

Review

Mathematical Modeling and the Use of Network Models as Epidemiological Tools

Javier Cifuentes-Faura , Ursula Faura-Martínez  and Matilde Lafuente-Lechuga 

Faculty of Economics and Business, University of Murcia, 30100 Murcia, Spain

* Correspondence: javier.cifuentes@um.es

Abstract: Mathematical modeling has served as an epidemiological tool to enhance the modeling efforts of the social and economic impacts of the pandemic. This article reviews epidemiological network models, which are conceived as a flexible way of representing objects and their relationships. Many studies have used these models over the years, and they have also been used to explain COVID-19. Based on the information provided by the Web of Science database, exploratory, descriptive research based on the techniques and tools of bibliometric analysis of scientific production on epidemiological network models was carried out. The epidemiological models used in the papers are diverse, highlighting those using the SIS (Susceptible-Infected-Susceptible), SIR (Susceptible-Infected-Recovered) and SEIR (Susceptible-Exposed-Infected-Removed) models. No model can perfectly predict the future, but they provide a sufficiently accurate approximation for policy makers to determine the actions needed to curb the pandemic. This review will allow any researcher or specialist in epidemiological modeling to know the evolution and development of related work on this topic.

Keywords: mathematical modeling; epidemiological network models; pandemic; review; COVID-19

MSC: 90-00



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1. Introduction

Faced with a global pandemic, many researchers have tried to respond to all of the issues and the social and economic impacts generated. They tend to collect data and develop models, algorithms or analytical tools that allow for a deeper understanding of the underlying factors [1–3]. In this way, results can be obtained that help decision makers to enact certain policies over others [4].

Mathematical modeling has served as an epidemiological tool to explain this type of phenomenon. From SARS to, more recently, COVID-19 or SARS-CoV-2, many studies have attempted to provide answers to the evolution and characteristics of epidemics [5–8]. These mathematical models allow researchers to understand the dynamics of epidemics and are useful for determining the strategies to follow and control policies. They also serve to better understand the system and prevent future epidemics, determine prevalence and incidence, and make objective decisions.

Building mathematical models for the study of a pandemic caused by an infectious disease will help to understand the spread of an infectious disease through a population under different scenarios, extract properties and characteristics of the relationships between the variables analyzed or predict the consequences of introducing specific changes.

There are two types of mathematical models: deterministic and stochastic models. In a deterministic model, it is possible to control all of the factors involved in the analysis of the phenomenon and to accurately predict its results. In a stochastic model, it is not possible to control all of the factors that intervene in the progress of the phenomenon, so the results are neither unique nor exact, but each outcome will occur with a certain probability. In the

epidemiological context, in a deterministic mathematical model, a single individual can cause a generalized epidemic, whereas in a stochastic mathematical model, it is possible that the disease does not evolve in the population and the epidemic dies out [9,10].

Epidemiological models usually assume that individuals are in one of several possible states, and depending on that state, the population is categorized into different categories: susceptible (S), infected (I) or removed (R) individuals, among others. The most important models are SI, SIS, SIR and SEIR, which can be represented by a deterministic or stochastic model.

With the recent COVID-19 pandemic, attempts have also been made to explain the structure of the underlying virus, how it spreads, prevention measures, containment and diagnostic protocols, vaccines and the global impact [11–13]. The vast majority of papers have employed the SIR model to predict the spread of the pandemic or the number of COVID-19 cases [14–18]. Other papers employed the SEIR model [19–21], and others were also found that refer to the SI [22,23] or SIS model [24,25].

The use of mathematical models for the study of infectious diseases should be used with caution [9,10], since it is difficult to understand a complex problem without performing minimum modeling, although it is not always possible to model the totality of real situations.

In this article, given the growing interest in explaining epidemics through mathematical models, we focus on some specific models, namely, network models, that take into account the interaction between individuals, which helps to improve the understanding of classical epidemiological models. Many studies have used these models, which have also been used to explain COVID-19. This review will allow any researcher or specialist in epidemiological models to learn about the evolution and development of related work on this subject.

Main Epidemiological Models

Discrete-time models or difference equations are used to formulate some epidemic models, such as SI, SIS, SIR or SEIR. Continuous approximations of these models are more frequently used in modeling situations as a consequence of their mathematical manageability. Difference equations do not perform as well as their continuous approximations [26,27].

Denoting by S the susceptible population (individuals without immunity to the infectious agent who can be infected) and by I the infected population, the discrete-time epidemic SI model has the following formulation [26]:

$$\begin{aligned} S_{n+1} &= S_n \left(1 - \frac{\alpha \Delta t}{N} I_n \right) \\ I_{n+1} &= I_n \left(1 + \frac{\alpha \Delta t}{N} S_n \right) \end{aligned} \tag{1}$$

where $\alpha > 0$ is the contact rate (the average number of individuals with which an infectious individual establishes sufficient contact to pass the infection during a unit time interval, Δt), N is the total population size, and the subscript n represents time $n\Delta t > 0$; S_n is the size of the susceptible subpopulation in time $n\Delta t > 0$. In addition, $S_0 > 0$ and $I_0 > 0$, satisfying $S_0 + I_0 = N$. In order to ensure that the solutions of (1) are positive, a necessary and sufficient condition is that $\alpha \Delta t \leq 1$.

The analogous differential system has the following form:

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\alpha}{N} SI \\ \frac{dI}{dt} &= \frac{\alpha}{N} SI \end{aligned} \tag{2}$$

with positive initial conditions satisfying $S(0) + I(0) = N$.

The discrete model for the standard SIR model divides the population into three compartments: susceptible, infectious and recovery, although the latter stage may include death or effective isolation [15,28]. The SIR model aims to predict the number of people

who are susceptible to infection, actively infected or have recovered from infection at a given point in time. The difference equations have the following expression:

$$\begin{aligned} S_{n+1} &= S_n \left(1 - \frac{\alpha \Delta t}{N} I_n\right) \\ I_{n+1} &= I_n \left(1 - \gamma \Delta t + \frac{\alpha \Delta t}{N} S_n\right) \\ R_{n+1} &= R_n + \gamma \Delta t I_n \end{aligned} \tag{3}$$

where $\gamma > 0$ is the probability that an infectious agent is eliminated from the infection process during a unit time interval. Similar to the SIR model, the initial conditions are $S_0 > 0$, $I_0 > 0$, and $R_0 \geq 0$, verifying $S_0 + I_0 + R_0 = N$.

The continuous version of this SIR model has the following form:

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\alpha}{N} SI \\ \frac{dI}{dt} &= I \left(\frac{\alpha}{N} S - \gamma\right) \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{4}$$

with positive initial conditions satisfying $S(0) + I(0) + R(0) = N$.

The SIS model is another of the most widely used mathematical epidemiological models to emulate epidemics [29–31], which is obtained by iterative computation. It is assumed that in a closed society, there are no deaths, no births and no population migration, and infected individuals are not reinfected after recovery. Clearly, these assumptions are valid for a limited time interval. In the SI model, over the course of the epidemic, almost everyone eventually becomes infected. In the SIS model, each individual belongs either to the susceptible set (S) or to the infected set (I).

Some infections do not confer immunity. These infections do not have a recovery state, and individuals become susceptible again after infection. This type of disease can be explained by the SIS model. Individuals who are cured do not develop permanent immunity as in the SIR model but are immediately susceptible to the disease. The SIS model moves individuals from the infectious class to the susceptible class, and therefore, there is no eliminated class. The model adopts the following expression:

$$\begin{aligned} S_{n+1} &= S_n \left(1 - \frac{\alpha \Delta t}{N} I_n\right) + \gamma \Delta t I_n \\ I_{n+1} &= I_n \left(1 - \gamma \Delta t + \frac{\alpha \Delta t}{N} S_n\right) \end{aligned} \tag{5}$$

with positive initial conditions $S_0 > 0$ and $I_0 > 0$, satisfying $S_0 + I_0 = N$.

The population size remains constant, and the solutions are positive for all initial conditions if and only if it is verified that $\gamma \Delta t \leq 1$ and $\alpha \Delta t < (1 + \sqrt{\gamma \Delta t})^2$.

The continuous SIS model does not exhibit periodicity.

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\alpha}{N} SI + \gamma I \\ \frac{dI}{dt} &= I \left(\frac{\alpha}{N} S - \gamma\right) \end{aligned} \tag{6}$$

where $S(0) + I(0) = N$.

The three models SI, SIS and SIR can be derived as special cases of the SIRS model. The deterministic development of this model can be found in Mena-Lorcat [32].

These models are quite simple, so they are easy to calculate, although sometimes they oversimplify complex epidemiological processes. For example, if one wants to take into account the time between the moment when an individual is exposed to a disease and the moment when that individual becomes infected and contagious (as in the case of coronavirus 19 disease), it is necessary to make some modifications, such as that offered by the SEIR model (“E” denotes exposed but not yet contagious) [20,33].

The continuous-time equations of this model are as follows [19]:

$$\begin{aligned} \frac{dS}{dt} &= -\beta S(I + qE)/N \\ \frac{dS}{dt} &= (-\beta S(I + qE)/N) - (E/\delta) \\ \frac{dI}{dt} &= (E/\delta) - (I/\gamma) \\ \frac{dR}{dt} &= I/\gamma \end{aligned} \quad (7)$$

The parameter β is the average number of contacts per person per time, multiplied by the probability of transmission of the disease, q , through contact between a susceptible individual and an individual who has the virus.

The transfer rate of the stage exposed to infection is a fraction of the number of exposed individuals, where δ is the average time it takes an exposed individual to become infected. The recovery rate is a fraction, $1/\gamma$, of the infected population, where γ is the average time it takes a person to recover from the infection.

Generalizations have been made based on these models, including births, deaths, migration [34–36], vaccination and treatment [37–39]. More complex models have been developed by taking into consideration spatial aspects to understand how the mobility of population migration affects the spatial distribution of the disease [40] and that contacts are made through individual interactions. Network models describe a population and its interactions, with the nodes (vertices) of the network representing individuals and the edges (links) representing interactions between individuals that could lead to transmission of infection [41–44]. Disease dynamics in networks can be determined by modifications of the equations of the main epidemiological models [45,46].

2. Materials and Methods

Based on the information provided by the WoS (Web of Science) database, an exploratory, descriptive investigation was carried out using the techniques and tools of bibliometric analysis of scientific production on epidemiological network models, describing how certain fields of research are related and how they evolve over time. This is one of the world's most important databases of bibliographic references and citations of periodicals that provides analysis tools to evaluate the scientific quality of publications. It also offers one of the best-known quality indicators, the Journal Citation Reports (JCR), which is used by organizations that evaluate research activity. The highest-quality journals in each field are indexed in WoS [47].

Given the importance of this topic, no time restrictions were included, obtaining all documents up to 30 October 2021. Several searches were performed combining different keywords: “model”, “network”, “epidemic”, “analysis”, “control” and “network model”. Some of these combinations resulted in a very small number of papers, and others included many papers that were unrelated to this research. The following combination of keywords was chosen: “network model”, “epidemic” and “analysis”, resulting in a total of 244 papers. We selected papers that were articles, including review articles. The result was $n = 212$ papers.

A preliminary reading of the abstract was performed initially, followed by a more exhaustive reading of each article to verify that it met the established inclusion criteria. For example, we discarded papers that, although they have these keywords, use them only to globally situate their analysis, such as analyzing fungicide performance. After this filtering, $n = 145$ documents were obtained.

The distribution of the keywords of the analyzed papers is shown in Figure 1. The most important keywords are, in addition to those used in the WoS search, “disease”, “transmission”, “spreading” and “modelling”.

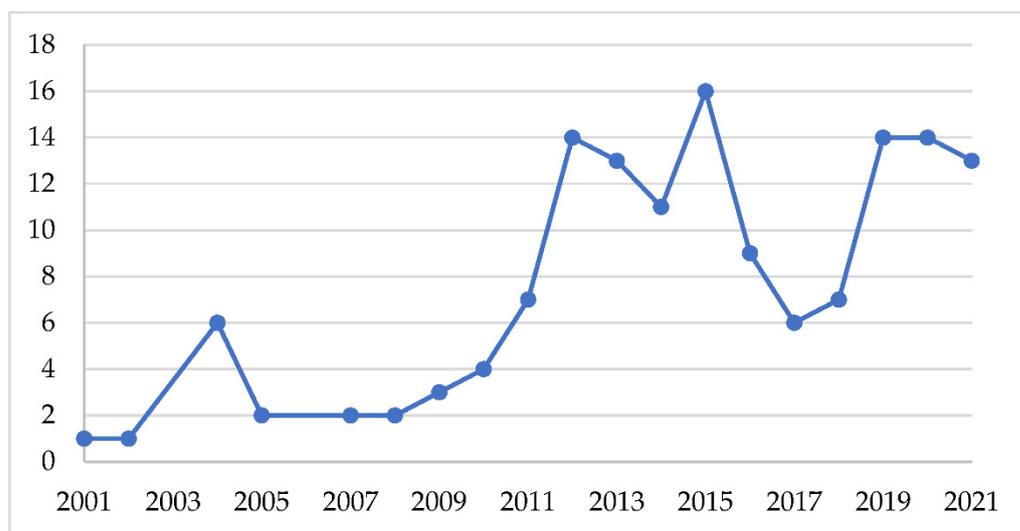


Figure 2. Number of publications per year (1993–2020).

Table 1. Countries of the authors of the publications.

Countries	Percentage
People R. China	25.51
United States	23.98
England	7.65
Canada	6.63
India	4.08
Italy	4.08
Australia	3.57
Switzerland	3.06
Japan	2.04
Others	19.39

Table 2. Authors with the most published articles.

Author	Number of Publications
Jin, Zhen	5
Bertuzzo, Enrico	4
Casagrandi, Renato	4
Craft, Meggan E.	4
Gatto, Marino	4
Kiss, Istvan Z.	4
Mari, Lorenzo	4
Rinaldo, Andrea	4
Sun, Gui-Quan	4
Wang, Bing	4
Wang, Y.	4

The journals in which the papers analyzed were published are varied (Table 3). A total of 87 different journals were found. *PLoS ONE* stands out with eight publications. This journal is multidisciplinary and accepts research from different areas, such as science, engineering, medicine, and the related social sciences and humanities. With five papers each, we find the *Journal of Mathematical Biology*, whose main aim is to identify and work with mathematical models derived from biological knowledge; the *Journal of The Royal Society Interface*, which publishes research that applies, among other disciplines, mathematics and physics to biological and medical sciences; and the *Journal of Theoretical Biology and Physical Review*, in which priority is given to biological aspects over mathematical ones.

Most of the journals are located in the first quartiles, and many of them belong to quartile 1, such as *Applied Mathematical Modelling* or *Advances in Difference Equations*, which shows the importance of the topic dealt with in this work for journals that are very well positioned in the JCR ranking.

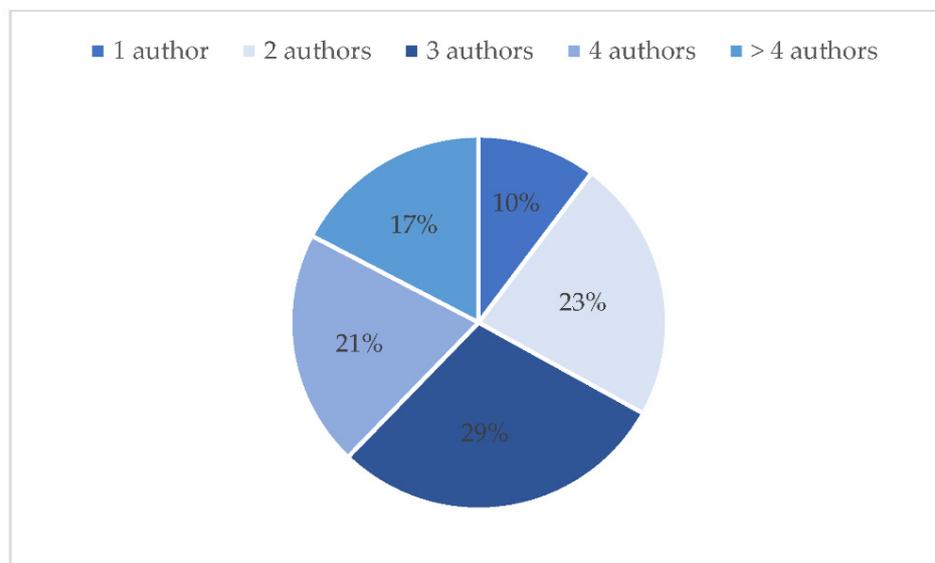


Figure 3. Percentage of papers by number of authors.

Table 3. Overview of the most productive journals of our dataset.

Journal	N° Articles	Quartile	Impact Factor 2020
<i>PLoS ONE</i>	8	Q ₂	3.240
<i>Journal of Mathematical Biology</i>	5	Q ₂	2.259
<i>Journal of The Royal Society Interface</i>	5	Q ₂	4.118
<i>Journal of Theoretical Biology</i>	5	Q ₂	2.691
<i>Physical Review E</i>	5	Q ₁	2.529
<i>Applied Mathematical Modelling</i>	4	Q ₁	5.129
<i>Mathematical Biosciences</i>	4	Q ₃	2.144
<i>Physica A: Statistical Mechanics and its Applications</i>	4	Q ₂	3.263
<i>Advances in Difference Equations</i>	3	Q ₁	2.803
<i>Complexity</i>	3	Q ₂	2.833
<i>Computational Biology</i>	3	Q ₁	4.475
<i>Preventive Veterinary Medicine</i>	3	Q ₁	2.670
<i>Proceedings of the Royal Society B: Biological Sciences</i>	3	Q ₁	5.349

Table 4 shows the research areas of the different journals. As can be seen, areas that stand out are Mathematics with 14.83% of papers and Science Technology Other Topics with 13.40%, followed by Mathematical Computational Biology, Life Sciences Biomedicine Other Topics and Physics. The research coming from these areas includes new methods and mathematical models, both from theoretical and applied points of view, which address calculating the probability of transmission of an epidemic, how to control the transmission, control measures, prevention, etc.

3.3. Epidemiological Models Developed

The epidemiological models used in the studies are diverse, with the SIS (Susceptible-Infected-Susceptible) model serving as the basis for 36 studies, the SIR (Susceptible-Infected-Recovered) with 27 and the SEIR (Susceptible-Exposed-Infective-Removed) with 12. Other less commonly used models are SEIQR (Susceptible, Exposed, Infected, In Quar-

antine and Recovered), SEAIR (Susceptible-Exposed-Asymptomatic-Infected-Symptomatic-Recovered), etc.

Table 4. Research areas of publications.

Research Areas	Percentage
Mathematics	14.83
Science Technology Other Topics	13.40
Mathematical Computational Biology	12.92
Life Sciences Biomedicine Other Topics	10.05
Physics	10.05
Computer Science	7.66
Engineering	7.18
Infectious Diseases	5.74
Environmental Sciences Ecology	3.83
Mechanics	3.35
Public Environmental Occupational Health	2.87
Biochemistry Molecular Biology	2.39
Veterinary Sciences	2.39
Telecommunications	1.91
Evolutionary Biology	1.44

Regarding the type of epidemic analyzed in the different studies, these have been classified into three areas: epidemics of infectious diseases and invasive species, epidemics transmitted through social networks (Internet, rumor spreading, etc.) and epidemics in general (Table 5). It should be noted that some of the models of rumor dissemination are different from those of epidemiology. In addition to using SIR models [48,49], there are the SBD models [50], SIQRS models (S—susceptible class; I—infective class; Q—quarantined class; R—removed class) [51], I2S2R models [52,53], DSIR models [54] and ILSR models [55], the most widely used being the sender-centric Independent Cascade (IC), in which each active node influences its inactive neighbors independently with given probabilities, and the receiver-centric Linear Threshold (LT), in which an inactive node is influenced by its active neighbors if the total weight exceeds a given threshold.

Table 5. Percentage of publications by field of application.

Scope	% of Publications
Epidemics of infectious diseases	57.93
Epidemics in general	32.41
Epidemics in social networks	9.66

Publications analyzing the spread of infectious disease epidemics are predominant, accounting for nearly 60% of the papers analyzed. The lowest percentage, 9.66%, corresponds to articles whose epidemiological study focused on social networks or the spread of rumors.

Of the papers published up to the time of data collection in October 2021, there are 21 research papers focusing on the current pandemic caused by the COVID-19 virus. Several more complex models have been developed to study the dynamics of the disease. Some take into account the perception of risk and the cumulative number of cases [56]. In the work of Giordano et al. [57], which extends the classical SIR model, they predict the evolution of epidemics by evaluating the impact of different strategies to contain the spread of infection, including blocking, social distancing and contact tracing. Purkayastha et al. [58], through five epidemiological models, forecast and evaluated the course of the pandemic in India.

Of the publications analyzed, 42.76% are papers in which theoretical models were developed with extensive mathematical demonstrations based on the epidemiological models used in the existing literature, 46.21% of the cases are theoretical but do not include

demonstrations, and only 11% of the articles are applications of the epidemiological models to concrete cases. To forecast the evolution of an epidemic, model approaches include the simulation of the dynamics of disease transmission and recovery or the empirical fitting of data trends.

Taking into account the epidemiological scope of the publications, Figure 4 shows that epidemiological models used perform exhaustive mathematical demonstrations are more frequently in articles on epidemics in general than in the rest, and those based on practical exercises are more frequently on epidemics of infectious diseases.

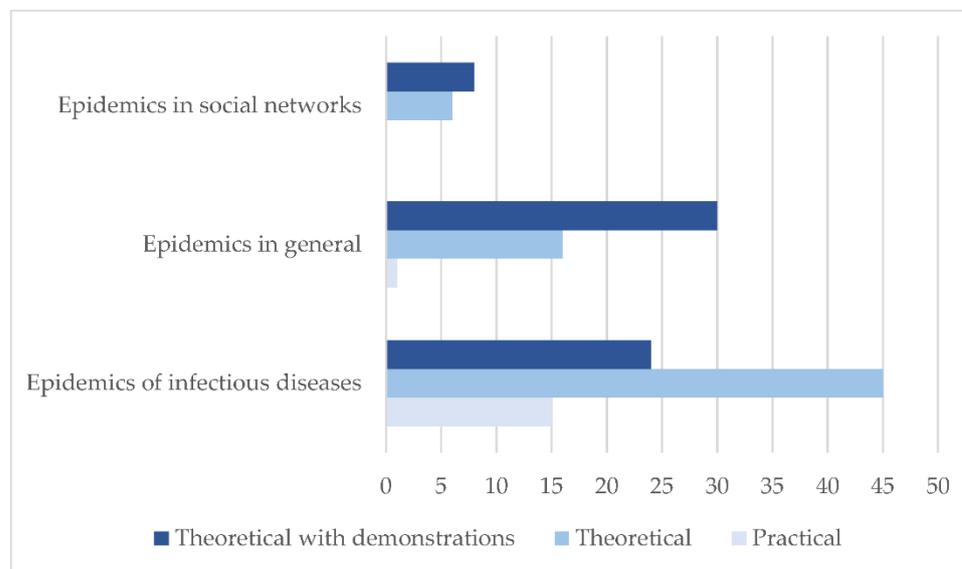


Figure 4. Articles by field and type of work.

3.4. Analysis of Citations

The citations of a work show its importance in the scientific community and its contribution, although this depends on other factors that can condition the dissemination of the work, such as the prestige of the author, the topicality of the subject or the journal in which it is published. The 145 papers collected have a total of 2416 citations in the period analyzed. This number has been growing exponentially, as shown in Figure 5.

The most cited article of those examined (Table 6), with an average annual citation count of 30.2, is titled “Spread of Zika virus in the Americas” and was written in 2017 by Zhang et al. [59], who performed a simulation of the spatial and temporal dynamics of the Zika virus epidemic in the Americas, providing probability distributions for the timing and location of Zika introduction in Brazil, the estimated attack rate, the timing of the epidemic in the affected countries and the projected number of newborns born to Zika-infected women. Next, there is the work conducted by Craft [56] in 2015: “Infectious disease transmission and contact networks in wildlife and livestock”, which reaches, on average, 20 citations per year and in which transmission dynamics in wildlife and livestock populations are studied, analyzing the mismatch between the contact networks measured in animal behavior and the relevant parasites in those networks.

Among the most cited authors in the database, taking into account the citations of the articles, the number of years that the article has been in the scientific community (year of publication) and the number of publications, those that stand out are Craft Meggan, Associate Professor, Department of Ecology, Evolution, and Behavior, University of Minnesota; Bertuzzo Enrico, University Cà Foscari Venice; Mari Lorenzo, Politecnico di Milano; Rinaldo Andrea, Professor of Hydrology and Water Resources and Università di Padova (IT); and Jin Zheng, Postdoctoral Researcher, Argonne National Lab of the University of Singapore.

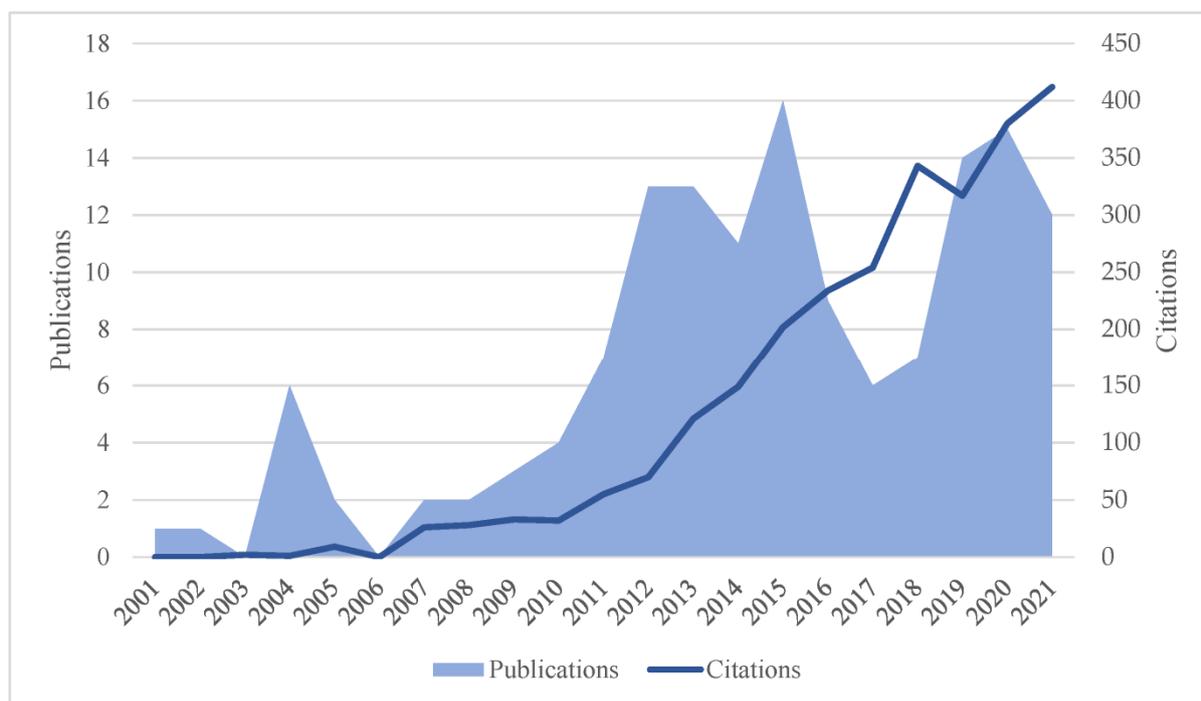


Figure 5. Time evolution of publications and their citations.

Table 6. Most cited articles.

	Total Number of Citations	Average Number of Citations per Year
Spread of Zika virus in the Americas [59]	151	30.2
Infectious disease transmission and contact networks in wildlife and livestock [60]	140	20.0
Global analysis of an SIS model with an infective vector on complex networks [61]	111	11.1
Integrating association data and disease dynamics in a social ungulate: bovine tuberculosis in African buffalo in the Kruger National Park [62]	98	5.4
Exogenous re-infection and the dynamics of tuberculosis epidemics: local effects in a network model of transmission [63]	85	5.7
Networks and the ecology of parasite transmission: A framework for wildlife parasitology [64]	82	9.1
Epidemic spreading in time-varying community networks [65]	66	8.3
Stability analysis of VEISV propagation modeling for network worm attack [66]	66	6.6
Distinguishing epidemic waves from disease spillover in a wildlife population [67]	63	4.8
Impact of media coverage on epidemic spreading in complex networks [68]	60	6.7

The papers are mainly cited in the 12 journals listed in Table 7, although in total, they are found in 1060 journals. *PLoS ONE* has 5.17% of the citations, *Scientific Reports* has 3.49% and *Physica A: Statistical Mechanics* has 3.36%.

Table 7. Most cited journals.

Journal	Percentage of Citations
<i>PLoS ONE</i>	5.17
<i>Scientific Reports</i>	3.49
<i>Physica A: Statistical Mechanics and its Applications</i>	3.36
<i>Physical Review E</i>	1.94
<i>Journal of Theoretical Biology</i>	1.81
<i>PLoS Computational Biology</i>	1.64
<i>PLoS Neglected Tropical Diseases</i>	1.29
<i>Chaos Solitons Fractals</i>	1.21

Table 7. Cont.

Journal	Percentage of Citations
<i>Epidemics</i>	1.16
<i>Applied Mathematics and Computation</i>	1.12
<i>Journal of The Royal Society Interface</i>	1.12
<i>Preventive Veterinary Medicine</i>	1.12

4. Conclusions

Modeling the dynamics of epidemics is essential to project their evolution through simulation and provide useful information for designing disease control and prevention strategies. The models help to determine the role of factors such as the mechanism of infection, total population variation and population movement in disease transmission dynamics and the role of the disease in the transmission of the disease. In recent years, this has become even more necessary due to the COVID-19 epidemic in order to adequately plan medical care.

Using the keywords “network model”, “epidemic” and “analysis”, we searched the WoS database to discover what research has been published on network epidemiological modeling, who the most relevant authors are and in which journals it is published. After an exhaustive review of each of the papers obtained in this search, the number of papers selected was 145, published from 2001 to October 2021.

It was found that great efforts have been made to consider the dynamics of diffusion in epidemiology, predominantly using the Susceptible-Infected-Susceptible (SIS) and Susceptible-Infected-Removed (SIR) models. In addition, simple models such as SI and more complex models such as SEIR, which also includes the “E” for Exposed, have been used.

The number of publications on the subject has grown in recent years. There are 491 authors with JCR publications in this field of research from various disciplines, and almost 50% come from People R. China and the United States. The author with the most articles is Zhen Jin from North University China. The journals in which most articles are published are very well placed in the rankings, belonging to the Q₁ and Q₂ quartiles. *PLoS ONE* stands out among all of them.

A large number of papers focus on diseases involved in infectious disease epidemics, and just over 14% focus on the current pandemic caused by the SARS-CoV-2 virus, representing almost 50% of the papers in 2020 and 2021.

The most cited papers are Zhang et al. [55], who performed a simulation of the spatial and temporal dynamics of the Zika virus epidemic in the Americas, and Craft (2015), in which transmission dynamics in wild animal and livestock populations are studied. *PLoS ONE* again stands out as the journal with the highest number of citations for these papers.

The epidemiological models used in the studies are diverse, with the SIS model serving as the basis for 36 studies, the SIR studied in 27 and the SEIR used in 12. Other less used models are SEIQR (Susceptible, Exposed, Infected, Quarantined and Recovered), SEAIR (Susceptible-Exposed-Asymptotically Infected-Symptomatically Infected-Recovered), etc.

As indicated, the use of epidemiological models in the related literature has resulted in very relevant publications, which suggest that factors such as the mechanism of infection, total population variation and population movement play an important role in disease transmission dynamics and therefore provide useful information for strategies designed for disease control and prevention.

Simpler models may provide less valid forecasts because they cannot control for some time-varying features of epidemic spread. A more complex model may provide a more detailed description of the epidemic, but the results may be sensitive to changes in the stated assumptions and depend on external preliminary estimates of epidemic and transmission characteristics, such as incubation duration and infectious periods. This determines that no

one model stands out more than another. Models can be useful tools, but they should not be over-interpreted. In addition, while none of them can perfectly predict the future, they are capable of providing a sufficiently accurate approximation to be useful in determining public policy so that the competent authorities can dictate the actions necessary to curb the epidemic.

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References

- Brauer, F.; Castillo-Chavez, C.; Feng, Z. *Mathematical Models in Epidemiology*; Springer: New York, NY, USA, 2019; Volume 32.
- Tang, Y.; Serdan, T.D.A.; Masi, L.N.; Tang, S.; Gorjao, R.; Hirabara, S.M. Epidemiology of COVID-19 in Brazil: Using a mathematical model to estimate the outbreak peak and temporal evolution. *Emerg. Microbes Infect.* **2020**, *9*, 1453–1456. [[CrossRef](#)] [[PubMed](#)]
- Bekiros, S.; Kouloumpou, D. SBDiEM: A new mathematical model of infectious disease dynamics. *Chaos Solitons Fractals* **2020**, *136*, 109828. [[CrossRef](#)] [[PubMed](#)]
- Xue, L.; Jing, S.; Miller, J.C.; Sun, W.; Li, H.; Estrada-Franco, J.G.; Hyman, J.M.; Zhu, H. A data-driven network model for the emerging COVID-19 epidemics in Wuhan, Toronto and Italy. *Math. Biosci.* **2020**, *326*, 108391. [[CrossRef](#)]
- Panovska-Griffiths, J. Can mathematical modelling solve the current Covid-19 crisis? *BMC Public Health* **2020**, *20*, 551. [[CrossRef](#)]
- Adiga, A.; Dubhashi, D.; Lewis, B.; Marathe, M.; Venkatramanan, S.; Vullikanti, A. Mathematical models for covid-19 pandemic: A comparative analysis. *J. Indian Inst. Sci.* **2020**, *100*, 793–807. [[CrossRef](#)] [[PubMed](#)]
- Xiao, S.; Cheng, G.; Yang, R.; Zhang, Y.; Lin, Y.; Ding, Y. Prediction on the number of confirmed Covid-19 with the FUDAN-CCDC mathematical model and its epidemiology, clinical manifestations, and prevention and treatment effects. *Results Phys.* **2021**, *20*, 103618. [[CrossRef](#)]
- Olivares, A.; Staffetti, E. Uncertainty quantification of a mathematical model of COVID-19 transmission dynamics with mass vaccination strategy. *Chaos Solitons Fractals* **2021**, *146*, 110895. [[CrossRef](#)]
- Allen, L.J.; Burgin, A.M. Comparison of deterministic and stochastic SIS and SIR models in discrete time. *Math. Biosci.* **2000**, *163*, 1–33. [[CrossRef](#)]
- Champagne, C.; Cazelles, B. Comparison of stochastic and deterministic frameworks in dengue modelling. *Math. Biosci.* **2019**, *310*, 1–12. [[CrossRef](#)]
- Cifuentes-Faura, J. Factors influencing the COVID-19 mortality rate in the European Union: Importance of medical professionals. *Public Health* **2021**, *200*, 1–3. [[CrossRef](#)]
- Abellán García, A.; Aceituno Nieto, P.; Allende, A.; de Andrés, A.; Arenillas, A.; Bartomeus, F.; Bastolla, U.; Benavides, J.; Cabal, B.; Castillo Belmonte, A.B.; et al. *Una Visión Global de la Pandemia COVID-19: Qué Sabemos y Qué Estamos Investigando Desde el CSIC*; CSIC: Madrid, Spain, 2021.
- Ram, V.; Schaposnik, L.P. A modified age-structured SIR model for COVID-19 type viruses. *Sci. Rep.* **2021**, *11*, 15194. [[CrossRef](#)]
- Chen, Y.C.; Lu, P.E.; Chang, C.S.; Liu, T.H. A time-dependent SIR model for COVID-19 with undetectable infected persons. *IEEE Trans. Netw. Sci. Eng.* **2020**, *7*, 3279–3294. [[CrossRef](#)]
- Cooper, I.; Mondal, A.; Antonopoulos, C.G. A SIR model assumption for the spread of COVID-19 in different communities. *Chaos Solitons Fractals* **2020**, *139*, 110057. [[CrossRef](#)] [[PubMed](#)]
- Kudryashov, N.A.; Chmykhov, M.A.; Vigdorowitsch, M. Analytical features of the SIR model and their applications to COVID-19. *Appl. Math. Model.* **2021**, *90*, 466–473. [[CrossRef](#)] [[PubMed](#)]
- Atkeson, A. On using SIR models to model disease scenarios for COVID-19. *Q. Rev.* **2020**, *41*, 1–35. [[CrossRef](#)]
- Muñoz-Fernández, G.A.; Seoane, J.M.; Seoane-Sepúlveda, J.B. A SIR-type model describing the successive waves of COVID-19. *Chaos Solitons Fractals* **2021**, *144*, 110682. [[CrossRef](#)]
- Rădulescu, A.; Williams, C.; Cavanagh, K. Management strategies in a SEIR-type model of COVID 19 community spread. *Sci. Rep.* **2020**, *10*, 21256. [[CrossRef](#)]

20. He, S.; Peng, Y.; Sun, K. SEIR modeling of the COVID-19 and its dynamics. *Nonlinear Dyn.* **2020**, *101*, 1667–1680. [[CrossRef](#)]
21. Annas, S.; Pratama, M.I.; Rifandi, M.; Sanusi, W.; Side, S. Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia. *Chaos Solitons Fractals* **2020**, *139*, 110072. [[CrossRef](#)]
22. Kosmidis, K.; Macheras, P. A fractal kinetics SI model can explain the dynamics of COVID-19 epidemics. *PLoS ONE* **2020**, *15*, e0237304. [[CrossRef](#)]
23. Demongeot, J.; Griette, Q.; Magal, P. SI epidemic model applied to COVID-19 data in mainland China. *R. Soc. Open Sci.* **2020**, *7*, 201878. [[CrossRef](#)] [[PubMed](#)]
24. Otunuga, O.M. Time-dependent probability distribution for number of infection in a stochastic SIS model: Case study COVID-19. *Chaos Solitons Fractals* **2021**, *147*, 110983. [[CrossRef](#)]
25. Brusset, X.; Davari, M.; Kinra, A.; Torre, D.L. Modelling COVID-19 Ripple Effect and Global Supply Chain Productivity Impacts Using a Reaction-Diffusion Time-Space SIS Model. In *IFIP International Conference on Advances in Production Management Systems*; Springer: Cham, Switzerland, 2021; pp. 3–12.
26. Allen, L.J. Some discrete-time SI, SIR, and SIS epidemic models. *Math. Biosci.* **1994**, *124*, 83–105. [[CrossRef](#)]
27. Allen, L.J.S.; Jones, M.A.; Martin, C.F. A discrete-time model with vaccination for a measles epidemic. *Math. Biosci.* **1991**, *105*, 111–131. [[CrossRef](#)]
28. Elazzouzi, A.; Alaoui, A.L.; Tilioua, M.; Tridane, A. Global stability analysis for a generalized delayed SIR model with vaccination and treatment. *Adv. Differ. Equ.* **2019**, *1*, 532. [[CrossRef](#)] [[PubMed](#)]
29. Li, J.; Ma, Z.; Brauer, F. Global analysis of discrete-time SI and SIS epidemic models. *Math. Biosci. Eng.* **2007**, *4*, 699. [[PubMed](#)]
30. Hassouna, M.; Ouhadan, A.; El Kinani, E.H. On the solution of fractional order SIS epidemic model. *Chaos Solitons Fractals* **2018**, *117*, 168–174. [[CrossRef](#)]
31. Wang, X.; Wang, Z.; Shen, H. Dynamical analysis of a discrete-time SIS epidemic model on complex networks. *Appl. Math. Lett.* **2019**, *94*, 292–299. [[CrossRef](#)]
32. Mena-Lorcat, J.; Hethcote, H.W. Dynamic models of infectious diseases as regulators of population sizes. *J. Math. Biol.* **1992**, *30*, 693–716. [[CrossRef](#)]
33. Harko, T.; Lobo, F.S.; Mak, M. Exact analytical solutions of the Susceptible-Infected-Recovered (SIR) epidemic model and of the SIR model with equal death and birth rates. *Appl. Math. Comput.* **2014**, *236*, 184–194. [[CrossRef](#)]
34. Nzokem, A.H. SIS epidemic model: Birth-and-death markov chain approach. *arXiv* **2021**, arXiv:2102.08992. [[CrossRef](#)]
35. Jardón-Kojakhmetov, H.; Kuehn, C.; Pugliese, A.; Sensi, M. A geometric analysis of the SIR, SIRS and SIRWS epidemiological models. *Nonlinear Anal. Real World Appl.* **2021**, *58*, 103220. [[CrossRef](#)]
36. Kusmawati, I.S.; Chandra, T.D. Stability analysis of SIRS epidemic model on measles disease spreading with vaccination and migration. *J. Phys. Conf. Ser.* **2021**, *1872*, 012033. [[CrossRef](#)]
37. Alonso-Quesada, S.; De la Sen, M.; Nistal, R. A SIRS Epidemic Model Supervised by a Control System for Vaccination and Treatment Actions Which Involve First-Order Dynamics and Vaccination of Newborns. *Mathematics* **2021**, *10*, 36. [[CrossRef](#)]
38. Boutayeb, H.; Bidah, S.; Zakary, O.; Lhous, M.; Rachik, M. On the optimal vaccination and travel-restriction controls with a discrete multi-region SIRS epidemic model. *Commun. Math. Biol. Neurosci.* **2021**, *2021*, 31.
39. Li, H.; Peng, R.; Wang, Z.A. On a diffusive SIS epidemic model with mass action mechanism and birth-death effect: Analysis, simulations and comparison with other mechanisms. *arXiv* **2018**, arXiv:1807.03451.
40. Keeling, M.J.; Eames, K.T. Networks and epidemic models. *J. R. Soc. Interface* **2005**, *2*, 295–307. [[CrossRef](#)]
41. Keeling, M. The implications of network structure for epidemic dynamics. *Theor. Popul. Biol.* **2005**, *67*, 1–8. [[CrossRef](#)]
42. Wang, W.; Tang, M.; Zhang, H.F.; Gao, H.; Do, Y.; Liu, Z.H. Epidemic spreading on complex networks with general degree and weight distributions. *Phys. Rev. E* **2014**, *90*, 042803. [[CrossRef](#)]
43. Galante, G.; Rizzi, R.L.; Rizzi, C.B. Simulating epidemiological processes using community-structured scale-free networks. *Rev. Bras. De Comput. Apl.* **2015**, *7*, 82–96. [[CrossRef](#)]
44. Pellis, L.; Ball, F.; Bansal, S.; Eames, K.; House, T.; Isham, V.; Trapman, P. Eight challenges for network epidemic models. *Epidemics* **2015**, *10*, 58–62. [[CrossRef](#)]
45. Della Rossa, F.; Salzano, D.; Di Meglio, A.; De Lellis, F.; Coraggio, M.; Calabrese, C.; Guarino, A.; Cardona-Rivera, R.; De Lellis, P.; Liuzza, D.; et al. A network model of Italy shows that intermittent regional strategies can alleviate the COVID-19 epidemic. *Nat. Commun.* **2020**, *11*, 5106. [[CrossRef](#)] [[PubMed](#)]
46. Britton, T. Epidemic models on social networks—With inference. *Stat. Neerl.* **2020**, *74*, 222–241. [[CrossRef](#)]
47. Stahlschmidt, S.; Stephen, D. *Comparison of Web of Science, Scopus and Dimensions Databases*; German Centre for Higher Education Research and Science Studies (DZHW): Hannover, Germany, 2020.
48. Qian, Z.; Tang, S.T.; Zhang, X.; Zheng, Z.M. The independent spreaders involved SIR Rumor model in complex networks. *Phys. A Stat. Mech. Appl.* **2015**, *429*, 95–102. [[CrossRef](#)]
49. Kabir, K.M.; Kuga, K.; Tanimoto, J. Analysis of SIR epidemic model with information spreading of awareness. *Chaos* **2019**, *119*, 118–125. [[CrossRef](#)]
50. Zhu, L.H.; Guan, G. Dynamical analysis of a rumor spreading model with self-discrimination and time delay in complex networks. *Phys. A Stat. Mech. Appl.* **2019**, *533*, 121953. [[CrossRef](#)]
51. Li, T.; Wang, Y.M.; Guan, Z.H. Spreading dynamics of a SIQRS epidemic model on scale-free networks. *Commun. Nonlinear Sci. Numer. Simul.* **2014**, *19*, 686–692. [[CrossRef](#)]

52. Zhang, Y.H.; Zhu, J.J. Stability analysis of I2S2R rumor spreading model in complex networks. *Phys. A Stat. Mech. Appl.* **2018**, *504*, 862–881. [[CrossRef](#)]
53. Zhang, Y.H.; Zhu, J.J. Dynamic behavior of an I2S2R rumor propagation model on weighted contract networks. *Phys. A Stat. Mech. Appl.* **2019**, *536*, 120981. [[CrossRef](#)]
54. Zan, Y.L. DSIR double-rumors spreading model in complex networks. *Chaos Solitons Fractals* **2018**, *110*, 191–202. [[CrossRef](#)]
55. Yang, A.Z.; Huang, X.Y.; Cai, X.M.; Zhu, X.F.; Lu, L. ILSR rumor spreading model with degree in complex network. *Phys. A Stat. Mech. Appl.* **2019**, *531*, 121807. [[CrossRef](#)]
56. Anastassopoulou, C.; Russo, L.; Tsakris, A.; Siettos, C. Data-based analysis, modelling and forecasting of the COVID-19 outbreak. *PLoS ONE* **2020**, *15*, e0230405. [[CrossRef](#)] [[PubMed](#)]
57. Giordano, G.; Blanchini, F.; Bruno, R.; Colaneri, P.; Di Filippo, A.; Di Matteo, A.; Colaneri, M. Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy. *Nat. Med.* **2020**, *26*, 855–860. [[CrossRef](#)]
58. Purkayastha, S.; Bhattacharyya, R.; Bhaduri, R.; Kundu, R.; Gu, X.; Salvatore, M.; Ray, D.; Mishra, S.; Mukherjee, B. A comparison of five epidemiological models for transmission of SARS-CoV-2 in India. *BMC Infect. Dis.* **2021**, *21*, 533. [[CrossRef](#)] [[PubMed](#)]
59. Zhang, Q.; Sun, K.; Chinazzi, M.; Pastore y Piontti, A.; Dean, N.E.; Rojas, D.P.; Merler, S.; Mistry, D.; Poletti, P.; Rossi, L.; et al. Spread of Zika virus in the Americas. *Proc. Natl. Acad. Sci. USA* **2017**, *114*, 4334–4343. [[CrossRef](#)]
60. Craft, M.E. Infectious disease transmission and contact networks in wildlife and livestock. *Philos. Trans. R. Soc. B Biol. Sci.* **2015**, *370*, 20140107. [[CrossRef](#)]
61. Wang, Y.; Jin, Z.; Yang, Z.; Zhang, Z.K.; Zhou, T.; Sun, G.Q. Global analysis of an SIS model with an infective vector on complex networks. *Nonlinear Anal. Real World Appl.* **2012**, *13*, 543–557. [[CrossRef](#)]
62. Cross, P.C.; Lloyd-Smith, J.O.; Bowers, J.A.; Hay, C.T.; Hofmeyr, M.; Getz, W.M. Integrating association data and disease dynamics in a social ungulate: Bovine tuberculosis in African buffalo in the Kruger National Park. *Ann. Zool. Fenn.* **2004**, *41*, 879–892.
63. Cohen, T.; Colijn, C.; Finklea, B.; Murray, M. Exogenous re-infection and the dynamics of tuberculosis epidemics: Local effects in a network model of transmission. *J. R. Soc. Interface* **2007**, *4*, 523–531. [[CrossRef](#)]
64. Godfrey, S.S. Networks and the ecology of parasite transmission: A framework for wildlife parasitology. *Int. J. Parasitol. Parasites Wildl.* **2013**, *2*, 235–245. [[CrossRef](#)]
65. Ren, G.; Wang, X. Epidemic spreading in time-varying community networks. *Chaos Interdiscip. J. Nonlinear Sci.* **2014**, *24*, 023116. [[CrossRef](#)] [[PubMed](#)]
66. Toutonji, O.A.; Yoo, S.M.; Park, M. Stability analysis of VEISV propagation modeling for network worm attack. *Appl. Math. Model.* **2012**, *36*, 2751–2761. [[CrossRef](#)]
67. Craft, M.E.; Volz, E.; Packer, C.; Meyers, L.A. Distinguishing epidemic waves from disease spillover in a wildlife population. *Proc. R. Soc. B Biol. Sci.* **2009**, *276*, 1777–1785. [[CrossRef](#)] [[PubMed](#)]
68. Wang, Y.; Cao, J.; Jin, Z.; Zhang, H.; Sun, G.Q. Impact of media coverage on epidemic spreading in complex networks. *Phys. A Stat. Mech. Its Appl.* **2013**, *392*, 5824–5835. [[CrossRef](#)] [[PubMed](#)]