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Comparative Analysis of SARS and COVID-19: Epidemiology and Pathogenesis

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Abstract: This study compares SARS-CoV-1 (SARS) with SARS-CoV-2 (COVID-19) epidemiology, pathophysiology, and worldwide effects. It examines the biological causes and widespread impacts of SARS-CoV-1 and SARS-CoV-2. The study examines transmission, immunological responses, and global expansion of these two diseases. Researchers have used various modelling methods to assess the pandemic's global and local effects. These methods include deterministic, data-driven, stochastic, agent-based, and their combinations. These models predict the epidemic's trajectory and evaluate non-pharmaceutical therapies' worldwide impact. Deterministic models like SIR and SEIR are used in infectious disease research. These models are applied to SARS-CoV-1 and SARS-CoV-2, highlighting their importance in public health measures. This article identifies crisis techniques like the pandemic to help modellers prepare for future outbreaks.

Keywords: COVID-19, Coronavirus, SARS, Virus, Pandemic, Epidemic, Mathematical modeling.

1. Introduction

Coronaviruses are single-strand RNA viruses that cause respiratory diseases in people and animals. Human coronaviruses have one of the biggest RNA viral genomes, 26.4 to 31.7 kilobases. The average viral particle and envelope sizes are 125 and 85 nm. [1]. Coronaviruses, with their crown-like spikes, are members of the Coronaviridae family and have four genera. SARS-CoV-1, which caused the 2002-2003 SARS outbreak, and SARS-CoV-2, which produced the COVID-19 pandemic in late 2019, are both β genus viruses that have caused worldwide health catastrophes. Mathematics has helped analyse pandemics, like COVID-19, by understanding transmission dynamics, anticipating virus spread, and predicting future behaviour. Shayak et al. showed how models can predict the epidemic curve, displaying infection rates over time, which is important for healthcare planning. These models can also replicate social separation and lockdowns to help policymakers make evidence-based decisions. [2]. Kucharski et al. noted that early intervention techniques can significantly reduce transmission rates, emphasising the importance of effective public health activities. [3]. Moore et al. noted that mathematical modelling is essential for vaccine efficacy and distribution optimisation. Managing continuing viral mutations requires regular vaccine changes to sustain pandemic control.[4]. Modelling the SARS and COVID-19 outbreaks improves our understanding and strategy for future outbreaks. These models are crucial for COVID-19 mitigation and pandemic preparedness.

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2. Epidemiology

2.1. Emergence and Spread of SARS-CoV-1

According to the WHO, SARS-CoV-1 first appeared in Guangdong Province, China, in November 2002.[5] Late February 2003 saw the pandemic extend to 4 countries. It was the first highly transmissible new disease of the 21st century, spreading over global aviation networks. [6]. Within months, the disease had spread to over 30 nations, mostly in Asia Pacific.

Despite the wide spatial dispersion, 2003 containment measures were successful because to effective public health strategies like quarantine and isolation. [7, 8].

2.2. Emergence and Spread of SARS-CoV-2 (COVID-19)

COVID-19 began in Wuhan, China, in December 2019 and spread worldwide. The WHO labelled it a pandemic on March 11, 2020, citing its global impact due to the lack of human immunity. Quarantines, social isolation, and travel restrictions were implemented in practically all countries as the virus spread.[9]. Since then, many varieties have been found. The Delta variety, which emerged in India in October 2020, dominated the US from autumn 2021 to spring 2022 due to its high transmissibility. The Alpha variant was first reported in southeastern England in December 2020, whereas the Beta variation appeared in South Africa and Nigeria. The Gamma strain, discovered in January 2021 among Brazilian travellers in Japan, is more contagious. Omicron was discovered in South Africa in November 2021 and quickly became the dominant strain. [10].

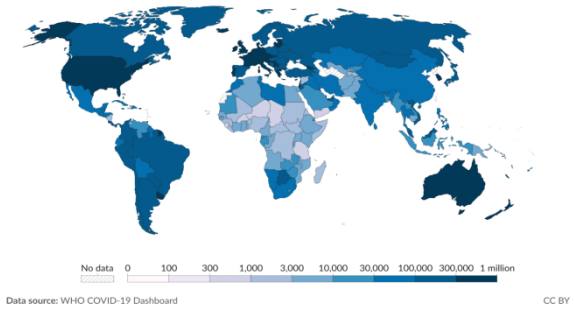

Figure 1: Cumulative Confirmed COVID-19 cases per million people. Our World in Data [11].

2.3. General Transmission Dynamics

SARS-CoV-1 and SARS-CoV-2 are spread through respiratory droplets and physical contact. For both viral strains, indirect transmission through contaminated surfaces is documented. [7]. R0 shows SARS-CoV-1 and CoV-2 transmission dynamics. The average number of secondary infections per sick person in a completely susceptible population is R0. When $R0 > 1$, the sickness can spread and become an epidemic, but when $R0 < 1$, it will die out. The transmission rate and recovery rate are in the R0 equation below.

$$R0 = \frac{\beta}{\gamma} \tag{1}$$

According to calculations, SARS-CoV-1's fundamental reproduction number (R0) falls between two and four. The R0 for SARS-CoV-2 was first estimated to be between 2 and 3.5, however it has since varied depending on the region and stage of the pandemic. [12, 13].

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2.4. Global Spread and Containment Efforts

Both viruses have spread globally, but COVID-19 has had a more extensive impact. Quarantine, isolation, social distancing, and the use of personal protective equipment (PPE) have played critical roles in controlling both outbreaks.

3. Virology and pathogenesis

SARS-CoV-1 and SARS-CoV-2 are both Betacoronaviruses, exhibiting structural similarities with 14 open reading frames (ORFs) that encode essential proteins for replication and structural integrity. They share analogous structural proteins: spike (S), envelope (E), membrane (M), and nucleocapsid (N). Both viruses utilise the ACE2 receptor for cellular entry and demonstrate approximately 82% genetic homology [14]. SARS-CoV-2 transmits more swiftly and lethally than SARS-CoV-1, notwithstanding structural resemblances. Genetic variations influence infectivity and immune evasion. Both viruses utilise the ACE2 receptor; however, SARS-CoV-2 exhibits a greater binding affinity, hence enhancing its transmissibility.

4. Immune Response

The immune response to both viruses involves antibody production and T-cell activation. In severe cases, an exaggerated immune response can lead to complications such as acute respiratory distress syndrome (ARDS). [16]. Fever, cough, and fatigue are prevalent symptoms in both diseases. COVID-19 frequently results in the loss of taste or smell, a symptom less commonly observed in SARS. Severe instances may lead to pneumonia, acute respiratory distress syndrome (ARDS), and multi-organ failure. COVID-19 exhibits an elevated incidence of severe cases and mortality, likely attributable to sustained T cell overactivation, which exacerbates tissue damage and systemic inflammation.

5. Diagnostic Modeling

Mathematical models are crucial for the analysis of infectious disease transmission and the effects of interventions. They assist in planning, identifying deficiencies, and formulating responses prior to an epidemic. Modelling provides insights into transmission forecasts, potential geographical spread, and the effectiveness of various interventions during a pandemic[16]. Infectious disease transmission models encompass deterministic, stochastic, and agent-based categories. This section will focus on deterministic models, particularly the SIR and SEIR models.

5.1. Deterministic Models

SIR and SEIR models are deterministic frameworks that employ fixed parameters to forecast the dissemination of diseases. The models categorise the population into distinct compartments. The subsequent models establish a basis for more intricate models that include extra compartments and parameters, including those that account for waning immunity and quarantine measures.

The SIR model categorises the population into three compartments: Susceptible (S), Infectious (I), and Recovered (R). The model is described by the subsequent differential equations:

$$\frac{dS}{dt} = -\beta SI \tag{2}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \tag{3}$$

$$\frac{dR}{dt} = \gamma I \tag{4}$$

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The susceptible population's change is modeled by the equation, where β represents the transmission rate and γ the recovery rate, allowing susceptible members to become infected, $\frac{dS}{dt} = -\beta SI$. S denotes susceptible individuals, while I represents infected individuals. The negative rate of change indicates a decline in the susceptible population due to infections. The term signifies new infections per time unit, assuming disease transmission occurs through contact between susceptible and infected individuals. The infected population's rate of change is described by the second equation, $\frac{dI}{dt} = \beta SI - \gamma I$. The term denotes the infection rate of susceptible individuals, while the other indicates the recovery rate of infected individuals transitioning to the recovered compartment. Thus, the equilibrium between new infections and recoveries dictates the fluctuation in the infected population over time. The recovery population's rate of change is described by the third equation, $\frac{dR}{dt} = \gamma I$. Therefore, the growth rate of the recovered population increases as individuals move from the infected compartment to the recovered compartment.

SEIR Models:

The SEIR model includes an exposed (E) compartment for individuals who are infected but not yet infectious, described by these equations:

$$\frac{dS}{dt} = -\beta SI \tag{5}$$

$$\frac{dE}{dt} = \beta SI - \sigma E \tag{6}$$

$$\frac{dI}{dt} = \sigma E - \gamma I \tag{7}$$

$$\frac{dR}{dt} = \gamma I \tag{8}$$

In this context, σ represents the rate at which individuals who have been exposed transition to an infectious state. The dynamics of the susceptible population are described by the initial equation, $\frac{dS}{dt} = -\beta SI$. In accordance with the equation utilized in the SIR model, as the populations of susceptible individuals (S) and infectious individuals (I) rise, the incidence of new exposures also escalates. The dynamics of the exposed population's growth are represented by the subsequent equation $\frac{dE}{dt} = \beta SI - \sigma E$. The term βSI continues to represent the rate at which susceptible individuals are becoming exposed. The term $-\sigma E$ represents the rate at which exposed individuals are becoming infectious. Consequently, the equation illustrates the overall variation in the count of exposed individuals. The dynamics of the infected population are represented by the third equation $\frac{dI}{dt} = \sigma E - \gamma I$. The equation illustrates the overall variation in the population of infected individuals, where σE represents the transition rate of exposed individuals to the infectious state, and −γI denotes the recovery rate of infected individuals. Finally, the dynamics of the recovered population are represented by the fourth equation. $\frac{dR}{dt} = \gamma I$. As people recover from the infection, they move from the infectious group (I) to the recovered group (R). The rate at which they recover is determined by γ, the recovery rate.

Application to SARS-CoV-1 and SARS-CoV-2:

SEIR models are crucial for simulating the transmission of infectious diseases such as COVID-19 and SARS, as they incorporate a compartment for exposed individuals. This is essential for diseases characterised by an incubation period, during which individuals do not become immediately infectious following exposure. The latent period is defined as the interval between viral entry into human cells and the initiation of infectiousness. The incubation period for SARS-CoV-1 typically

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ranges from 2 to 7 days, with a possible extension to 10 days [5]. The average incubation period for SARS-CoV-2 is approximately 3.69 days, followed by an infectious phase of about 3.48 days. [17]SEIR models incorporate the exposed stage, thereby refining transmission dynamics and improving predictions of disease spread. They assist public health officials and policymakers in evaluating the impact of the disease, assessing the effectiveness of interventions (e.g., social distancing, quarantine, vaccination), and making informed decisions to mitigate outbreaks. Simulations conducted by these models predict future cases, hospitalisations, and fatalities, thereby informing resource allocation and control strategies to protect public health.

6. Conclusion

This paper presents a comparative analysis of SARS-CoV-1 and SARS-CoV-2, emphasizing their epidemiology, pathogenesis, and global impact. Both viruses have significantly influenced global health, with SARS-CoV-2 exhibiting greater transmissibility and diverse symptoms. Understanding their transmission dynamics and immune responses has been vital for public health strategies. Insights from the 2003 SARS outbreak have guided COVID-19 responses, particularly the rapid genome sequencing that enabled swift identification and diagnostic test development. This underscores the necessity of early detection and prompt action in outbreak containment. For COVID-19, rapid sequencing of SARS-CoV-2 has similarly advanced PCR tests, serological assays, and vaccine development. Mathematical modeling, including SIR and SEIR models, has been crucial in predicting the spread and evaluating interventions. As COVID-19 data continues to grow, future research may explore more complex models.

However, despite advancements in data and computational power, current models still have their limitations. For instance, the high amounts of data are difficult to analyze without the use of supercomputers [19]. In conclusion, Mathematical and computational models are essential for understanding and managing the COVID-19 pandemic, offering a framework for testing scenarios and interventions that inform public policy and predict future trends. Ongoing refinement of these models is vital for addressing future outbreaks and enhancing public health responses.

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