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Modeling the Effects of Media Coverage on the COVID-19 Transmission Dynamics

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Abstract The increasing importance of media's role in the transmission of diseases presents a novel dimension to be considered in disease modeling. In our study, we introduce a comprehensive model based on a system of ordinary differential equations to elucidate the dynamics of COVID-19's spread, with a particular focus on the influence of media. Utilizing established compartmental modeling approaches, our model categorizes individuals into six distinct groups: Asymptomatic, Clinically Ill, Quarantined, Hospitalized, Recovered, and Vaccinated. This classification enables a nuanced analysis of the interactions that significantly impact disease propagation. Furthermore, our model innovatively incorporates parameters specifically designed to evaluate media exposure's effect on disease transmission dynamics. Employing the Next-Generation Method, we derive an explicit formula for the Basic Reproduction Number, a critical metric in epidemiology. Our findings include the conditions necessary for achieving a Disease-Free Equilibrium, highlighting the potential for disease eradication or persistence within a population. Through stability analysis, we identify specific conditions under which COVID-19 can be either eliminated or become endemic, offering insights into effective disease management strategies. Our analysis is enriched with numerical simulations, utilizing data from the Virginia Department of Health among other sources, to validate our model's predictions. This study not only sheds light on the pivotal role of media in disease spread but also provides a foundational framework for predicting and managing the dynamics of COVID-19 transmission.

Keywords COVID-19, Epidemiology, Asymptomatic Individuals, Media Effects

1 Introduction

COVID-19, [1], a disease caused by a new coronavirus SARS-CoV-2, has drastically affected our way of life over the last four years. The first case of COVID-19, documented in Virginia, occurred on March 7, 2020, in which a Marine at Fort Belvoir in Richmond, VA tested positive for the virus. [2] At the time, the governor and other state public health officials stated "evidence has not been seen of COVID-19 spreading in Virginia and said the risk is low" and advised Virginia residents to "wash their hands, cover their mouths while coughing, and use hand sanitizer". Two years and 20,424 deaths of Virginians later (and counting) we see that stronger methods for mitigation were necessary [2].

Anyone can get COVID-19, and most infections are mild. The older you are, the higher your risk of severe illness is. The virus attacks the respiratory tract which includes one's sinuses, nose, and throat as well as the lower respiratory tract which includes the windpipe and lungs [3]. Symptoms include, but are not limited to, fever, coughing, shortness of breath, trouble breathing, fatigue, and chills; sometimes with shaking, body aches, headache, sore throat, and congestion/ runny nose. Some people infected with the disease have also experienced a loss of smell or taste, nausea, and diarrhea [3].

Investigations as to its origin are ongoing. Currently, there is no specific treatment for the virus. Perhaps this knowledge gap is the cause of distrust in historically trusted media sources [3].

Fortunately, we can use the power of traditional and social media to help spread information about the disease and ways

to combat the spread. But is media a double-edged sword? The inspiration for our model was based on a Canadian study [4], in which the authors hypothesized that the more access to media an individual has, the more likely that individual would be to practice social distancing measures. However, in the United States, does the same notion hold true? And would it hold in a traditional Red State where the views of vaccination versus non-vaccination tend to differ along political lines? Researchers at the University of Wisconsin-Milwaukee have found that the type of media people are exposed to will determine behavior: whether people will mask up, remain six feet apart, continue to isolate when experiencing COVID symptoms, or even choose to get vaccinated and boosted [5]. Some of the misinformation about mitigating measures for the coronavirus included the proliferation of fake COVID cures, the overt mocking of experts and containment measures, a libertarian view of placing personal economic liberty over public safety, rumors asserting the pandemic is over and there is no further threat of infection, likening the severity of COVID to that of influenza, religious concerns, incorrect information about the effectiveness of social distancing, and finally the insistence that COVID is an overall hoax [5]. Many of these ideas were pervasive regionally and had demographic correlations.

Because there is no specific treatment for COVID-19, it's imperative that those in the population who are medically able to become vaccinated do so to combat the spread of the disease. Fortunately, several vaccines have become available on the market. The Pfizer-BioNTech vaccine was approved in August 2021 for people anyone over the age of 16 and adolescents between the ages of 5 to 15 with Emergency Use Authorization (EUA) [6], [7]. Based on an independent analysis of the FDA, this vaccine has an efficacy of 95%. However, data is still being gathered about its effectiveness against COVID variants. Moderna: Moderna was granted approval by the FDA for people over the age of 18 in January 2022. This vaccine also has an efficacy of 95% [6], [7]. Johnson & Johnson: Johnson & Johnson was authorized for use in February 2021 and also available for anyone above the age of 18 and is the preferred alternative for individuals who can not, for medical reasons, take other vaccines. This vaccine, however, has an efficacy of 67% "in preventing moderate to severe/critical disease by 14 days after vaccination—and 66% effective 28 days after vaccination" [6], [7]. Novavax is an additional vaccine that is recommended for Emergency Use Authorization, EUA. At the time of the writing of this paper, it has not been FDA approved; however, it is available in 40 other countries with an efficacy of 90% in its clinical trials. Finally, Oxford-AstraZeneca is set apart from its competitors by its cost-effectiveness and storage. It also has an efficacy of vaccine of 76%.

However, with all of these epidemiological advances, the permeation of the disease in our communities (in which some communities are more vulnerable than others) greatly depends on our willingness to heed the advice of health care professionals. In particular, those in vulnerable populations should especially heed the advice of medical professionals and absorb fact-based media. Those most vulnerable include individuals with cancer, chronic kidney, liver & lung disease, dementia, and diabetes. Also, people with disabilities like ADHD, cerebral palsy, birth defects, intellectual and developmental disabilities, learning disabilities, spinal cord injuries, and Down syndrome are particularly susceptible to the disease [8]. In general, people who find it difficult to engage in and interact with the world around them, and those dependent on others for their daily needs, are highly susceptible to infection. Individuals who suffer from heart conditions, HIV infection, immuno-compromising conditions & weakened immune systems, mood disorders (such as depression), schizophrenia spectrum disorders, obesity, and engage in little or no physical activity are at high risk of infection. Ironically, people have become more inactive during the pandemic due to isolation and work-from-home advances. Pregnancy, Sickle-Cell disease, current and former smokers, those who have had organ or blood stem cell transplants, stroke or cerebrovascular disease, substance abusers (alcohol, opioid, cocaine use disorder), and Tuberculosis are also at higher risk of getting the virus than others [8].

We see that this list of vulnerable sub-populations has great socio-economic implications. People with some types of disabilities may be more likely to get very sick from COVID-19 because of underlying medical conditions, living in congregate settings, or systemic health and social inequities [8]. People living with mental health conditions may have certain mood disorder symptoms interfering with their ability to care for themselves and be engaged with their health properly. Also, those impacted by the societal ills of poverty, lack of access to preventative health care, ability to understand health recommendations, lack of access to affordable health care, and also living in tight spaces or facilities like nursing homes, homeless shelters, prison, or psychiatric inpatient units are at high risk.

1.1 Integrating Media Influence into Epidemiological Models.

The role of media coverage has emerged as a pivotal factor influencing public behavior and, consequently, the transmission rates of the virus. Recent studies have shed light on how media coverage impacts COVID-19 transmission dynamics, enriching the foundation for our research on modeling these effects. The inclusion of media coverage in epidemiological models offers a nuanced understanding of its potential to alter transmission dynamics. A study by [Dai et al.] [9], proposes a fractional-order SIS model that incorporates media coverage, revealing how heightened media awareness can significantly reduce the basic reproduction number, $\mathcal{R}_0$, of the virus. This finding aligns with our approach to include media parameters in our model, underscoring the theoretical and practical implications of media influence on disease spread.

Research by Mach et al. [10], explores the variability in news media coverage of COVID-19 and its effects on public

health behaviors and policy-making. The study highlights the disparity in coverage and the potential for certain narratives to either enhance or undermine public health efforts. This underscores the need for our model to account for different types of media influence, ranging from traditional news to social media platforms, and their varying impacts on public perception and behavior.

An innovative model presented by Mohsen et al. [11], examines the combined effects of quarantine strategies and media reports, showcasing how effective communication through media can enhance compliance with quarantine measures. This research supports our hypothesis that media coverage not only affects public awareness but also influences the success of public health interventions, such as quarantine and social distancing guidelines.

The study by Li et al. [12], considers the saturation of media coverage and its interplay with limited medical resources. It suggests that there is a threshold beyond which additional media coverage may not yield further benefits in controlling the spread of COVID-19. This insight is crucial for our model, emphasizing the need to explore the diminishing returns of media influence as part of the transmission dynamics.

Finally, the impact of social media advertisements on COVID-19 transmission dynamics, investigated by Rai et al. [13], highlights the power of targeted social media campaigns in altering public behaviors. This aligns with our research that focuses on modeling the effects of media coverage, suggesting that social media can serve as a critical tool in disseminating health information and encouraging protective behaviors among the population.

The aforementioned studies collectively reinforce the significance of incorporating media coverage into models of COVID-19 transmission dynamics. They provide a multifaceted view of how media influences public perceptions, behaviors, and, ultimately, the effectiveness of health interventions. By integrating these insights into our research, we enhance the robustness of our model and contribute valuable perspectives to the ongoing efforts to mitigate the COVID-19 pandemic.

Our paper is organized as follows: we introduce two traditional compartmental models with media influence and without in Section II, followed by analytical results of each system in Section III. In Section IV, we present the results of our numerical simulations. Section V contains our conclusions and managerial implications on public health and public policy. Section VI outlines the limitations of our study and plans for future work.

2 Materials and Methods

2.1 Modeling COVID-19 Transmission with Media Effect

In our endeavor to understand the intricate dynamics of COVID-19's spread, we use traditional compartmental modeling techniques to analyze the spread of COVID-19. We divide the population into distinct groups, or *compartments*, based on their disease status, such as susceptible (S), exposed (E), infected (I), and recovered (R). Each compartment represents a stage in the disease's progression, and individuals move between compartments based on the disease dynamics and interventions applied. The movement is modeled using differential equations describing the rate of change in the population size of each compartment over time. In the case of our model, the total population N is divided into subpopulations: *Susceptible* (S), *Latent/Exposed* (E), *Asymptomatic* (A), *Infectious* (I), Quarantined (Q), *Hospitalized* (H), *Vaccinated* (V) and *Recovered* (R). The population is assumed to be completely susceptible to the disease at the beginning of the epidemic.

Compartmental models offer a balance between simplicity and the ability to capture the essential dynamics of disease spread. This simplicity makes them particularly useful for understanding the fundamental aspects of the epidemic, such as the transmission rate and potential impact of public health interventions, in a clear and interpretable manner. Additionally, these models are flexible and can be extended or modified to include more compartments (such as vaccinated or hospitalized individuals) or additional factors influencing disease transmission, such as media influence in the context of our study. Despite their simplicity, compartmental models can provide valuable predictions about the course of an epidemic, including peak infection times, the total number of cases, and the duration of the epidemic. These predictions are crucial for planning and implementing effective public health responses, especially for emerging infectious diseases like COVID-19, where rapid decision-making is essential.

Compartmental models allow for the explicit inclusion of various public health interventions, such as vaccination, quarantine, social distancing, and, as in our study, media influence. This capability enables researchers and policymakers to assess the potential effectiveness of different strategies in controlling the disease spread and to explore various scenarios that can inform public health decisions. These models can be parameterized and calibrated using epidemiological data, making them a powerful tool for real-time analysis and forecasting. In the case of COVID-19, where data is continuously being collected and updated, compartmental models can be adapted to reflect new information, providing insights into the evolving dynamics of the pandemic.

Specifically, for our study, compartmental models are particularly well-suited to explore how media coverage affects public behavior and, consequently, the transmission dynamics

of COVID-19. By integrating media influence parameters into the model, we can quantitatively assess the impact of media on the effectiveness of public health measures and the overall course of the epidemic. This approach provides a novel perspective on the role of information dissemination and public perception in epidemic control.

We define β as the effective transmission rate where susceptible individuals can contract COVID from infectives. We also note that a susceptible individual can become infected by both *Asymptomatic* (A) and *Symptomatic* I individuals. Asymptomatic individuals are carriers of the disease but do not exhibit any physical signs or symptoms. Asymptomatic people can still spread the disease to others, often without knowing it, which is a significant factor in the transmission of infectious diseases like influenza and COVID-19. When an individual is *symptomatic*, they exhibit physical or psychological symptoms that are indicative of COVID-19. Symptomatic individuals are usually aware of their illness due to the presence of these signs and symptoms, as a result, we assume asymptomatic individuals are infectious with a lower rate of infectiousness, represented by the parameter μ.

Once a susceptible individual interacts with an infectious individual, there is a latent period k, for the incubation of the disease. A proportion of the *Exposed*, p, become *Symptomatic*, I, and a proportion $(1-p)$ become *Asymptomatic*, A.

As the disease progresses, symptomatic infectives either Isolate/*Quarantine* at a rate of ϵ or become *Hospitalized* at a rate of θ. Those individuals who quarantine can become extremely ill and later become hospitalized at a rate of σ. We assert that symptomatic infectives can die from COVID before they are admitted to the hospital and after hospitalization at rates of δ_1 and δ_2 respectively. The parameters of γ_1, γ_2, and γ_3 are used to represent the recovery rates from A, I, and Q respectively.

Susceptible individuals become *Vaccinated* at a rate of ξ. Based on the fact that the COVID-19 vaccines provide *partial* immunity, a vaccinated individual can still become infected after contact with asymptomatic and symptomatic infectives. This subpopulation of the vaccinated moves to the *Latent* class E and we define u as our vaccine efficacy.

The previous description corresponds to our baseline model, Model (1). A new model, Model (2), features the fact that individuals adopt behaviors, influenced by media that may reduce the probability of becoming infected. Some parameters will be modified accordingly. For instance, the inclusion of the media effect on the COVID-19 transmission dynamics is considered by the following parameters:

- m is the proportion of people who use media.
- σ_s represents the proportion of people who practice social distancing measures.
- Correspondingly, σ_v, is the proportion of individuals affected by media who get vaccinated.

Some of the parameter values for the simulations corresponding to the year 2021 were found in [14], [15]. Therefore, in Model (2), we consider $\tilde{\xi} = \xi(1 + m\sigma_v)$ and $\tilde{\beta} = \beta(1 - m\sigma_s)$ and $\tilde{u} = u(1 + m\sigma_v)$. In Model (2), the rest of the parameters remain the same. A thorough analysis of epidemiological models for human infectious diseases is included in [16].

Figure [1] is a flowchart that represents the transfer between the compartments with no media effect.

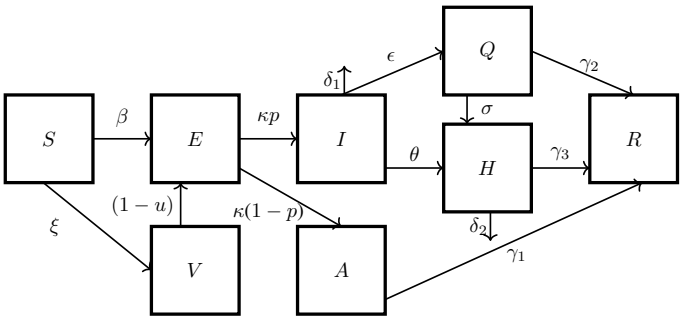

Figure 1. Transfer between compartmental classes and their corresponding parameters

Descriptions of the variables and parameters illustrated in Figure (1) used in our study are outlined in Table (1).

The goal of this study is to analyze the effect of media on the dynamics of COVID-19 transmission. Two models were considered:

- **Model (1)**, without the inclusion of media effect into the parameters and does not include any parameter with media.
- **Model (2)**, with the inclusion of media effect. These media parameters are defined as:

$$\tilde{\xi} = \xi(1 + m\sigma_v)$$
$$\tilde{\beta} = \beta(1 - m\sigma_s)$$
$$\tilde{u} = u(1 + m\sigma_v).$$

We introduce a system of ordinary differential equations, **Model (1)**, to model the dynamics between the compartmental classes for COVID-19 as follows:

Symbol	Description	Value
β	Baseline Transmission Rate	0.2
μ	Transmission Rate of Asymptomatic Infectives	$0.02 - 0.05$
ξ	Immunization Rate	$0.7 - 0.8$
u	Immunization Efficiency Rate	0.8
k	Latent Period Rate	0.833
ϵ	Quarantine Rate	$0.70 \times \frac{1}{7}$
θ	Rate of Hospitalization	$0.30 \times \frac{1}{7}$
σ	Rate from Quarantine to Hospitalization	0.02
δ_1	Disease-Death Rate of Infected Individuals	0.01
δ_2	Disease-Death Rate of Hospitalized Individuals	0.111
γ_1, γ_2,γ_3	Recovery Rates for the Asymptomatic, Quarantined, & Hospitalized	$0.99, 0.98, 0.889$
m	Proportion of Individuals exposed to Media	0.9
σ_s	Proportion of Individuals exposed to media who Socially Distance	$[0.2, 0.76]^*$
σ_v	Proportion of Individuals affected by Media who Vaccinate	$[0.29, 0.69]^{**}$

Table 1. Parameters Values. *The proportion of people exposed to media is changing during the year 2021, depending on the regulations imposed by the government. **Vaccination rates for the year 2021 are increasing between this interval.

$$\begin{aligned}
\frac{dS}{dt} &= -\beta S\frac{(I+\mu A)}{N} - \xi S,\\
\frac{dE}{dt} &= \beta S\frac{(I+\mu A)}{N} + (1-u)\beta V\frac{(I+\mu A)}{N} - kE,\\
\frac{dI}{dt} &= kpE - (\delta_1+\epsilon+\theta)I,\\
\frac{dA}{dt} &= k(1-p)E - \gamma_1 A,\\
\frac{dV}{dt} &= \xi S - (1-u)\beta V\frac{(I+\mu A)}{N},\\
\frac{dQ}{dt} &= \epsilon I - (\sigma+\gamma_2)Q,\\
\frac{dH}{dt} &= \theta I + \sigma Q - (\delta_2+\gamma_3)H,\\
\frac{dR}{dt} &= \gamma_1 A + \gamma_2 Q + \gamma_3 H. \qquad (1)
\end{aligned}$$

Here, the total population, denoted N, satisfies

$$S + E + I + A + V + Q + H + R = N.$$

We also introduce the following system for **Model (2)**:

$$\begin{aligned}
\frac{dS}{dt} &= -\tilde{\beta} S\frac{(I+\mu A)}{N} - \tilde{\xi} S,\\
\frac{dE}{dt} &= \tilde{\beta} S\frac{(I+\mu A)}{N} + (1-\tilde{u})\tilde{\beta} V\frac{(I+\mu A)}{N} - kE,\\
\frac{dI}{dt} &= kpE - (\delta_1+\epsilon+\theta)I,\\
\frac{dA}{dt} &= k(1-p)E - \gamma_1 A,\\
\frac{dV}{dt} &= \tilde{\xi} S - (1-\tilde{u})\tilde{\beta} V\frac{(I+\mu A)}{N},\\
\frac{dQ}{dt} &= \epsilon I - (\sigma+\gamma_2)Q,\\
\frac{dH}{dt} &= \theta I + \sigma Q - (\delta_2+\gamma_3)H,\\
\frac{dR}{dt} &= \gamma_1 A + \gamma_2 Q + \gamma_3 H. \qquad (2)
\end{aligned}$$

3 Analysis of the Model

In this section, the analysis of Model (1) is developed. Traditional techniques of analyzing dynamical systems are used to analyze the system locally around the equilibrium points. (See [17] for details on dynamical systems theory.) Equilibrium points are steady states/fixed points where our system's subpopulations do not change over time. These points represent the state of the system where the rates of input and output for each compartment are balanced, leading to no net change in the populations within our compartments. We evaluate the Disease-Free Equilibrium (DFE), E_0, and Basic Reproduction Number of the disease, denoted $\mathcal{R}_0$. The DFE is a state where no disease exists in our population. Analyzing the stability of the DFE helps in understanding the conditions under which the disease can be eradicated. The Basic Reproduction Number,$\mathcal{R}_0$, is a fundamental concept in epidemiology that measures the average number of secondary infections produced by a single infection in a completely susceptible population. $\mathcal{R}_0$ represents the transmission potential of COVID-19, indicating how many people (on average) and an infected person will spread the disease to, in the absence of any preventive measures or immunity in the population. The $\mathcal{R}_0$ of the disease is crucial for understanding the infectiousness and potential spread of an epidemic within a community. Its value serves as a threshold parameter that helps in determining the stability of DFE and endemic states within a population. More specifically:

- If $\mathcal{R}_0 < 1$, each existing infection causes less than one new infection. Consequently, the disease will decline and eventually die out, leading to a stable disease-free equilibrium.
- If $\mathcal{R}_0 > 1$, each existing infection causes more than one new infection, suggesting the disease will spread through the population, potentially leading to an epidemic or endemic state.

The calculation and analysis of $\mathcal{R}_0$ are fundamental in guiding public health interventions. By reducing $\mathcal{R}_0$ to less than

one through vaccination, social distancing, and other control measures, it is possible to control and eventually eliminate the spread of the disease. In the context of our study on COVID-19 dynamics, understanding $\mathcal{R}_0$ is pivotal in assessing the effectiveness of media influence and public health strategies in altering the course of the epidemic. Through our models, we examine how media coverage impacts public behavior and, subsequently, the value of $\mathcal{R}_0$, offering insights into how strategic communication can be leveraged to mitigate the spread of COVID-19.

To compute the expression for $\mathcal{R}_0$, we use the Next-Generation Matrix Method, as outlined in [18]. This method involves partitioning the model into new infections and transitions between compartments, leading to the construction of two matrices: the Next-Generation Matrix F (new infections) and the transition matrix V (transitions between compartments). The spectral radius (dominant eigenvalue) of the matrix FV^{-1}, where V^{-1} is the inverse of V, gives the value of $\mathcal{R}_0$. The justification for using the Next-Generation Matrix Method in our study lies in its robustness and accuracy in determining $\mathcal{R}_0$ for complex infectious disease models, including those with multiple compartments and stages of infection. This method allows for a clear distinction between the contributions of different infectious compartments to disease transmission, enabling a precise calculation of $\mathcal{R}_0$. Such precision is crucial for understanding the dynamics of COVID-19 spread and assessing the effectiveness of control measures. The method's flexibility in accommodating various model structures makes it particularly suitable for our study, which includes compartments for asymptomatic, symptomatic, quarantined, and hospitalized individuals, among others. By applying the Next-Generation Matrix Method, we can quantitatively evaluate how each group contributes to the spread of COVID-19, informing targeted interventions. Moreover, this method aligns with the World Health Organization and the Centers for Disease Control and Prevention guidelines for infectious disease modeling, ensuring our study's findings are relevant and comparable with other research in the field.

The positivity of solutions for Model (1) (see Theorem 1) and stability results for DFE, E_0, (see Theorem 2) are included in the Appendix.

Model (1) can be rearranged, and its solutions can be expressed as $\mathbf{x} = (E, I, A, S, V, Q, H, R)$. Using the Next-Generation Matrix Method, we define the two vectors $\mathscr{F}$ and $\mathscr{V}$ as follows:

$$\mathscr{F} = \begin{bmatrix} \beta S \frac{(I+\mu A)}{N} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

and

$$\mathscr{V} = \begin{bmatrix} -(1-u)\beta V \frac{(I+\mu A)}{N} + kE \\ -kpE + (\delta_1 + \epsilon + \theta)I \\ -k(1-p)E + \gamma_1 A \\ \beta S \frac{(I+\mu A)}{N} + \xi S \\ -\xi S + (1-u)\beta V \frac{(I+\mu A)}{N} \\ -\epsilon I + (\sigma + \gamma_2)Q \\ -\theta I - \sigma Q + (\delta_2 + \gamma_3)H \\ -\gamma_1 A - \gamma_2 Q - \gamma_3 H \end{bmatrix}$$

Model (1) has the Disease-Free Equilibrium $x_0 = (0, 0, 0, N, 0, 0, 0, 0)$. Notice the only new infected compartments are E, I, A, the matrices $F = \left(\frac{\partial \mathscr{F}}{\partial x_i}\right)\Big|_{x_0}$ and $V = \left(\frac{\partial \mathscr{V}}{\partial x_i}\right)\Big|_{x_0}$, corresponding to the new infection terms are given by:

$$F = \begin{bmatrix} 0 & \beta & \beta\mu \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} k & 0 & 0 \\ -kp & \delta_1 + \epsilon + \theta & 0 \\ -k(1-p) & 0 & \gamma_1 \end{bmatrix}.$$

Matrix F is non-negative and V is non-singular. Therefore, FV^{-1} is also non-negative. The spectral radius (largest eigenvalue of FV^{-1})is the Basic Reproduction Number $\mathcal{R}_0$ for Model (1):

$$\mathcal{R}_0 = \frac{\beta p}{\delta_1 + \epsilon + \theta} + \frac{\beta\mu(1-p)}{\gamma_1}. \tag{3}$$

Similarly, the basic reproduction number for Model (2) (with media parameters), is given by:

$$\mathcal{R}_m = \frac{\beta(1-m\sigma_s)p}{\delta_1 + \epsilon + \theta} + \frac{\beta(1-m\sigma_s)\mu(1-p)}{\gamma_1}. \tag{4}$$

If $\mathcal{R}_0 < 1$, the DFE for Model (1) is locally asymptotically stable. If $\mathcal{R}_0 > 1$, then the DFE is unstable. This statement and its proof are included in the Appendix as Theorem (2).

4 Numerical Simulations

Numerical simulations are included to analyze the implications of changing different parameter values with the basic reproduction numbers $\mathcal{R}_0$ and $\mathcal{R}_m$. First, the proportion of asymptomatic individuals, p, was varied on both reproduction numbers. Then, the proportion of quarantined individuals, ϵ, was varied in both reproduction numbers. Lastly, the recovery rate for asymptomatic individuals, γ_1, was varied for both reproduction numbers. The ranges for parameters p, ϵ, and γ_1 are taken from public data from governmental institutions.

The parameters for Model (1) depend on the region to be considered. Several conditions had to be evaluated. For example, government regulations for each state changed during the time of the pandemic. Many of those state regulations were influenced by the federal government; therefore, the parameters used in the simulations are approximations and were found in reliable literature. Isolation parameters are summarized for the year 2021 in Table (2).

- **Isolation Case #1** refers to small gatherings of people, such as get-togethers with family and friends.
- **Isolation Case #2** refers to people going to public places, such as stores and restaurants.
- **Isolation Case #3** refers to people participating in events with large crowds, such as concerts, festivals, and sporting events.

	Isolation Case #1	**Isolation Case #2**	**Isolation Case #3**
January 2021	0.57	0.61	0.76
April 2021	0.37	0.44	0.60
July 2021	0.2	0.27	0.4

Table 2. Isolation Rates based on Government Measures in 2021.

For the vaccination rates, we use the same time periods; meaning: $\sigma_v = 0.29$ in January 2021; $\sigma_v = 0.38$ in April 2021; $\sigma_v = 0.61$ in July 2021; and $\sigma_v = 0.69$ in October 2021. (See [19] for more details.)

In Figure (2), the Basic Reproduction Number $\mathcal{R}_0$, was calculated using the values from the COVID-19 data. We note that when the proportion of asymptomatic individuals p increases, the value of $\mathcal{R}_0$ increases, irrespective of different values for the transmission rate from asymptomatic individuals, μ. When the same simulations are performed when media is included in the model, we also note the values for $\mathcal{R}_0 < \mathcal{R}_m$ as p and μ are varied. Thus, our assumption of including media as an important component when modeling in epidemiology has a positive effect especially if people with access to the media follow the regulations given by the health entities.

Notice when the proportion of Quarantined individuals, ϵ, increases, the value of $\mathcal{R}_0$ decreases for different values of μ. When the same simulations are performed for $\mathcal{R}_m$, we also see the values for $\mathcal{R}_0 < \mathcal{R}_m$ as ϵ increases and μ is varied. The sensitivity analyses for the basic reproduction number are included in Figure (3).
Sensitivity Analyses for $\mathcal{R}_0$ and $\mathcal{R}_m$ is performed when varying the recovery rate γ_1 and transmission rate μ from the asymptomatic individuals. The corresponding plots are included in Figure (4). Notice that $\mathcal{R}_0 > 1$; however, when media is included (Model (2), $\mathcal{R}_m$ becomes less than one.
Simulations for Model (1) were performed and shown in Figure (5). We used parameter values from previous literature and health organizations in the States. We see as the number of vaccinated individuals increases, the disease dies out in our population.

A similar behavior is observed when simulations were performed for Model (2). In Figure (6), (including media effects), when comparing the asymptomatic individuals with Model (1), we notice that the maximum number for Model (2) is less than the maximum number for Model (1). We can see that including media has a positive effect on the dynamics of COVID-19.

5 Conclusions

In the eve of *alternative facts*, we have seen an uptick in the distrust of traditionally trusted institutions. We are bombarded with information through social and traditional media outlets and must decipher for ourselves truth versus fiction. What we absorb impacts our behaviors. And our behaviors can severely impact the spread of communicable diseases. Through decades of study of communicable diseases, using traditional compartmental SIR and SEIR modeling techniques, researchers have proven that our actions can affect the spread of the disease. In this case, our actions can be defined by our political persuasions, cultural beliefs, and socio-economic standing. In this study, we explore if our consumption of media does indeed have a *media effect* on the spread of COVID-19. And what is this effect? Does it positively or negatively impact

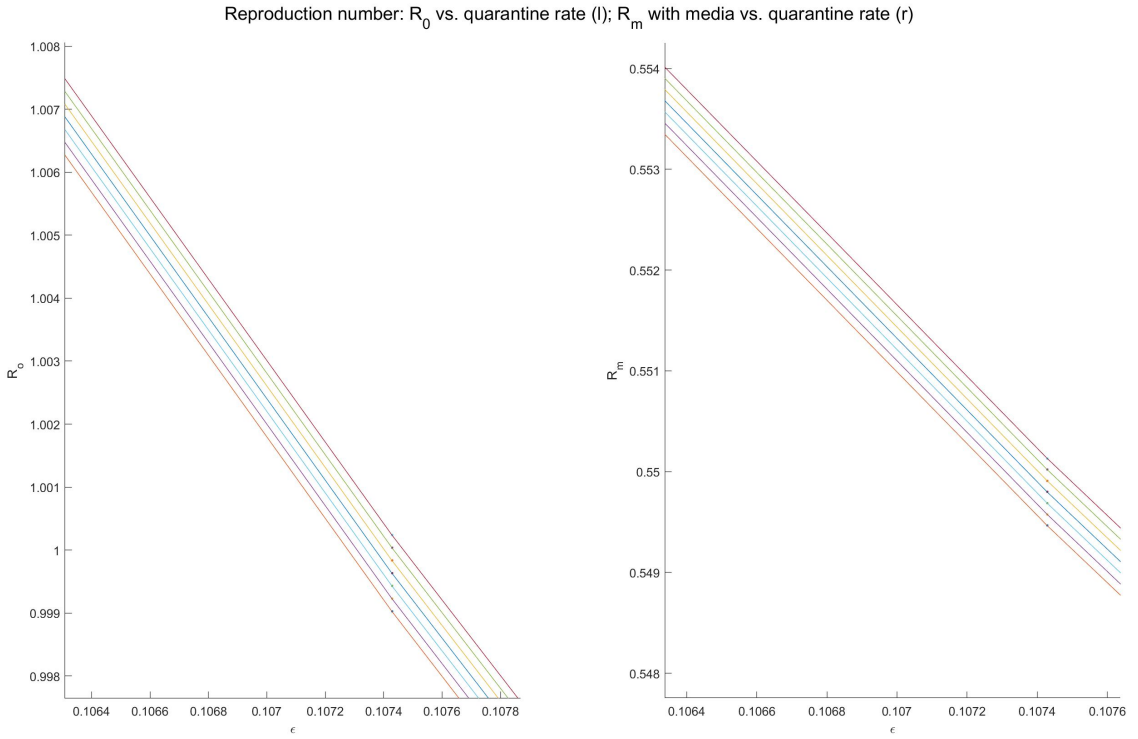

Figure 2. Sensitivity Analyses of the Basic Reproduction Numbers, $\mathcal{R}_0$ and $\mathcal{R}_m$ for Model (1) and Model (2), respectively. The proportion of Asymptomatic individuals p varies in $[0.6 - 0.7]$. The transmission rate of asymptomatic infectives, μ, varies in $[0.02 - 0.05]$.

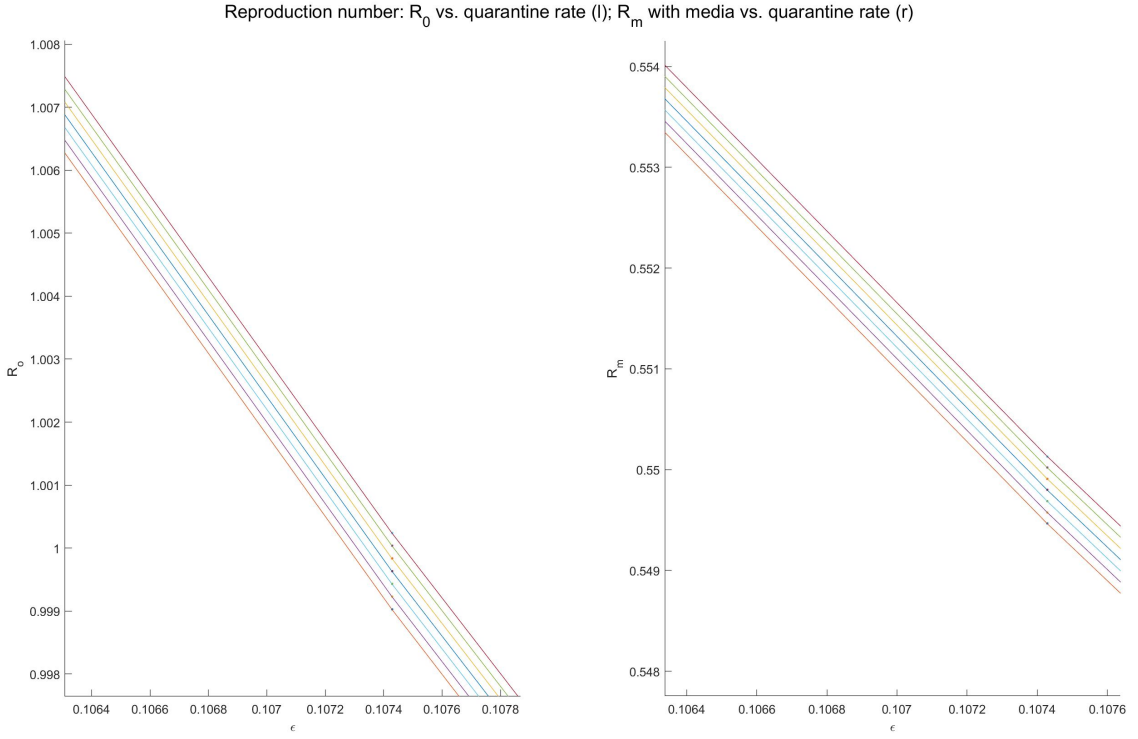

Figure 3. Sensitivity Analyses of the Basic Reproduction Numbers $\mathcal{R}_0$ and $\mathcal{R}_m$ for Models (1) and (2) respectively. The proportion of Quarantine individuals, ϵ, varies in $[0.0714 - 0.1194]$. The transmission rate of asymptomatic infectives, μ, varies in $[0.02 - 0.05]$.

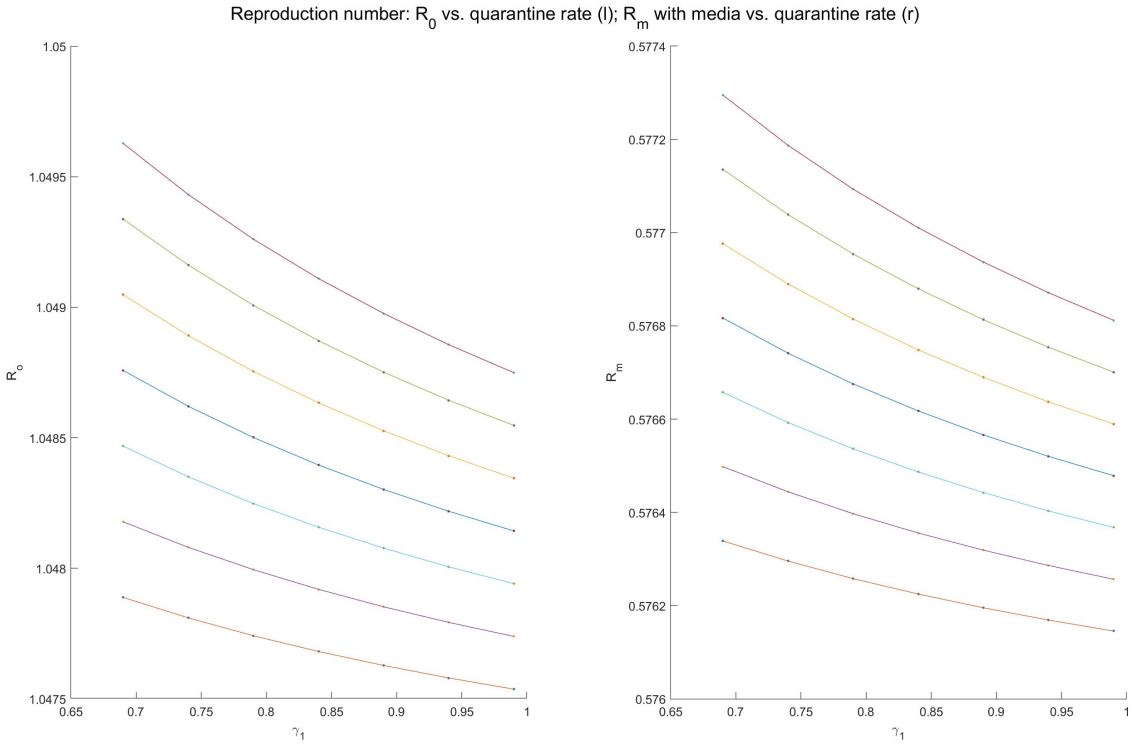

Figure 4. Sensitivity Analyses of the Basic Reproduction Numbers $\mathcal{R}_0$ and $\mathcal{R}_m$ for Models (1) and (2), respectively. The recovery rate for Asymptomatic Individuals, γ_1, varies in $[0.69 - 0.99]$, while the Immunization Efficiency Rate, μ, varies in $[0.02 - 0.05]$.

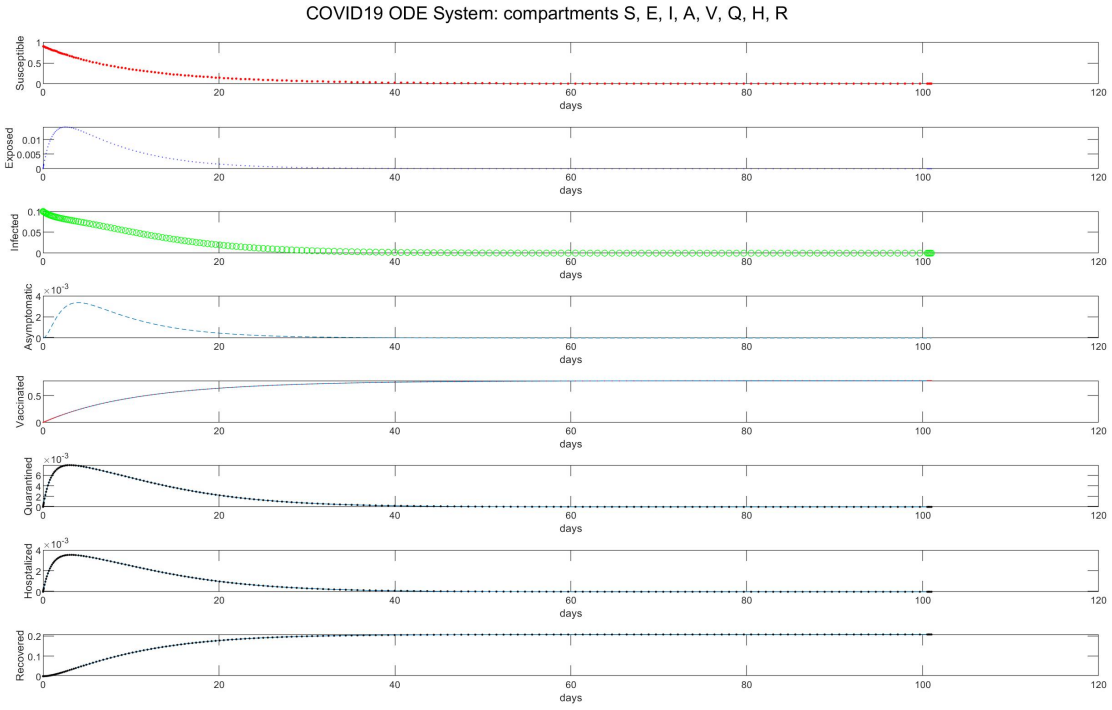

Figure 5. Dynamics of Model (1), including all compartments S, E, I, A, V, Q, H, R, parameters are taken from Table (1).

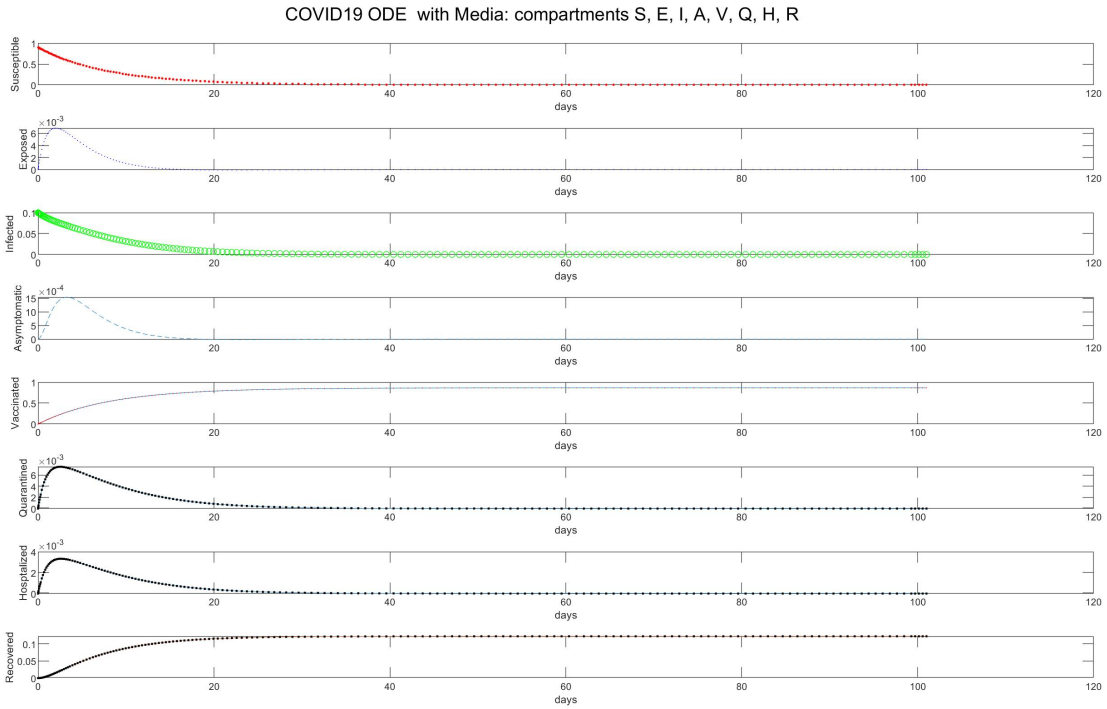

Figure 6. Dynamics of Model (2) with media effect, including all compartments S, E, I, A, V, Q, H, R, parameters are taken from Table (1), with the variations $\tilde{\xi}$, β, and $\tilde{u}$.

our society and protect the most vulnerable in our population? We expand upon the present research on the COVID-19 virus by including the impact an individual's media consumption has on the spread of the disease. Our study introduces a novel approach to understanding the dynamics of COVID-19 transmission, with a specific focus on the influence of media coverage. By employing a system of ordinary differential equations, this research has successfully modeled the interactions between *Asymptomatic*, *Clinically Ill*, *Quarantined*, *Hospitalized*, *Recovered*, and *Vaccinated* individuals. More specifically, we integrate parameters reflecting media impact on disease transmission, providing a unique lens to examine this contemporary issue.

We present two (2) systems of ordinary differential equations modeling the spread of COVID-19: Model (1), without the inclusion of media effect and Model (2), with the inclusion of media effect.

The findings underscore the critical role media plays in shaping public behavior during a pandemic. Notably, the model demonstrates that media coverage can effectively alter the course of COVID-19 spread by influencing public adherence to health guidelines. It is relevant to mention that the inclusion of *Asymptomatic* individuals in modeling infectious diseases is fundamental since a large part of the population can transmit the diseases without showing the symptoms themselves. The simulation results, particularly with the inclusion of media effects in the model, indicate a decrease in the number of asymptomatic individuals, suggesting that informed populations are more likely to engage in preventative measures.

Media effects are also significantly visible from our numerical simulations where we observe that the Basic Reproduction Number becomes lower than one for Model (2), faster than for Model (1), when the proportions of Asymptomatic infectives and quarantined individuals are varied, along with the transmission rates of those asymptomatic carriers. We have shown that individuals will take the information from the media and generally tend to follow the regulations from the health department organizations or government mandates; hence, lowering the intensity of the epidemic.

We hypothesized that the absorption of media played a role in whether or not an individual would become vaccinated. Previous studies as in [20] took a similar approach; however, did not consider vaccination or the role of media. These researchers concluded that "to reduce the burden of the disease on the society, it is necessary to incentivize such altruistic behavior by infected individuals as voluntary self-isolation". They also found that "the second wave of the pandemic can be attributed to rational behavior of susceptible individuals and multiple waves of the pandemic are possible if the rate of social learning of infected individuals is sufficiently high". We assert that this *rational* behavior involves getting vaccinated and maintaining exposure to informative media. A review of

popular mathematical models used to describe the dynamics of COVID-19 can also be found in [21], interesting models using qualitative analysis for COVID-19 have been developed as well [22].

When comparing the dynamics for all the compartments from Model (1) and Model (2), it is observed that the disease dies out in approximately 100 days, but in reality, we experienced more waves of COVID-19 during the year 2020 and 2021. Several reasons can be attributed to these dynamics. For example, the fact that the vaccine was developed and distributed when the disease already reached epidemic levels, the vaccine was not fully approved by health organizations, and misinformation started populating the media, about the negative side effects of the vaccine, among other reasons.

Furthermore, the sensitivity analyses conducted on various parameters like the proportion of asymptomatic individuals and quarantine rates reveal a direct correlation between informed public behavior and the control of disease spread. This aligns with the hypothesis that access to accurate media information increases public compliance with health protocols, thereby reducing transmission rates.

5.1 Managerial Implications

Our model underscores the importance of incorporating media dynamics into epidemiological models, enabling policymakers to make data-driven decisions based on more comprehensive data that includes social behavior and media influence. The findings suggest policies should be tailored to account for the varying effects of media coverage across different demographics and regions. This could involve targeted messaging to address vaccine hesitancy or compliance with public health measures in specific communities. Our study also highlights the need for strategic allocation of resources to enhance media campaigns that promote health awareness, emphasizing the role of clear and consistent messaging in managing public health crises.

We also see the effectiveness of public health campaigns can be increased through enhanced communication strategies by leveraging media influence, as outlined in the paper. This involves using media to disseminate accurate information about COVID-19 transmission, prevention, and vaccination. Public health authorities should monitor and analyze media content to understand its impact on public behavior. This can inform the development of strategies to counter misinformation and promote health-positive behaviors. Also, engaging in partnerships with media outlets can facilitate the dissemination of accurate public health information, ensuring that the public receives consistent messages across different platforms.

Media organizations have a critical role in shaping public perceptions and behaviors during health crises. The findings advocate for responsible reporting practices that prioritize the accuracy and clarity of information related to COVID-19. Media outlets should actively engage with public health experts to provide audiences with reliable and up-to-date information. This can include hosting expert Q & A sessions, debunking myths, and highlighting personal stories to encourage adherence to health guidelines. Recognizing the diverse impact of media on different population segments, media communication should be inclusive and consider cultural sensitivities to effectively reach and influence a broader audience.

Our research provides valuable insights into the complex interplay between media coverage and COVID-19 transmission dynamics. By highlighting the significant influence of media on public behavior and the spread of the virus, we offer a roadmap for policymakers, public health officials, and media practitioners to develop more effective strategies for managing current and future health crises. The managerial implications derived from our findings underscore the need for a collaborative approach that leverages media as a powerful tool in the fight against pandemics, ensuring that communication strategies are data-driven, inclusive, and aimed at promoting public health and safety.

In conclusion, including asymptomatic individuals and media when modeling epidemics contributes to having a better understanding of the dynamics of human diseases. Our research makes a significant contribution to the field of epidemiology by incorporating the dimension of media influence into the modeling of COVID-19 transmission. The study's findings highlight the pivotal role of media in disseminating information that can either curb or accelerate the spread of infectious diseases. The results advocate for responsible media coverage, especially in pandemic situations, where public perception and behavior can dramatically impact the disease dynamics.

6 Study Limitations & Future Work

By incorporating media influence into a compartmental model, we have shed light on the potential mechanisms through which public information dissemination can impact disease spread. However, our research is not without limitations that warrant further discussion.

1. **Model Assumptions**: Our model is predicated on several assumptions that may limit its generalizability. For instance, we assume homogeneous mixing within the population and a uniform impact of media across all demographics. Real-world interactions can be more complex, and media influence can vary significantly among different population segments.

2. **Data Availability**: The effectiveness of media coverage was modeled based on available data, which may not fully capture the nuances of public response to media messaging. Additionally, the model does not explicitly account for the evolution of public health guidelines over time or the emergence of new COVID-19 variants.

3. **Media Coverage Saturation**: Our model does not consider the saturation point of media coverage, beyond

which additional information may not lead to further changes in public behavior. This saturation effect could play a crucial role in shaping the pandemic's trajectory.

To address this, our future epidemic models will incorporate heterogeneity in population and media impact. This could involve segmenting the population by age, socioeconomic status, or geographical location to more accurately model the differential impacts of media. Also, incorporating dynamic elements that reflect the evolution of public health guidelines and the emergence of new viral strains could enhance the model's applicability. This would allow for a more nuanced understanding of how media coverage adapts and influences public behavior over time.

Future work can also be done by analyzing the different strains of COVID-19 and the phases of vaccination programs with media effects.

Further investigation of the saturation effects of media coverage on public behavior could provide insights into optimizing information dissemination strategies. Future studies could explore the thresholds beyond which additional media coverage ceases to have a significant impact. There is also a need for empirical studies to validate the model's predictions against real-world data. This could involve longitudinal studies that track public behavior changes in response to media campaigns. Finally, engaging in interdisciplinary research that combines insights from epidemiology, sociology, and media studies could yield more comprehensive models. Collaborations between public health officials, media practitioners, and researchers are essential to design effective communication strategies.

Our study highlights the potential of media coverage to influence the dynamics of COVID-19 transmission. While our model provides a foundational understanding, it underscores the complexity of the real-world interplay between media and public health. By addressing the outlined limitations and pursuing the suggested future research directions, we can refine our models to better inform policy-making, enhance public health strategies, and optimize media communication practices in the face of ongoing and future pandemics. Our study not only provides a robust mathematical model to understand the complexities of COVID-19 transmission but also opens avenues for further research into the interplay between media influence and public health strategies. As the world grapples with ongoing and future health crises, insights from this study could be instrumental in shaping more effective communication and intervention strategies to manage disease spread.

7 Appendix

In this section, we present the following mathematical results and proofs for Model (1):

1. The Existence and Positively of Solutions.
2. The Stability Analysis of the Disease-Free Equilibrium, DFE.
3. The process to obtain the Final Epidemic Size Relation.

Theorem 1. *Let* $S_0, E_0, A_0, I_0, Q_0, H_0, R_0, V_0 \geq 0$*, with* $S_0 + E_0 + A_0 + I_0 + Q_0 + H_0 + R_0 + V_0 = N_0$*, then there exist non-negative solutions* $S(t)$*,* $E(t)$*,* $A(t)$*,* $I(t)$*,* $V(t)$*,*$Q(t)$*,* $H(t)$*,* $R(t)$ *to Model (1) with initial conditions* S_0*,* E_0*,*A_0*,* I_0*,* V_0*,* Q_0*,* H_0*,* R_0*, defined for all* $t \geq 0$*.*

Proof. The right-hand side of Model (1) is continuously differentiable and hence, it is locally Lipschitz, therefore there exists a unique solution $S(t), E(t), A(t), I(t), V(t), Q(t), H(t), R(t)$ to Model (1), with initial conditions S_0, A_0, I_0, Q_0, H_0, R_0, V_0. Consider the set:

$$\begin{aligned} \Omega \quad = \quad & \{(S, E, A, I, V, Q, H, R) : \\ & 0 \leq S + E + A + I + Q + H + R + V \leq N\}. \end{aligned} \tag{5}$$

The first equation from System (1), is equivalent to:

$$\frac{dS}{S} = -\beta \frac{I + \mu A}{N} - \xi, \tag{6}$$

Then,

$$S(t) = S_0 exp\left\{ - \int \left(\beta \frac{I + \mu A}{N} + \xi \right) dt \right\}. \tag{7}$$

Since $S_0 \geq 0$ then $S(t) \geq 0$. Similarly, from Model (1), specifically the 5th equation, since $S(t) \geq 0$ then:

$$\frac{dV}{dt} \geq -(1 - u)\beta V \frac{I + \mu A}{N}. \tag{8}$$

From here it holds:

$$V(t) \geq V_0 exp\left\{ - \int (1 - u)\beta \Big(\frac{I + \mu A}{N} \Big) dt \right\}, \tag{9}$$

Then $V(t)$ is strictly increasing since $V_0 \geq 0$.

Similar analysis can be carried out for the rest of the equations from Model (1) to prove that all the solutions $E(t)$, $A(t)$, $H(t)$, $Q(t)$, $R(t)$ are non-negative. The $\frac{I(t)}{dt}$ equation can be solved analytically:

$$\frac{dI}{dt} + (\delta_1 + \epsilon + \theta) I = kpE \Rightarrow \tag{10}$$

$$I(t) = \left[I_0 + \int_0^t kpE(\zeta) e^{(\delta_1 + \epsilon + \theta)(\zeta - t_0)} d\zeta \right] e^{(\delta_1 + \epsilon + \theta)(t_0 - t)} \tag{11}$$

Notice since $E(t)$ is non-negative, all the terms inside of the integral are positive. Then if $I_0 \geq 0$, this implies $I(t) \geq 0$, for all $t \geq 0$. □

Theorem 2. *If $\mathcal{R}_0 < 1$, the Disease-Free Equilibrium point, DFE, for Model (1) is locally asymptotically stable. If $\mathcal{R}_0 > 1$, then DFE is unstable.*

Proof. The sub-matrix $J_1(DFE)$, from the Jacobian for Model, (1) evaluated at DFE, includes only the entries corresponding to the new infectious compartments E, I, A. This is given by:

$$J_1(DFE) = \begin{bmatrix} -k & \beta & \beta\mu \\ kp & -(\delta_1+\epsilon+\theta) & 0 \\ k(1-p) & 0 & -\gamma_1 \end{bmatrix}. \quad (12)$$

Using the Routh-Hurwitz Criterion for the characteristic polynomial for $J_1(DFE)$:

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0, \quad (13)$$

The criterion establishes that the eigenvalues for $J_1(DFE)$ have a negative real part if the following conditions are satisfied:

$$a_2 > 0,\, a_0 > 0, \text{ and } a_2a_1 > a_0 \quad (14)$$

where,

$$a_2 = -trace[J_1(DFE)] \text{ and } a_0 = -det[J_1(DFE)]. \quad (15)$$

After some calculations, we found:

$$\begin{aligned} -a_0 &= det[J_1(DFE)] \quad (16)\\ &= k\gamma_1(\delta_1+\epsilon+\theta)\left[\frac{\beta p}{\delta_1+\epsilon+\theta} + \frac{\beta\mu(1-p)}{\gamma_1} - 1\right], \end{aligned}$$

which is equivalent to:

$$\begin{aligned} -a_0 &= det[J_1(DFE)] \\ &= k\gamma_1(\delta_1+\epsilon+\theta)\left[\mathcal{R}_0 - 1\right]. \quad (17) \end{aligned}$$

If $\mathcal{R}_0 < 1$, then $a_0 > 0$. Also,

$$a_1 = (\gamma_1+k)(\delta_1+\epsilon+\theta) + k\gamma_1 - kp\beta - k\beta\mu(1-p) \quad (18)$$

and

$$\begin{aligned} -a_2 &= trace[J_1(DFE)] \\ &= -\left[k + (\delta_1+\epsilon+\theta) + \gamma_1\right] < 0, \quad (19) \end{aligned}$$

which implies $a_2 > 0$. Furthermore, after some computations, $a_2a_1 > a_0$. We now conclude the DFE for Model (1) is locally asymptotically stable. □

Theorem 3. *If $\mathcal{R}_0 \leq 1$, the disease-free equilibrium point for System 1 is globally asymptotically stable. If $\mathcal{R}_0 > 1$, then DFE is unstable.*

Proof. Define the Liapunov functional L over $\Omega_0 \subseteq \Omega$, for $x \in \Omega_0$, as follows:

$$L(x) = \mu(\delta_1+\epsilon+\theta)A + \gamma_1 I. \quad (20)$$

To apply the Liapunov Theorem for global stability, L must satisfy:

(i) $L \in C'(\Omega_0)$, $L(DFE) = 0$, and $L(x) > 0$ for $x \in \Omega_0$.

For Model (1), $I = 0$, and $A = 0$, for the DFE, then $L(DFE) = 0$. Furthermore, since $A > 0$ and $I > 0$ over Ω_0, and all the parameters are positives, then $L(x) > 0$ over Ω_0.

(ii) $\frac{dL}{dt} < 0$ for $x \in \Omega_0$ and $x \neq DFE$.

We see that:

$$\begin{aligned} \frac{dL}{dt} &= \mu\left(\delta_1+\epsilon+\theta\right)\frac{dA}{dt} + \gamma_1\frac{dI}{dt} \Rightarrow \\ \frac{dL}{dt} &= \mu\left(\delta_1+\epsilon+\theta\right)\left[k(1-p)E - \gamma_1 A\right] \\ &\quad +\gamma_1\left[kpE - \left(\delta_1+\epsilon+\theta\right)I\right]. \quad (21) \end{aligned}$$

After some calculations:

$$\begin{aligned} \frac{dL}{dt} &= \gamma_1\left(\delta_1+\epsilon+\theta\right)\cdot \quad (22)\\ &\quad \left[kE\left(\frac{\mu(1-p)}{\gamma_1} + \frac{p}{\delta_1+\epsilon+\theta}\right) - \mu A - I\right]. \end{aligned}$$

Since, $0 < \beta(I+\mu A) - kE$, then:

$$\frac{kE}{\beta(I+\mu A)} < 1. \quad (23)$$

Recall $\mathcal{R}_0 = \frac{\beta p}{\delta_1+\epsilon+\theta} + \frac{\beta\mu(1-p)}{\gamma_1}$. If $\mathcal{R}_0 < 1$, then:

$$kE\frac{\mathcal{R}_0}{\beta} - \mu A - I < 0, \quad (24)$$

which is equivalent to:

$$kE\left[\frac{\mu(1-p)}{\gamma_1} + \frac{p}{\delta_1+\epsilon+\theta}\right] - \mu A - I < 0. \quad (25)$$

It follows that $\frac{dL}{dt} < 0$ for $x \in \Omega_0$ and $x \neq DFE$.

□

A final size relation between the final size of the epidemic and the basic reproduction number (following the general Age-of-Infection approach by Kermack and Mckendrick, [23]) is presented in the following result.

Theorem 4. *The final epidemic size relation for Model (1), for a finite time T, is given by:*

$$\ln\left(\frac{S_0}{S_T}\right) \leq \frac{\beta\varepsilon T}{N_0} + \mathcal{R}_0\left[S_0 - S_T\right]. \tag{26}$$

Proof. The mean infectiousness at age ξ, for the I and A classes and their functions of infected members are defined as:

$$\Lambda_I(\xi) = 1, \qquad \Pi_I(\xi) = pe^{-(\delta_1+\epsilon+\theta)\xi}, \tag{27}$$

$$\Lambda_A(\xi) = \mu, \qquad \Pi_A(\xi) = (1-p)e^{-\gamma_1\xi}. \tag{28}$$

The total mean infectiousness at the infection age, ξ, is defined by:

$$\Gamma(\xi) = \Lambda_I(\xi)\Pi_I(\xi) + \Lambda_A(\xi)\Pi_A(\xi) \tag{29}$$

Then,

$$\Gamma(\xi) = pe^{-(\delta_1+\epsilon+\theta)\xi} + \mu(1-p)e^{-\gamma_1\xi} \tag{30}$$

The Basic Reproduction Number, $\mathcal{R}_0$, can be evaluated as follows:

$$\begin{aligned}\mathcal{R}_0 &= \beta\int_0^\infty \Gamma(\xi)d\xi \\ &= \beta\int_0^\infty \left(pe^{-(\delta_1+\epsilon+\theta)\xi} + \mu(1-p)e^{-\gamma_1\xi}\right)d\xi \\ &= \frac{\beta p}{\delta_1+\epsilon+\theta} + \frac{\beta\mu(1-p)}{\gamma_1}\end{aligned} \tag{31}$$

which agrees with the previous calculation for $\mathcal{R}_0$. We denote the total initial population size as N_0. The Age-of-Infection can be written as:

$$\frac{S'}{S} = \frac{-\beta\psi(t)}{N_0} \tag{32}$$

where,

$$\psi(t) = \psi_0(t) + \int_0^t \frac{\beta}{N_0} S(t-\xi)\psi(t-\xi)\Gamma(\xi)d\xi \Rightarrow \tag{33}$$

$$\psi(t) = \psi_0(t) + \int_0^t -S'(t-\xi)\left[pe^{-(\delta_1+\epsilon+\theta)\xi} + \mu(1-p)e^{-\gamma_1\xi}\right]d\xi. \tag{34}$$

Integrating Equation (32) from 0 to ∞, it was obtained:

$$\int_0^\infty \frac{S'}{S}dt = \tag{35}$$

$$\int_0^\infty \left[\frac{\beta}{N_0}\left(\psi_0(t) + \int_0^t -S'(t-\xi)\left(pe^{-(\delta_1+\epsilon+\theta)\xi} + \mu(1-p)e^{-\gamma_1\xi}\right)d\xi\right)\right]dt, \tag{36}$$

Then,

$$\int_0^\infty \frac{S'}{S}dt = \tag{37}$$

$$\int_0^\infty \frac{\beta}{N_0}\psi_0(t)dt + \mathcal{R}_0\int_0^\infty -S'(t-\xi)d\xi. \tag{38}$$

Since, $|\psi_0(t)| = |I_0(t) + \mu A_0(t)|$, and $|I_0(t) + \mu A_0(t)| \leq \varepsilon$, for some positive ε, then for a finite time T, the final epidemic size relation for Model (1), is:

$$\ln\left(\frac{S_0}{S_T}\right) \leq \frac{\beta}{N_0}\varepsilon T + \mathcal{R}_0[S_0 - S_T]. \tag{39}$$

□

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