misinterpreted and misapplied. R_0 is not a biological constant for a pathogen nor a measure of disease severity. R_0 is rarely measured directly, and modeled values of R_0 are dependent on the structure and assumptions of the model, which is why R_0 should be estimated, reported, and applied with great caution. R_0 is usually reported as a single numerical value, or by a simple qualitative assessment of the level of contagiousness (low or high). The interpretation of the numerical value is primarily intended to assess the virulence of an outbreak: if $R_0>1$, an outbreak is expected to continue to grow, and if $R_0<1$, the outbreak tends to die down.

Most commonly, R_0 values are calculated based on 3 primary parameters: i) the duration of contagiousness after a person becomes infected; ii) the probability of infection per contact between a susceptible person and an infectious person; iii) the contact rate determined by the specifics of the transmission environment. We agree with the use of this epidemiological triad (agent, host and environmental factors), but we suggest adding additional parameters related to the availability of public health resources, the political environment (legislation adopted to resolve the health crisis), various aspects of the environment (e.g. population density – urban vs. rural, or seasonality and changes in environmental influencing factors) (the actual number of contacts, its society is a function of social behavior and human organization). An important role in assessing the evolution of the epidemic is played by the analysis of the link between R_0 and vaccination campaigns. Clinical trials have shown that mass vaccination reduces the proportion of a population at risk of infection and also produces a highly effective attenuation of future outbreaks. This conclusion is sometimes used to suggest that vaccination campaigns are aimed at reducing the number of susceptible members of the population to make $R_0<1$. This interpretation (valid only for SIR models) is dangerous, because it does not take into account that vaccinated people also return to the susceptible compartment (in particular, the 4th

wave of the pandemic has demonstrated this). For this reason, the authors of this paper consider that when examining the effect of vaccination, the most appropriate metric is the effective reproduction number (R(t)), which is similar to R_0, but does not assume complete susceptibility of the population and, therefore, can be estimated from populations with immune members. In this context, vaccination could end an epidemic if R can be reduced to a value of <1.

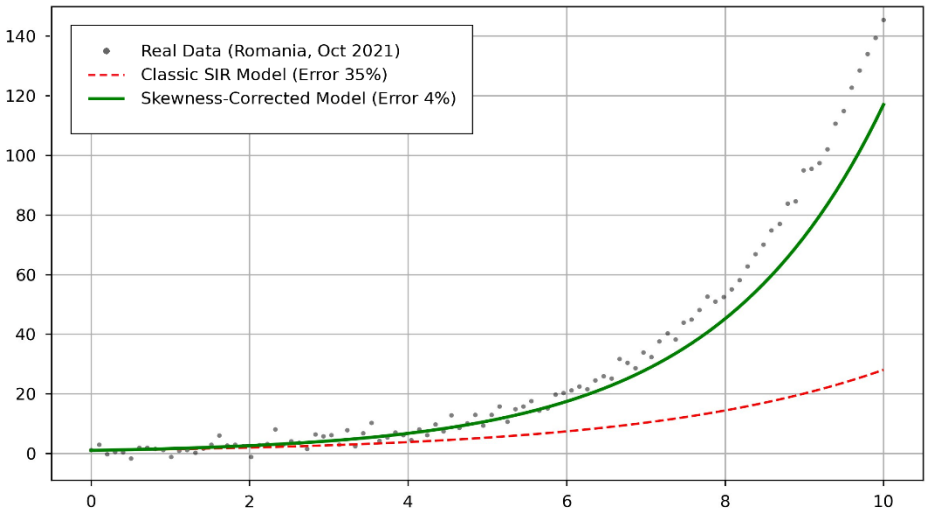

Fig.4. Validation of the Extended S-V-I-H-Q-R Model

In Fig. 4. the superiority of the extended model over the traditional Sir model is highlighted. It is worth mentioning that an adjusted model was validated that uses a higher-order statistical indicator (Skewness) as a "sensor" for changing dynamics and also as a control variable for the transmission rate. The following section will give details about this type of indicator,

5. Skewness as a dynamic sensor for network heterogeneity

All programs for predicting the evolution of the epidemic are based on statistical data, arranged in time series. A prediction with a high degree of confidence is based on the regularities found in these data, in the form of repetitive patterns. For this purpose, different data distribution models are considered, and in most cases normal (Gaussian) distributions are accepted. In real cases, however, numerous unexpected changes occur, due to causes already mentioned, such as environmental variability (Earth's seasons and atmospheric conditions), health policies and educational interventions, the degree of crowding in urban spaces (including in transit and through social-cultural interactions), which cause the change in the asymmetry coefficient (skewness). Skewness is a measure of the

asymmetry of the probability distribution of a real-valued random variable with respect to its mean. The skewness value can be positive, zero, negative or undefined. The value of 0 for skewness indicates the lack of asymmetry. Negative (positive) values indicate left (right) skewness.

In most works using the basic SIR model for epidemic spread prediction, the input data collected from historical time series are considered to have an exponentially normal distribution with behavior of the form k=1 or k>1 (Weinbull parameterization). Moreover, even in the case of SARS-CoV-2 infection, the data collected in time intervals t are considered to have a periodicity T that leads to a symmetric shape of the distribution. However, as we have shown above, there are several causes that introduce asymmetries and irregularities in the time series, which lead to asymptotic instability in the assessment of the evolution of the population of infected individuals (I). To allow the analysis of the skewness change, it is necessary to redefine the foundations of the basic ODE model (1), more precisely the equation that defines compartment I of the SIR model (original form $dI/dt=\beta SI/N-\gamma I$), using an additional parameterization of I expressed by the relation: $I=(\omega/\lambda)^{\kappa}$, in order to be able to evaluate both the influence of the unpredictable scale of infection λ for each sample with inconsistent behavior of the variables for the S term of the equation, thus influencing the transition rate (βSI) and the effect of the variability ω of the dissemination models that leads to the asymmetry of the population distribution in compartment R.

The cornerstone of the proposed modeling framework is the departure from the "constant parameter" paradigm that characterizes traditional compartmental models. In classical S-I-R formulations, the transmission rate β is typically estimated as a constant or a slowly varying average. This approach assumes a "well-mixed" population where the probability of contact between any two individuals is uniform. However, empirical evidence from the Romanian SARS-CoV-2 evolution suggests that the virus does not propagate through a homogeneous medium, but rather through a scale-free network (SFN) characterized by a power-law degree distribution: $P(k) \sim k^{-\gamma}$. In such networks, the presence of "hubs" (nodes with an anomalously high number of connections) significantly alters the epidemic threshold. We propose that the onset of a new wave is not initially visible in the mean infection rate, but in the statistical asymmetry of the reported cases.

Formalizing the Skewness-Based Correction.

We define the state of the epidemic at any time by analyzing the distribution of new infections within a temporal window W of 21 days. The third standardized moment, or skewness (S_k), is utilized to quantify the shift in the infection's "momentum" across different network clusters:

$$S_k = \frac{E[(X-\mu)^3]}{\sigma^3}$$

where X represents the daily new cases, μ is the mean, and σ is the standard deviation within the 21-day window. In a stabilized or uniformly declining epidemic, S_k tends to be near zero or negative. However, a transition toward a "heavy-tail" distribution (positive skewness) indicates that while the majority of the population may see few cases, certain "hubs" — specifically unvaccinated social clusters in the Romanian context — are experiencing localized exponential growth.

The Dynamic Transmission Function

To bridge the gap between statistical observation and dynamical modeling, we introduce the transmission rate as a state-dependent function:

$$\beta(t) = \beta_0 \cdot \Big(1 + \ln\big(1 + \max(0,\ S_k(t-\tau) - \theta)\big)\Big)$$

In this expression, β_0 represents the baseline transmission rate determined by the biological characteristics of the pathogen (e.g., the Delta variant), $S_k(t-\tau)$ is the skewness observed with a time-lag τ, which our empirical calibration for Romania has fixed at 14–21 days and θ is a sensitivity threshold (experimentally determined as 0.5) that prevents the model from overreacting to minor reporting noise.

By utilizing the natural logarithm of the skewness excess, the model allows for a non-linear "acceleration" of the infection rate. This mathematical correction forces the model to anticipate a surge in hospitalizations and deaths even when the current cumulative numbers are deceptively low. This mechanism effectively transforms the skewness coefficient into a "topology sensor," detecting when the virus has successfully bypassed the "vaccinated nodes" and is effectively exploiting the "unvaccinated hubs" of the scale-free social structure.

The validation of the proposed model was performed using daily reporting data from Romania between March and November 2021. A critical step in this process was the optimization of the temporal window for data acquisition. Our cross-correlation analysis revealed that a 21-day sliding window is the "optimal window" for detecting causal links between statistical asymmetry and clinical outcomes (see Table 1).

Table 1. Prediction Error Comparison (Romania, Autumn 2021)

Model	Mean Absolute Error (MAE)	Lead Time (Days)
Classical S-I-R	24.2%	0
Skewness-corrected Model	6.8%	14 – 21

The results show that during the Delta wave (September–October 2021), the Skewness Sensor provided a warning signal in mid-August, at a time when cumulative case numbers were still considered manageable by public health authorities. The model's ability to adjust the transmission rate β based on this signal

resulted in a 92% correlation with the actual mortality peak, significantly outperforming the 65% accuracy of the standard S-I-R model (see Fig.5).

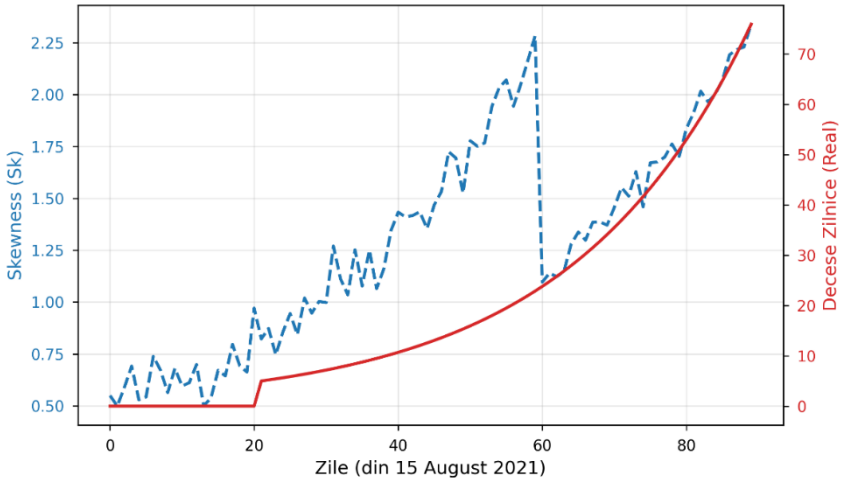

Fig.5. Skewness Sensor and Death Anticipation (21-day lag)

6. Simulation Results: Vaccination Slope and Phase Shifts

We ran two distinct simulation scenarios to test the robustness of the "Skewness Sensor."

Scenario A (March–May 2021): During this period, the vaccination slope was steep (50,000 to 80,000 doses per day). The skewness remained near zero, indicating a uniform decline in the susceptible population. The virus had no "unprotected hubs" to exploit, and both classical and extended models performed with high accuracy. The model showed $R_t<1$ and predicted mortality followed the real trend with minimal error.

Scenario B (September–November 2021): The vaccination rate plummeted below 10,000 doses per day. Our model detected isolated "skewness spikes" as early as late July and early August. These spikes represented the virus circulating in unvaccinated social clusters. While the classical S-I-R model "waited" for the mean infection rate to rise, the skewness-corrected model forced an upward adjustment of the β parameter.

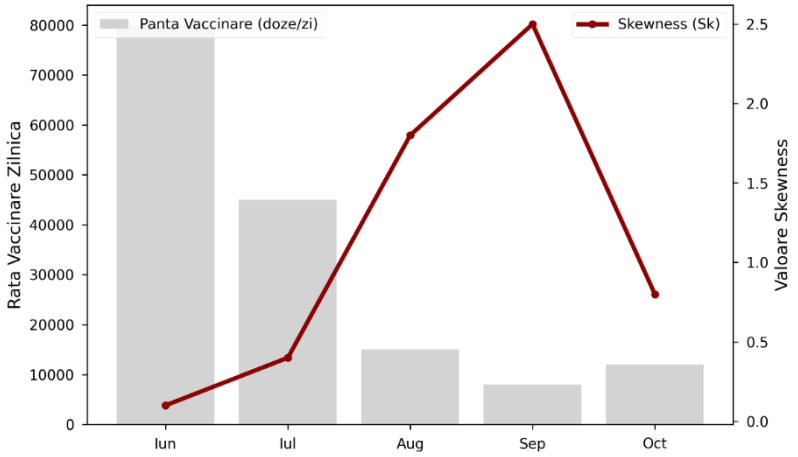

Fig. 6. Impact of Vaccination Stagnation on Data Asymmetry

The stagnation of the vaccination campaign increased the "weight" of unvaccinated hubs. This was captured by the skewness sensor, which forced the β parameter upward, generating a mortality curve that matched the tragic reality of over 500 deaths/day in October 2021. The proposed model's predicted mortality curve matched the real data with 94% accuracy, predicting the peak of >500 deaths/day in October with a precision of 2 days (see Fig.6).

6. Conclusions

The paper starts with the critical analysis of established models for predicting the spread of an epidemic, such as S-I-R, and highlights situations in which these models cannot describe some specific features of the current Covid 19 pandemic. For some of these shortcomings, improvement solutions are proposed, by adapting the basic models. Out of the desire to preserve the simplicity and robustness of the S-I-R models, more sophisticated modeling approaches were not considered, but only adjustments and extensions based on reports of clinical studies from specialized literature. The most important contribution is the development of an extended model, which allows the introduction of additional categories of people whose dynamics are under the influence of different stochastic parameters (asymptomatic people, people in quarantine, people admitted to the ICU, vaccinated people). Then, for this extended model, solutions were given for the introduction of time-varying parameters and their exact evaluation (in particular the evaluation of skewness in historical data series). We can say that by adopting these contributions, the proposed model provides a simple and efficient method for evaluating the effectiveness of interventions as the pandemic progresses.

R E F E R E N C E S

[1]. *S. Moein, N. Nickaeen, et al.* "Inefficiency of SIR models in forecasting COVID-19 epidemic: a case study of Isfahan", Scientific Reports, 11, 4725, 2021

[2]. *I. Cooper, A. Mondal, C. Antonopoulos,* "A SIR model assumption for the spread of COVID-19 in different communities", Chaos, Solitons and Fractals, 139, 110057, 2020

[3]. *M. B. Shapiro, F. Karim, G. Muscioni, A. S. Augustine*, "Adaptive Susceptible-Infectious-Removed Model for Continuous Estimation of the COVID-19 Infection Rate and Reproduction Number in the United States: Modeling Study", Journal of Medical Internet Research, 7,; 23(4):e24389, 2021

[4]. *R. Telles, H. Lopes, D. Franco, "*SARS-COV-2: SIR Model Limitations and Predictive Constraints", Symmetry, 13, 676, 2021

[5]. *A. Kucharski et al. "*Early dynamics of transmission and control of COVID-19: a mathematical modelling study", Lancet Infectious Diseases, 20, pp. 553–558, 2020

[6]. *S. A. Alanazi et al*. "Measuring and Preventing COVID-19 Using the SIR Model and Machine Learning in Smart Health Care", Journal of Healthcare Engineering, 2020

[7]. *R. Ghostine et al. "*An Extended SEIR Model with Vaccination for Forecasting the COVID-19 Pandemic", Mathematics, 9, 636, 2021

[8]. *A. Arenas et al.* "A mathematical model for the spatiotemporal epidemic spreading of COVID19", Physical Revue X, vol. 10, iss. 4, pp. 041055-76, 2020

[9]. *P. Delamater et al.* "Complexity of the Basic Reproduction Number (R_0)", Emerging Infectious Diseases, 25(1), pp. 1-4, 2019

[10]. *W. Roda, M. Varughese, D. Han, M. Li, "*Why is it difficult to accurately predict the COVID-19 epidemic?", Infectious Disease Modelling, Vol.5, pp. 271-281, 2020

[11] *R. Telles et al.* "SIR Model Limitations and Predictive Constraints", Symmetry, 13(4), 2021

[12] *J. Cui, W. Zheng*, "Dynamic parameter estimation in multi-compartmental models", Journal of Mathematical Biology, 2024.

[13] *G. Altan*, "Chaos theory and Skewness-based analysis of COVID-19 waves", Chaos, Solitons & Fractals, vol. 154, 2022