Pandemic Flu Simulation And The Impact Of Ultraviolet Light On Reproduction Ratio

Nathan Kurtz-Enko

Table of contents

Abstract

Influenza and similar viruses represent a threat to the health of populations across the globe. Therefore, it is important to study the behavior of these viruses and the impact of mitigation strategies on them. Compartmental and stochastic models have been used to show that mitigation strategies like vaccination, prophylaxis antiviral agents, and travel restrictions can have some impact on or fully contain the spread of influenza. While some strategies have been more widely studied, others, such as ultraviolet (UV) light exposure, have received less attention. We examined the number of days of UV treatment required to contain a flu pandemic in the

United States of America with an initial reproduction ratio of $R_0 = 1.8$. During a 100 day simulation, beginning once 5% of the overall population had been infected, we determined that roughly 30 days of UV treatment would reduce the reproduction ratio of influenza to approximately $R_0 \approx 1$, meaning that the spread of the virus is no longer in an outbreak status.

Introduction

Understanding the spread of infectious diseases, like the flu, is critical for advising policy making with respect to public health and wellness. For example, studying the behavior of virus transmission and recovery rates allows health professionals and policy-makers to define the quantity of antiviral agents that should be readily available, regions and demographics that should be prioritized for vaccination, and other strategies intended to prevent a pandemic. Furthermore, being able to communicate the results of an infectious disease model/simulation is very important, as this will facilitate the adoption of containment measures.

Many researchers studying the spread of viruses explicitly cite the 1918 influenza outbreak as a motivating factor for their work (Berkessel et al. 2021; Germann et al. 2006; Johnson and Mueller 2002; Valle, Mniszewski, and Hyman 2012). In the case of Johnson and Mueller (2002) and Patterson and Pyle (1991), the objective was to look back through previous records and data to verify morbitity of the 1918 pandemic. Most recently, Johnson and Mueller (2002) estimated the death toll from the 1918 influenza was on the order of 50 million people.

Clearly, the capacity for large-scale damage from infectious disease outbreaks is a very real threat. Hence, understanding how viruses spread and the impact that different mitigation strategies have on the rate of spread and severity of illness is critical.

Literature Review

In the 1980s, Russian scientists first began to implement models for estimating the spread influenza and other viruses (Longini, Fine, and Thacker 1986). These early models were deterministic, and they described the movement of viruses using differential equations, and partitioned populations into several subgroups. These are referred to as SIR (i.e., susceptible, infected, and recovered groups), SEIR (i.e., susceptible, exposed, infected, and recovered groups), and other derivatives.

Initial reactions to these determinsitic models by academics were skeptical for a few reasons. First, geographic regions being studied were relatively isolated, hence the ability to apply these models for global study was uncertain (Longini, Fine, and Thacker 1986). Second, for the time, the models were computationally expensive, meaning it was impractical to use them.

So, academics remained skeptical of their applicability for real-world use. However, this is less of an issue today because high-performance computing is readily available.

While relatively old, determinstic models still have utility. Valle, Mniszewski, and Hyman (2012) used an SIR model to show that an individual's behavior will likely change in response to an outbreak; some individuals will adopt new behavior to protect themselves, thereby influencing the probability of transmission per contact.

More recently, stochastic models have become prevalent because they are able to capture more complex spreading patterns. Colizza et al. (2007), Cooper et al. (2006), Ferguson et al. (2005), Germann et al. (2006), Samsuzzoha, Singh, and Lucy (2013), and Tan et al. (2021) all used stochastic processes to model the spread of influenza or other infectious diseases, and analyze different mitigation strategies. Colizza et al. (2007) found that a large-scale application of prophylaxis antiviral agents could successfully contain an H5N1 influenza pandemic given a viral reproduction ratio of 1.9 or lower. Cooper et al. (2006) found that significant air travel restrictions could delay the spread of influenza but not contain an epidemic altogether. The findings of Germann et al. (2006) determined that rapid and preferential vaccination of children could contain an outbreak when the reproduction ratio of influenza is at or below 1.9.

Others have explored the relationship between geographic variables (e.g., altitude and solar radiation) and incidence rates for COVID-19, as well as severity of illness (Arias-Reyes et al. 2021; Stephens, Chernyavskiy, and Bruns 2021). Arias-Reyes et al. (2021) concluded that above 1000 meters above sea level, incidence rates and severity for COVID-19 decline. Through analysis of public geographic and COVID-19 data, Stephens, Chernyavskiy, and Bruns (2021) determined that for every increase of 495 meters in elevation above Los Angeles County, and for areas of equivalent population density, infection rates were 12.82%, 12.01%, and 11.72% respectively. However, mortality rates were not statistically significantly different between high and low elevation areas. Stephens, Chernyavskiy, and Bruns (2021) note that other environmental variables such as increased solar radiation, larger swings of high/low temperatures throughout the day, and long-term exposure to altitude hypoxia could explain these observed differences.

Since influenza and similar viruses spread via aerosols, Sagripanti and Lytle (2007) proposed solar radiation as a contributing factor to the seasonality of influenza. Specifically, the authors calculated the expected inactivation of influenza by UV radiation in several cities and different times of the year. They estimated that a full day of sunlight exposure will reduce influenza by 99% in regions at a similar latitude to Mexico City, Mexico, and by 90% in regions at a similar latitude to Miami, Florida. Jensen (1964) found that aerosol influenza A, when exposed to 0.03 watt-minutes per square foot (i.e., approximately 19.41286 Joules per square meter) of UV light, had an inactivation of greater than 99.9% at a flow rate 100 cubic feet per minute, and approximately 99.86% inactivation at 200 cubic feet per minute.

Therefore, higher altitude, higher solar radiation, and higher UV light exposure could theoretically influence the spread of influenza. The purpose of this paper is to simulate flu outbreak

and identify critical thresholds for some of these covariates that can be use to minimize the risk of a pandemic.

Method

In this analysis, we used Epiverse-TRACE (Gupte, Eggo, and Van Leeuwen 2025; Lambert, Kucharski, and Tamayo 2025; Gupte, Van Leeuwen, and Kucharski 2025), Tidyverse (Wickham et al. 2025; Wickham and Henry 2025; Wickham, Vaughan, and Girlich 2025; Wickham, Hester, and Bryan 2024; Wickham 2016), and other tools (Rudis 2022; Walker and Herman 2025; Xie 2025) in the R programming language. The objective was to use contact and population estimates to implement an SEIR mode and simulate the spread of influenza in the United States of America.

Model Theory

As previously alluded, there are two general approaches to simulating virus spread: deterministic and stochastic models. Here, we present the SIR model (Débarre, n.d.; "An Introduction to Deterministic Infectious Disease Models," n.d.) because it is relatively simple, and this establishes a foundation for considering other approaches.

The SIR model categorizes a population into three groups: susceptible, infected, and recovered. The rate of change of each of these groups is defined using differential equations, and we can account for mortality and birth rates in their definitions as well.

$$\begin{aligned}
\frac{dS}{dt} &= \delta - \frac{\beta \times S \times I}{N} - \mu \times S \\
\frac{dI}{dt} &= \frac{\beta \times S \times I}{N} - (\gamma + \mu) \times I \\
\frac{dR}{dt} &= \gamma \times I - \mu \times R
\end{aligned}$$

Here, S, I, R stand for the susceptible, infected, and recovered populations respectively. The total population is denoted by N, and the rates of transmission, recovery, mortality, and birth are denoted by β, γ, μ, and δ.

The outbreak status is described by the reproduction ratio, R_0, which is the ratio between the transmission rate and recovery rate. When $R_0 = \beta/\gamma > 1$, more people are becoming infected than are recovering, hence the system is in a state of outbreak. And, supposing that there is some percentage of the population that is already vaccinated (i.e., already in the recovered group), this ratio becomes:

$$R_0 = (\beta \times S(0))/(\gamma \times N)$$

Here, $S(0)$ is the number of people in the susceptible group at time $t = 0$.

These facts make it easy to define different transmission, recovery, and vaccination target rates to avoid a significant outbreak. Hence, the SIR is a simple model, but effective at clearly communicating actionable results to policy makers and authority figures.

Data

We used contact data collected by Breen, Mahmud, and Feehan (2022) for different age groups across the United States of America. Additionally, we accessed U.S. Census results from 2022 via Walker and Herman (2025) to estimate overall populations for each of the age groups defined by Breen, Mahmud, and Feehan (2022). We aggregated the state-wide Census results population counts for all ages, ranging from one to 100, into bins defined in the contact data. The data are displayed in Table 1 and Table 2.

Table 1: USA National Contact Matrix

	[0,18)	[18,25)	[25,35)	[35,45)	[45,55)	[55,65)	[65,100]
[0,18)	47402.285	24645.765	31405.06	43077.563	25249.87	9696.327	5821.438
[18,25)	10550.575	43999.421	15290.51	9758.546	10319.81	5849.805	3840.709
[25,35)	19076.694	21417.483	55106.90	23122.012	17274.64	13386.274	6885.919
[35,45)	23716.460	12519.840	21174.81	45537.642	20427.31	11326.252	9290.538
[45,55)	14583.902	13840.380	16543.69	21371.308	34937.29	17599.507	9497.310
[55,65)	5625.521	7913.416	12956.29	11994.823	17783.26	20681.963	12011.882
[65,100]	4015.851	6154.390	7929.90	11646.666	11359.92	14203.840	27637.752

Table 2: USA National Population Estimates

Age Group	Population
[0,18)	72450827
[18,25)	31328131
[25,35)	45501300
[35,45)	43695365
[45,55)	40431645
[55,65)	42085437
[65,100]	57794852

Initial Conditions

There are two approaches to defining initial conditions (e.g., number of members in each model subgroup at initialization, influenza spread parameters, etc.) for the model: define some reasonable values or derive values from current/historic flu data. For convenience, we used the former approach and used values for rates discussed in literature.

Nikbakht, Baneshi, and Bahrampour (2018) provided multiple estimates for the basic reproduction ratio of influenza in the United states given 2017-2018 data. Specifically, using Maximum Likelihood Estimation, they estimated $R_0 = 1.8$ with a 95% confidence interval of $[1.78, 1.8]$.

Virlogeux et al. (2015) provided an estimate for the incubation period of influenza. Specifically, they noted that incubation period follows a Weibull distribution with shape = 2.101 and scale = 3.839. We used the expected value of the incubation period probability distribution.

$$E[\text{Incubation Period}] = \text{scale} \times \Gamma\left(1 + \frac{1}{\text{shape}}\right) = 3.400167 \text{ days}$$

Tang et al. (2020) noted that the inverse incubation period is equivalent to the infection rate, therefore $\alpha = 0.2941032$.

According to CDC (2024), the infectious period of influenza is about five to seven days typically. For the purpose of this paper, we assumed a worst case scenario and used an infectious period of seven days. Turinici (2025) stated that the inverse of the infectious period is equivalent to the recovery rate, therefore $\gamma = 0.1428571$.

The final parameters are collected and displayed in Table 3.

Table 3: Parameters for SEIR Model

Name	Symbol	Value
Reproduction Ratio	R_0	1.8000000
Infection Rate	α	0.2941176
Recovery Rate	γ	0.1428571
Transmission Rate	β	0.2571429

In order for the flu to progress through a population, there must be some members in the infected group at the start of the simulation. We assumed that 5% of the population is already infected. Furthermore, we assumed that this proportion of infected individuals was consistent across all age groups.

Mitigation Strategies

As noted by Jensen (1964) and Sagripanti and Lytle (2007), solar radiation and ultraviolet light can effectively inactivate influenza. One mitigation strategy to limit the spread of influenza during a pandemic could be UV light exposure to sanitize air and surfaces in environments that many people regularly interact with.

Since Jensen (1964) determined that approximately 19 Joules per square meter of UV light results in over 99% viral inactivation for aerosol influenza, and since influenza often spreads via an aerosol mechanism, we assumed a best case scenario, that the transmission rate would be reduced by 99% for each application of the UV light intervention. The objective was to determine the number of days of this measure that reduces the reproduction ratio to approximately $R_0 \approx 0$.

Andreasen (2011) defined the relationship between basic reproduction ratio and the final size of the epidemic. We used a simplified implementation of this work to estimate the reproduction ratio at the end of the simulations before and after the UV light intervention.

$$R_0 = ln\left(\frac{S_{100}}{S_0}\right) / \left(1 - \frac{S_{100}}{S_0}\right)$$

Here, S_0 is the population of susceptible individuals at time $t = 0$ and S_{100} is the population of susceptible individuals at time $t = 100$ (i.e., the end of the simulation).

Discussion

In this paper, we implemented a deterministic SEIR model to study the spread of influenza rather than a stochastic model. This was due to time constraints, the simplicity of the model, and ease of communicating the process and results. Additionally, we examined the impact of UV light exposure on the spread of influenza in the United States of America given contact data and epidemic rates defined in literature.

Simulation Results

We ran this simulation iteratively, with each iteration pertaining to an additional day of UV light intervention. We compared the reproduction ratio at the end of each iteration against each other and a base output in which there are no intervention measures taken. Our objective was to reduce the reproduction ratio of spreading influenza to a contained rate (i.e., $R_0 \approx 1$).

We observed that after about 30 days of 19 Joules per square meter of UV light exposure, the reproduction ratio of the outbreak approaches $R_0 \approx 1$, indicating there is less benefit

to additional application of UV light. Specifically, at the 30th day, the reproduction ratio estimate was 1.0790409. These results are presented in Figure 1.

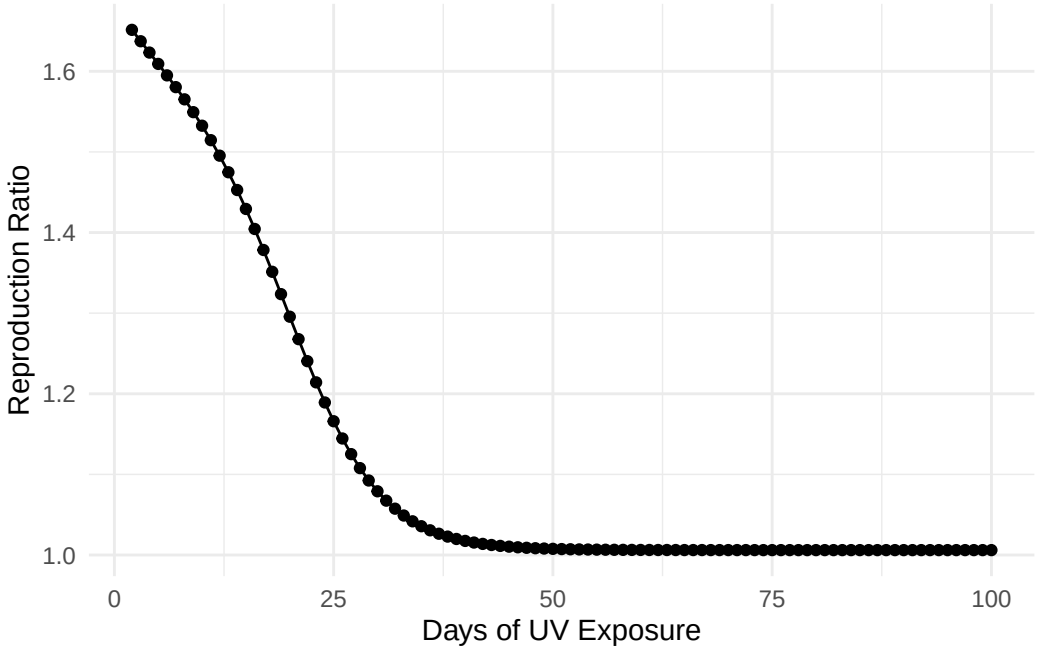

Figure 1: Reproduction ratios from iterative influenza simulation with UV light intervention spanning 100 days.

The simulation results of our base case and the 30 days of UV light exposure are presented in Figure 2 and Figure 3, and show a clear difference in the spread of influenza, with the latter visualization depicting a well controlled virus over the course of the 100 day simulation.

Conclusions

Assuming that the initial reproduction ratio, transmission rate, infection rate, and recovery rate of an influenza outbreak are respectively approximately 1.8, 0.2571429, 0.2941176, and 0.1428571, then the results of this simulation suggest that an application of 19 Joules per square meter of UV exposure to air and surfaces that populations regularly interact with for 30 days after 5% of the population is infected should reduce the reproduction ratio to approximately 1.0790409. This suggests that using UV light exposure as a mitigation strategy could contribute to containing an influenza pandemic.

In this paper, we did not account for heterogeneity in age groups and contacts when estimating the reproduction ratio after mitigation strategies are applied. Furthermore, our results were not validated against real world case data. Therefore, additional work to conclusively state the

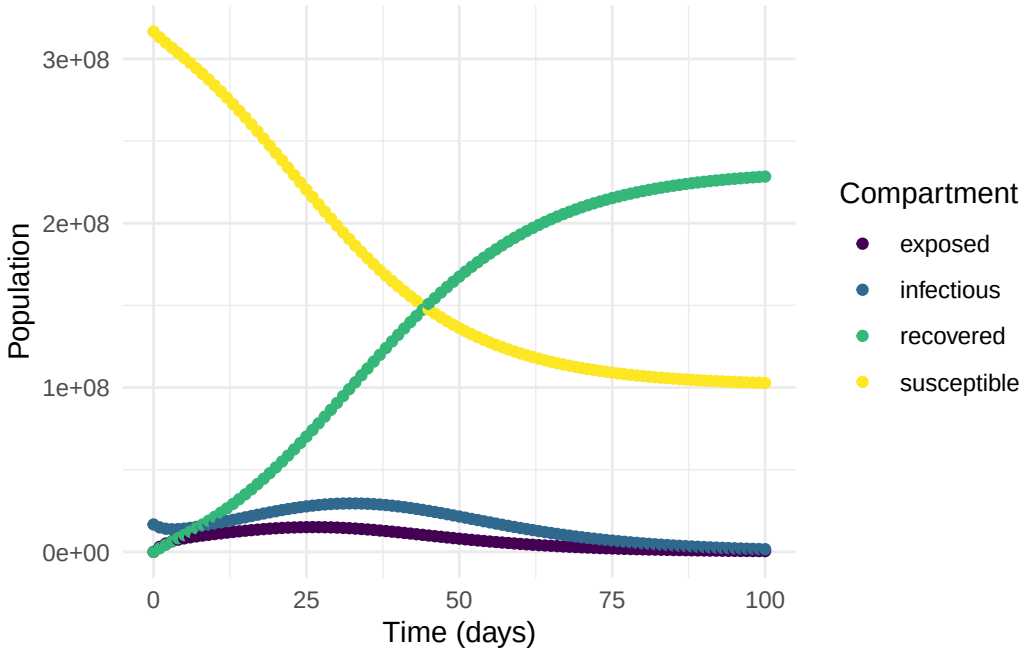

Figure 2: Influenza simulation output results without any intervention and spanning 100 days.

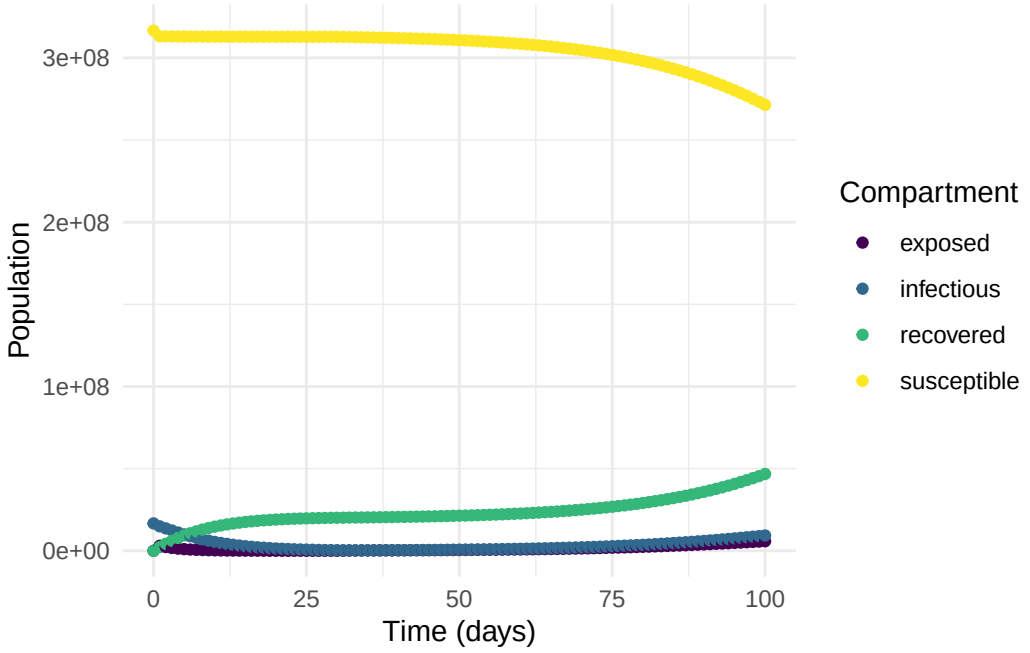

Figure 3: Influenza simulation output results with UV light intervention spanning until day 30 of the 100 day simulation.

impact of UV light exposure as a mitigation strategy should be performed. Also, future work could involve stochastic simulaiton and combined epidemiology, climatology, and public health models to capture and study more complex interactions between individuals, the environment, and mitigation strategies. Finally, the practicality of implementing UV light exposure as a mitigation step was not discussed.

So, while not definitive, this simulation does generally suggest that UV light can be used to mitigate/contain an influenza epidemic.

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