Sumer Journal for Pure Science (ISSN: 2790-7031)
SJPS 2026 (June), Vol. 5, No. 1, p.p.: 64-75

An Extended SIRD Model Incorporating Healthcare System Overload Effects

Banen Kadhim Jawad[1]*, Ali Hussein Shuaa AL-Taie[2]
[1,2]*Department of Mathematics, College of Education for Pure Science, Wasit University, Al-Kut, Iraq*
* Email address of the Corresponding Author: std.2024205.b.jawad@uowasit.edu.iq
Phone number: +964 774 032 7090

Abstract

This paper proposes an expanded model of the SIRD epidemiological model of COVID-19, which differs from traditional models in that it calculates the cumulative mortality rate of COVID-19, calculating direct deaths from the disease and indirect deaths resulting from pressure on the health care system. Initially, simple linear equations were used, but numerical simulations using MATLAB revealed a discrepancy in the paths, which led to a decrease in the total number of deaths. To correct this discrepancy, the research developed the model using a nonlinear saturation mechanism linked to the number of people recovered. The model shows that the number of recoveries is inversely proportional to the burden on health facilities. Based on our model developed, all parameters of the model are biologically reasonable as shown in results. The simulation results confirm that adding a stress factor to the healthcare model contributes positively to improving the effectiveness of the model in predicting the dynamics of mortality development in the epidemic. This framework is a useful guide that enables decision makers to evaluate the effectiveness and impact of public health interventions.

Keywords: SIRD model, Extended SIRD model, COVID-19

نموذج SIRD موسع يتضمن آثار زيادة الضغط على نظام الرعاية الصحية

بنين كاظم جواد[1]*, علي حسين شعاع الطائي[2]
[1,2]قسم الرياضيات – كلية التربية للعلوم الصرفة – جامعة واسط – واسط – العراق

الخلاصة

تقترح هذه الورقة نموذجًا موسعًا لنموذج SIRD الوبائي لمرض كوفيد-19، والذي يختلف عن النماذج التقليدية في أنه يحسب معدل الوفيات التراكمي لمرض كوفيد-19، من خلال حساب الوفيات المباشرة الناتجة عن المرض والوفيات غير المباشرة الناتجة عن الضغط على نظام الرعاية الصحية. في البداية، تم استخدام معادلات خطية بسيطة، إلا أن المحاكاة العددية باستخدام برنامج MATLAB أظهرت وجود اختلاف في المسارات، مما أدى إلى انخفاض في العدد الكلي للوفيات. ولتصحيح هذا الاختلاف، قام البحث بتطوير النموذج باستخدام آلية تشبع لاخطية مرتبطة بعدد الأفراد المتعافين. يبيّن النموذج أن عدد المتعافين يتناسب عكسيًا مع العبء على المرافق الصحية. وتؤكد نتائج المحاكاة أن إضافة عامل ضغط إلى نموذج الرعاية الصحية يساهم بشكل إيجابي في تحسين فعالية النموذج في

التنبؤ بديناميكيات تطور الوفيات في الوباء. ويعد هذا الإطار دليلاً مفيداً يمكّن صناع القرار من تقييم فعالية وتأثير التدخلات الصحية العامة.

1. Introduction

Infectious diseases represent one of the most pressing global concerns facing medical infrastructure systems. Because of this, quantitative modeling has proven to be a key approach for understanding the transmission of infectious diseases, predicting outbreak trajectories, and assessing the appropriateness of various disease control methods. Models derive from splitting the population into several disease compartments and linking these to dynamic equations that help to describe the temporal flow of individuals between these population compartments [1]. The SIR model, pioneered by Kermack and Mackendrick in 1927, is the best known model in this domain. It divides the population into three main categories: susceptible persons (S), infected persons (I), and recovered persons (R). This model had served as the basis for many later epidemiological models, which are designed to better capture the transmission of diseases [2]. The SIRD model, which incorporates a mortality class (D) to distinguish deaths from disease, is a model modification of the SIR model that has been proposed with the rise of epidemiological research [3]. Multiple epidemics, such as the COVID-19 pandemic, have been extensively studied using these models which serve as estimators of the rate of disease propagation and a finding for where the peak took place [4]. However, health system capacity does not have a bearing on mortality, which is fixed, as conventional epidemiological models have claimed. In fact, in large epidemics, health systems are confronted with severe pressure due to the staggering rise in patient numbers, which can result in medical material insufficiency, postponed treatment delivery and lowered quality of health care [5]. In these cases, indirect deaths may occur, attributable not only to the disease but also to pressure on the health care system and its limited resources [6]. Therefore, there was a need to develop more realistic mathematical models that take into account the impact of health system pressure on mortality rates and the undocumented prevalence of cases [7]. Integrating these mechanisms and the impact of travel restrictions into epidemiological models can provide a more accurate description of epidemic dynamics and clarify the relationship between health system capacity and epidemic intensity [8]. In 2020, a mathematical model of SIR was studied in Italy, comparing detected and undetected infections, different degrees of disease severity, non-life-threatening cases (asymptomatic or mildly symptomatic; mild or moderate infection), and potentially life-threatening cases (serious or severe) requiring admission to the intensive care unit [9]. In this study, we first analyze a modified SIRD model that includes indirect mortality effects associated with increased burden on the healthcare system. Numerical simulations of this formula reveal epidemiologically unrealistic behavior in cumulative mortality dynamics, and to address this shortcoming, we propose an expanded and improved SIRD model that presents a nonlinear saturation mechanism driven by the recovering community.

2. First Model (Initial SIRD Model)

The COVID-19 pandemic highlights the need for mathematical models to understand and predict the dynamics of infectious disease transmission. In this section, we will study on to the more detailed SIRD model, where deaths are separated into a separate column (D) to enable us to track mortality statistics directly and indirectly related to the pandemic due to healthcare system overload. Removing the dead changes the population balance, which affects the number of susceptible individuals and forecast accuracy. Without the category (D), all infected

individuals are categorized as recovered, which is unrealistic for fatal disorders like COVID-19. The system of equations that is given below describes the modified SIRD epidemic model:

$$\frac{dS}{dt} = -\beta\, S(t)I(t) \tag{1}$$

$$\frac{dI}{dt} = \beta\, S(t)I(t) - \gamma\, I(t) - \mu\, I(t) \tag{2}$$

$$\frac{dR}{dt} = \gamma\, I(t) \tag{3}$$

$$\frac{dD}{dt} = \lambda\, S(t) + \lambda\, I(t) - \alpha\, R(t) \tag{4}$$

It is important to note that variable D does not only represent the rate of direct deaths (caused by the epidemic), but it also represents indirect deaths, which are those resulting due to pressure from the health care system (such as delayed treatments, lack of resources, complications acquired from hospitals, and delays in operations).

$\lambda\, S(t)$: represents indirect mortality among susceptible individuals (not infected), primarily due to healthcare system overcrowding and the consequent failure of medical interventions. Examples include delayed operations, hospital-acquired infections, untreated bacterial infections, and sometimes antibiotic shortages.

$\lambda\, I(t)$: Denotes mortality among infected individuals despite receiving treatment, reflecting the limitations of medical care in saving critical cases.

$-\alpha\, R(t)$:The recovered population contributes to alleviating pressure on the healthcare system, thereby reducing overall mortality. This creates an inverse or protective population effect, whereby an increase in the number of recovered individuals enhances community immunity, mitigates healthcare burden, and consequently lowers indirect deaths. In Table 1, we explained the basic variables used in the model and their meaning.

In accordance with our model, we made the following assumptions throughout the simulations:

1. The model calculates both direct deaths from the pandemic and indirect deaths due to failure of medical interventions. Examples include delayed operations, hospital-acquired infections, untreated bacterial infections, and sometimes antibiotic shortages.

2. We assume that recovered individuals indirectly contribute to reducing mortality by reducing the pressure on healthcare resources (because they have acquired immunity and will not return to the hospital soon).

3. All state variables remain non-negative.

Figures 1 and 2 shows the numerical simulation of the SIRD model developed, where the time trajectories of the different categories are shown in different colors: blue for susceptible individuals (S), red for infected individuals (I), green for recovered individuals (R), and black for deceased individuals (D).

Table 1- Model parameters and their meanings.

Symbol	Description
S	Individuals susceptible to infection.
I	People infected (carriers of the coronavirus).
R	Recovered individuals from the disease and acquired temporary immunity.
D	The cumulative number of deaths resulting from both direct and indirect effects of the epidemic.
β	The rate of virus transmission from infected individuals to those susceptible to infection through contact.
γ	Recovery rate describes the speed at which infected individuals recover.
μ	Death rate from the disease, representing deaths directly caused by the infection.
λ	Indirect mortality rate associated with health care system overload and treatment failure, affecting both susceptible and infected individuals.
α	The indirect protective effect of recovered patients by reducing pressure on the healthcare system.

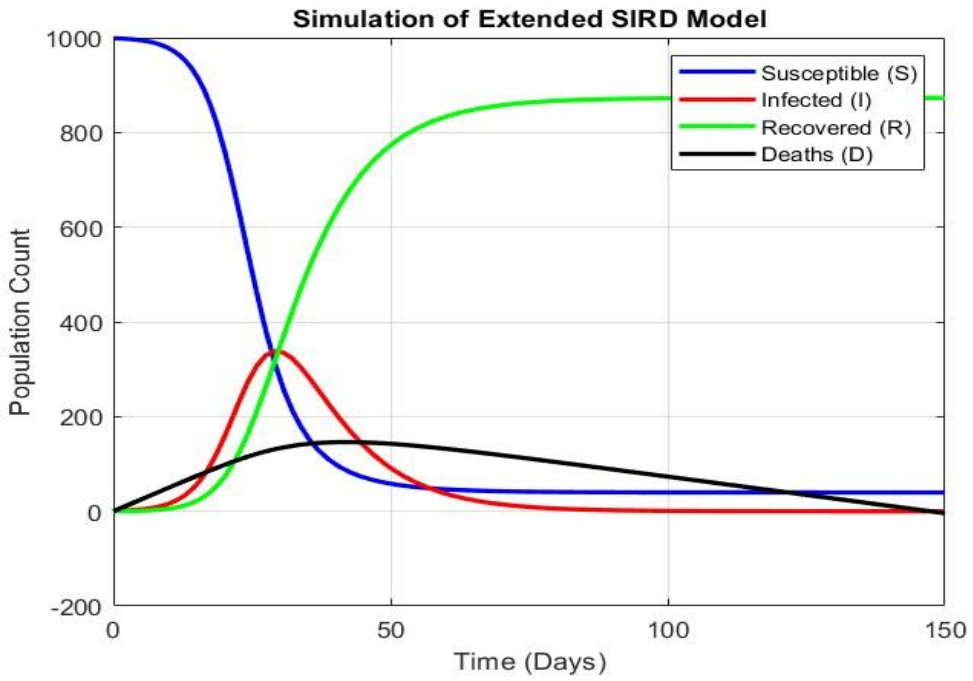

Figure -1 Numerical simulation of the SIRD model developed at $\beta = 0.4, \gamma = 0.1, \mu = 0.01, \lambda = 0.005,\ \alpha = 0.002, t = 150$ and $N = 1000$.

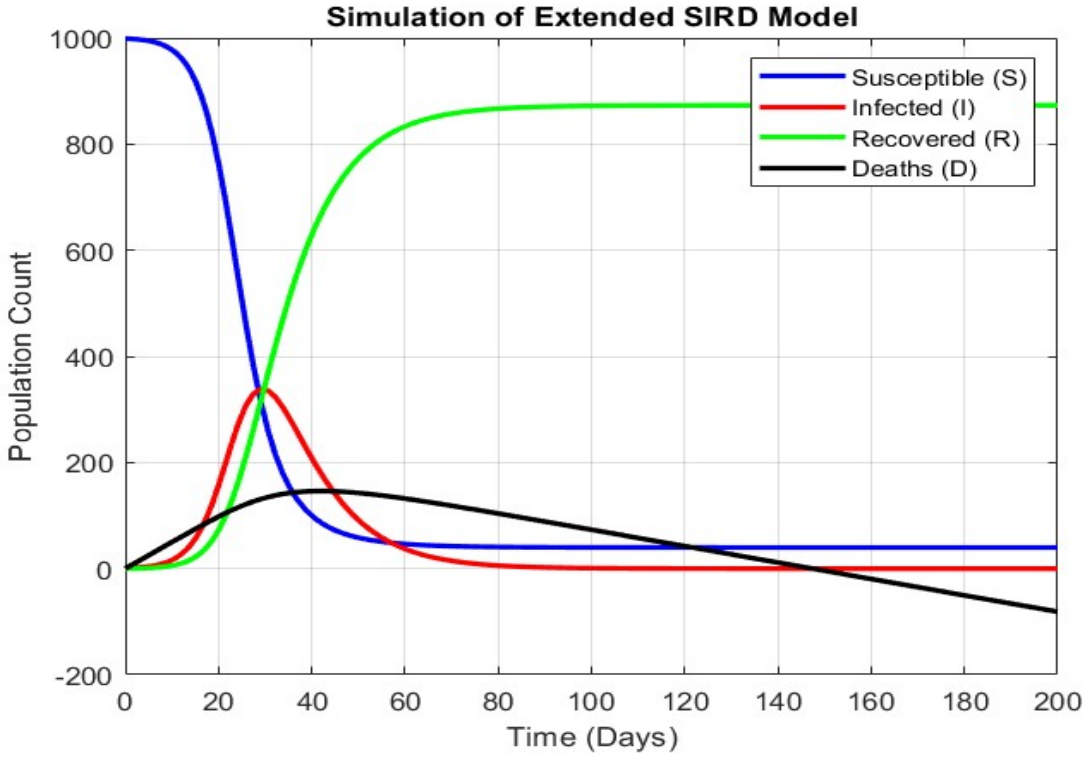

Figure -2 Numerical simulation of the SIRD model when $t = 200$.

2.1 Problem in the First Model

By carefully observing the results obtained from the numerical simulation of the first model as shown in Figure 1, a flaw in the epidemiological realism of the mortality curve (D) becomes apparent. We observe that the curve begins to decline after a certain period, mathematically suggesting a decrease in the cumulative number of deaths, which is illogical in the study of pandemics. This is due to the linear structure of the mortality equation, which allows negative values to appear when the number of recoveries (R) increases. Therefore, an expanded version of the SIRD model was adopted. This modification is designed to address the observed deviation in model behavior.In addition, the proposed framework ensures that parameters remain within realistic biological ranges and compatible numerical limits. As described in the next section. This model replaces the linear limit with a nonlinear saturation mechanism. This conversion maintains a positive cumulative increase in deaths over time.

3. Proposed Extended SIRD Model

In order to better understand the dynamics of large-scale epidemics such as COVID-19, the proposed extended SIRD model presents a nonlinear structure. This model differs from traditional models that assume mortality rates from disease only in that this model calculates rates of direct mortality from disease and indirect mortality associated with pressure on the health care system. The model is based on a preventive representation based on the saturation principle, which is directly related to recovery dynamics within the community. The higher the number of recovered individuals, the less pressure on health services. As a result, indirect deaths are gradually decreasing, especially those resulting from the limited capacity of the medical system. This nonlinear interaction enhances the model's more accurate representation of epidemiological reality. However, its mathematical basis is nevertheless coherent and analyzable. The balance in the system maintains a gradual and stable increase of cumulative deaths where no irrational behavior occurs. The model offers a much more integrated view of

epidemic dynamics by associating the effectiveness of a health system with the degree of community immunity. It enables deeper analyses of mortality trajectories despite limited medical capabilities. This is of increasing importance in places where health services are under pressure from the spread of disease. The system of equations that is given below describes the modified SIRD epidemic model:

$$\frac{dS(t)}{dt} = -\beta\, S(t)I(t) \tag{5}$$

$$\frac{dI(t)}{dt} = \beta\, S(t)I(t) - \gamma\, I(t) - \mu\, I(t) \tag{6}$$

$$\frac{dR(t)}{dt} = \gamma\, I(t) \tag{7}$$

$$\frac{dD(t)}{dt} = \mu\, I(t) + \frac{\lambda\big(S(t) + I(t)\big)}{1 + \alpha\, R(t)} \tag{8}$$

Where $\mu\, I(t)$: This limit refers to the direct mortality rate associated with infected individuals. That is, it describes cases that end in death despite receiving medical care. Its value increases as the severity of the infection increases.

$\lambda(S(t) + I(t))$: This concept encompasses indirect deaths, extending beyond those infected to include those at risk of infection. The main reason for these deaths is the limited capacity of the health system during periods of high stress. In such circumstances, necessary medical procedures may be delayed, the likelihood of hospital-acquired infections may increase, and some important treatments may be in short supply.

$\frac{1}{(1+\alpha R(t))}$: This term refers to the protective effect that a population gains as a result of individuals' recovery. Increasing the number of recoveries leads to a decrease in the number of indirect deaths because it reduces pressure on health care and enhances community immunity.

α: This term refers to the effectiveness of recovered populations in reducing the health care burden. A large value of (α) indicates a stronger protective effect and a greater reduction in indirect mortality. In Table 2, we explained the basic variables used in the model and their meaning.

To verify that the proposed model is mathematically correct before performing numerical simulations given non-negative initial conditions, the model keeps all state variables non-negative, ensuring that population segments remain epidemiologically meaningful over time. Moreover, the aggregate population remains bounded, precluding implausible unbounded growth and guaranteeing that trajectories evolve within a biologically feasible region. The cumulative mortality compartment is formulated to remain monotonically non-decreasing, thus preserving alignment with empirical fatality records.

Table 2- Model parameters and their meanings.

Symbol	Description
S	Individuals susceptible to infection.
I	People infected (carriers of the coronavirus).
R	Recovered individuals from the disease and acquired temporary immunity.
D	The cumulative number of deaths resulting from both direct and indirect effects of the epidemic.
β	The rate of virus transmission from infected individuals to those susceptible to infection through contact.
γ	Recovery rate describes the speed at which infected individuals recover.
μ	Death rate from the disease, representing deaths directly caused by the infection.
λ	The indirect mortality rate is related to the strain on the healthcare system. Its impact is subject to a saturation mechanism that depends on the number of recoveries, reflecting a decrease in indirect mortality as the strain on the healthcare system decreases.
α	The indirect protective effect of recovered patients by reducing pressure on the healthcare system.

Key points of the presented model serve as validation for its theoretical rigor and its appropriateness in qualitative methods, such as the equilibrium and stability analyses. Figure 3 presents the temporal evolution of model variables. The compartments are depicted with color: the susceptibles (S) are plotted in blue, the infected inhabitants (I) in red, the recovered inhabitants (R) in green, and the cumulative deaths (D) in black. The number of the infected increases initially; it reaches a peak and then declines slowly. The recovered class, on the other hand, grows over time, because immunity accumulates in the population. The trajectories both trend towards a saturating state and do not experience an unrealistic death curve drop. This behavior suggests a more realistic construction than the classical SIRD-type models. This is an effect due to nonlinear nature in which the indirect mortality behavior is well controlled. Thus the cumulative deaths remain monotonically increasing, eliminating the inconsistency observed in the earlier formulation.

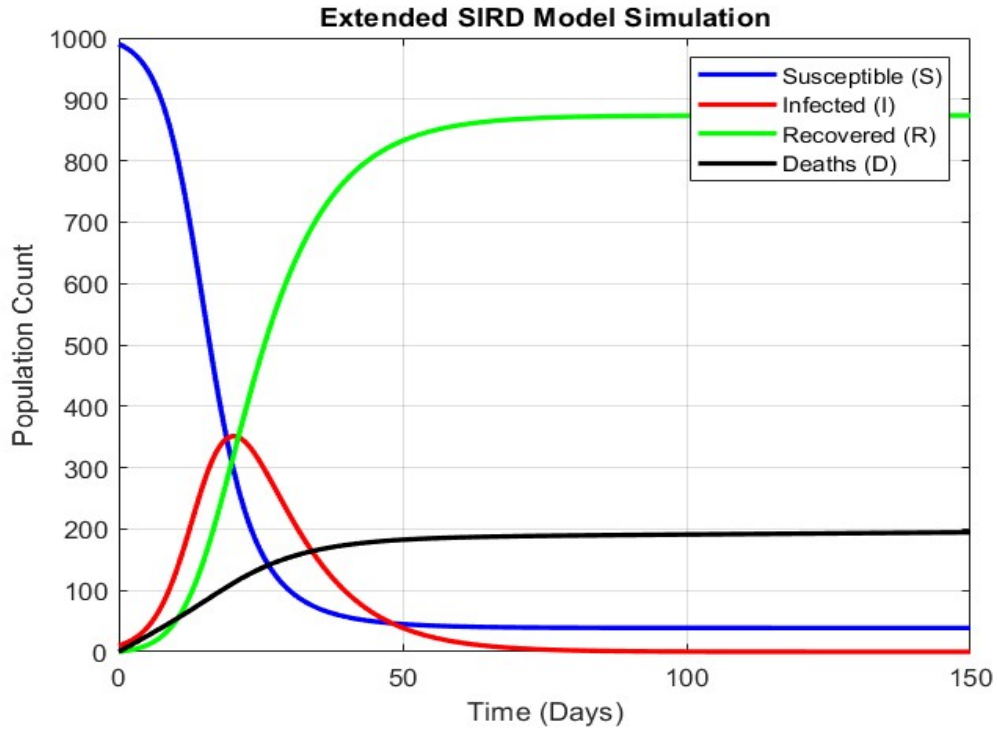

Figure -3 Numerical simulation of the SIRD model when $\beta = 0.4$, $\gamma = 0.1$, $\mu = 0.01$, $\lambda = 0.005$, $\alpha = 0.002$, t=150.

3.1 Basic Reproduction Number (R_0)

The basic reproduction number R_0 is introduced to describe the transmission potential of the infection within the proposed model [10]. It represents the expected number of secondary infections generated by a single infected individual. This analysis assumes that the entire population is initially susceptible. Under this assumption, if If $R_0 > 1$, the infection is able to spread through the population, whereas if $R_0 < 1$, the disease gradually disappears.

Let

$$s = \frac{S}{N}, \quad i = \frac{I}{N}, r = \frac{R}{N}, \text{ and } d = \frac{D}{N}$$

denote the normalized state variables, where N is the total population. The infection equation becomes:

$$\frac{di}{dt} = \beta si - \gamma i - \mu i \tag{9}$$

At the onset of the epidemic, the entire population is assumed susceptible, so $s \approx 1$. Thus, the equation reduces to:

$$\frac{di}{dt} = (\beta - \gamma - \mu)i \tag{10}$$

The epidemic grows $\left(\frac{di}{dt} > 0\right)$ if and only if $\beta > \gamma + \mu$. Dividing both sides by $(\gamma + \mu)$, the basic reproduction number is defined as [10]:

$$R_0 = \frac{\beta}{(\gamma + \mu)} \tag{11}$$

The mortality compartment D has no effect on R_0. Deaths are recorded in D without being fed back into the dynamics of S, I, or R. Thus, D acts only as a counter and plays no role in shaping transmission. Substituting the model parameters ($\beta = 0.4, \gamma = 0.1, \mu = 0.01$):

$$R_0 = \frac{0.4}{(0.1 + 0.01)} = \frac{0.4}{0.11} \approx 3.64 \tag{12}$$

The values of R_0 that have been computed are greater than 1, which means that the epidemic spreads under those conditions. The reported estimates of COVID-19 place pooled R_0 between 2.81 and 3.82 [11], and the amount found fits this range. The model captures real-world transmission dynamics. In SIRD-based frameworks, this threshold parameter was identified by Hussien et al. as the key quantity that governs epidemic behavior [10].

3.2 Stability Analysis

Local stability of the equilibrium points is examined to understand the qualitative behavior of the model. Two outcomes are possible: the system either converges to a disease-free state, or endemic transmission is sustained. Which outcome occurs depends on the model parameters [11].

When all equations equal zero, one steady state emerges with biological meaning the Disease-Free Equilibrium (DFE):

$$E_0 = (s*, i*, r*) = (1, 0, 0) \tag{13}$$

At E_0, the infected compartment is empty. No births or immigration are included, so a state where infection persists has no place in this model. The Jacobean matrix is then evaluated at (1, 0, 0):

$$J^* = \begin{vmatrix} 0 & -\beta & 0 \\ 0 & \beta - \gamma - \mu & 0 \\ 0 & \gamma & 0 \end{vmatrix} \tag{14}$$

Because J^* is upper triangular, eigenvalues are read directly from the diagonal [11]:

$$\lambda_1 = 0, \quad \lambda_2 = \beta - \gamma - \mu, \quad \lambda_3 = 0$$

Stability is controlled by λ_2. A negative value means the DFE holds. This condition is met only when $R_0 < 1$. Substituting the parameters gives $\lambda_2 = 0.29$. The DFE is therefore unstable. Epidemic spread is confirmed by this result, and agreement with Figure 3 is observed.

3.3 Comparison with Existing Models

Three models are placed side by side in this section. The classical SIRD model and the framework built by Giordano et al. in 2020 [9], serve as the reference points.

- The classical SIRD model divides the population into four groups: S, I, R, and D. Death from infection is tracked using a fixed rate μ. No adjustment is made when hospitals become overloaded. Indirect deaths those caused by delayed care or shortage of resources are not captured anywhere in the model [5].

- Eight compartments were used by Giordano et al. in 2020, to build a richer picture of COVID-19 spread [9]. Disease severity and the effect of diagnosis on transmission are both addressed. However, no link is drawn between hospital capacity and death rates. A large set of parameters is needed, many of which are hard to pin down from real data.

- The model presented here keeps the four-compartment layout of SIRD. One addition is made to the death equation:

$$\frac{dD}{dt} = \mu I(t) + \frac{\lambda\big(S(t) + I(t)\big)}{\big(1 + \alpha R(t)\big)} \tag{15}$$

- Two death sources are separated by this term. Direct deaths come from infection itself. Indirect deaths shrink as more people recover, since pressure on health services drops. Neither the classical SIRD nor the Giordano model contains this mechanism.

3.4 Model Validation

The main contribution of this paper is the theoretical formulation of a new nonlinear saturation mechanism for indirect mortality. The basic reproduction number (R_0) of this model is 3.64, which falls within the reported empirical range for COVID-19, which is (2.81-3.82) [11], confirming that our choice of parameters is not random and is consistent with published epidemiological data. The coefficients (λ, α) that determine indirect mortality represent a new mechanism for which there are currently no experimental estimates in previous studies. Comparison with real data is a priority for future work.

4. Conclusions

In this study, an improved version of the SIRD model was developed with the aim of characterizing mortality dynamics with a higher degree of accuracy. This development is based on incorporating the impact of pressure on health infrastructure into the model structure. Unlike traditional models that assume constant mortality rates, the proposed framework relies on a nonlinear regulatory mechanism. We can utilize this mechanism to present the constraints on medical resources, while underscoring the beneficial effect of individual recovery in counteracting this strain. Numerical results showed that including health stress as a component for mortality during the epidemic provides more accurate representations of its path. The model becomes able to distinguish between direct deaths from the disease and indirect deaths from exceeding the absorptive capacity of the health system. The simulation also indicates that a higher percentage of recoveries contributes to the reduction in burden on public health and other systems. In such cases, such reductions in secondary mortality and in other indicators of disease contribute to the overall improvement of disease and the epidemiology indicators. Overall, the developed model gives a richer analytical structure to analyze the dissemination of infectious diseases in low-resource settings. For future studies examining dynamic stability, identification of stable cases, and evaluation of the effects of different health intervention

strategies on epidemics, this study should provide the foundation on which the following works are intended to build.

Acknowledgement: The author would like to express sincere gratitude to Dr. Ali Hussein Shuaa for his valuable guidance, insightful comments, and continuous support throughout this research.

Conflict of Interest: The authors declare that they have no conflicts of interest.

References

[1] H. W. Hethcote, "The mathematics of infectious diseases," *SIAM Review*, vol. 42, no. 4, pp. 599–653, 2000.

[2] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics," *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character*, vol. 115, no. 772, pp. 700–721, 1927.

[3] F. Brauer and C. Castillo-Chavez, *Mathematical Models in Population Biology and Epidemiology*, 2nd ed. New York, NY: Springer, 2012.

[4] World Health Organization, *Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19)*, 2020. Available: https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf. Accessed: Mar. 26, 2026.

[5] P. Sandhu, A. B. Shah, F. B. Ahmad, J. Kerr, H. B. Demeke, E. Graeden, S. Marks, H. Clark, J. M. Bombard, M. Bolduc and K. Hatfield-Timajchy, "Emergency department and intensive care unit overcrowding and ventilator shortages in US hospitals during the COVID-19 pandemic, 2020–2021," *Public Health Reports*, vol. 137, no. 4, pp. 796–802, 2022.

[6] E. Mathieu, H. Ritchie, L. Rodés-Guirao, C. Appel, D. Gavrilov, C. Giattino, J. Hasell, B. Macdonald, S. Dattani, D. Beltekian, E. Ortiz-Ospina and M. Roser, *COVID-19 Pandemic*, OurWorldInData.org, 2020. [Online]. Available: https://ourworldindata.org/coronavirus

[7] R. Li, S. Pei, B. Chen, Y. Song, T. Zhang, W. Yang and J. Shaman, "Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV-2)," *Science*, vol. 368, no. 6490, pp. 489–493, 2020.

[8] M. Chinazzi, J. T. Davis, M. Ajelli, C. Gioannini, M. Litvinova, S. Merler, A. Pastore y Piontti, K. Mu, L. Rossi, K. Sun and C. Viboud, "The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak," *Science*, vol. 368, no. 6489, pp. 395–400, 2020.

[9] G. Giordano, F. Blanchini, R. Bruno, P. Colaneri, A. Di Filippo, A. Di Matteo and M. Colaneri, "Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy," *Nature Medicine*, vol. 26, no. 6, pp. 855–860, 2020.

[10] H. H. Hussien, K. R. Genawi, N. H. Hagabdulla and K. M. Ahmed, "Understanding the Basic Reproduction Number (R_0): Calculation, Applications, and Limitations in Epidemiology," *Open Journal of Epidemiology*, vol. 15, no. 2, pp. 272–295, 2025.

[11] Y. Alimohamadi, M. Taghdir and M. Sepandi, "Estimate of the basic reproduction number for COVID-19: a systematic review and meta-analysis," *Journal of Preventive Medicine and Public Health*, vol. 53, no. 3, p. 151, 2020.