ASSESSING POTENTIAL COVID-19 OUTCOMES FOR A UNIVERSITY CAMPUS WITH AND WITHOUT PHYSICAL DISTANCING

Robert Smith?

Department of Mathematics and Faculty of Medicine
The University of Ottawa
150 Louis-Pasteur Pvt
Ottawa ON K1N 6N5
rsmith43@uottawa.ca

ABSTRACT

By early March of 2020, it became apparent that COVID-19 was going to have a significant impact on institutions throughout society. On March 12, the University of Ottawa commissioned an informal modeling study to aid in their decision-making process. Here we present the results from that study: a differential-equation model was created in order to describe the states of susceptibility, exposure, infection, asymptomatic individuals, recovery and death. Our results shows that, in the worst-case scenario, the epidemic would peak at 7000 cases on campus with 63 dead, starting 115 days after the first case. Reducing contacts by 50% could lower the number of cases and fatalities, but it would expand the timeframe by several years. One day after this modeling study was completed, the University of Ottawa closed campus entirely. It follows that modeling and simulation in the midst of a fast-moving pandemic can be valuable tools for decision-makers.

Keywords: COVID-19, university campus, differential equations, reproduction number.

1 INTRODUCTION

COVID-19 is a respiratory disease with flu-like symptoms that is the causative agent of a potentially fatal disease that has significant public-health concerns (Rothan and Byrareddy 2020). It originated and gained traction in the city of Wuhan, in the Hubei Province of China at the end of 2019 (Liu, Gayle, Wilder-Smith, and Rocklöv 2020) and was first identified in early December 2019 (Guan et al. 2020). The most common symptoms at onset of COVID-19 illness are fever, cough and fatigue, while other symptoms include sputum production, headache, haemoptysis, diarrhea, dyspnoea and lymphopenia (Ren et al. 2020), (Huang et al. 2020), (Wang, Tang, and Wei 2020), (Carlos et al. 2020). The period from the onset of COVID-19 symptoms to death, depending on the age of the patient and their immune status, ranges from 6 to 41 days, with a median of 14 days (Wang, Tang, and Wei 2020).

There were 425 laboratory-confirmed cases in Wuhan on January 22, 2020, leading to an initially estimated reproduction number of 2.2 (95% CI, 1.4 to 3.9) and a doubling time of 7.4 days (Li et al. 2020). By February 16th, the number of cases had climbed to 51,857 (Rothan and Byrareddy 2020). By March 3rd, 90,870 cases of COVID-19 had been confirmed (Sohrabi et al. 2020). Multiple

SummerSim'20, July 20–July 22, 2020, Virtual Event;

countries have since instituted temporary restrictions on travel with an eye toward slowing the spread (Fauci, Lane, and Redfield 2020).

Mathematical models can aid in predictions during the early stages of pandemics. Early COVID-19 models have determined patterns of growth (Tribe and Smith? 2020), the speed of spread (Zhang et al. 2020), assessed existing outbreaks (Kucharski et al. 2020), made long-term predictions (Atkeson 2020) and examined control strategies such as isolation of cases (Hellewell et al. 2020) or reduced social mixing (Prem et al. 2020).

On March 12, 2020, the University of Ottawa (Canada) requested an informal modeling study in order to determine answers to the following questions: 1. What combinations of transmission rates and serial intervals and so on make for the best case, and which for the worst? 2. What is the consequence for outbreak size and potential mortality for waiting until case #1 physically appears with symptoms on campus, versus being proactive? Here we present the results of that study.

This paper is organized as follows. In Section 2, we introduce the mathematical model. In Section 3, we describe the data — as best we knew it on March 12, 2020 — and its sources. In Section 4, we present the simulations of various scenarios. In Section 5, we discuss the practical outcome of this result. In Section 6, we examine some alternative scenarios not considered at the time and discuss the ensuing global changes since the original study took place. We conclude with a discussion.

2 THE MATHEMATICAL MODEL

We propose an SEIARD model, consisting of six compartments: susceptible individuals (S), exposed individuals (E), symptomatic individuals (I), asymptomatic individuals (A), recovered individuals (R) and dead individuals (D). We define symptomatic individuals as those who have symptoms and are capable of transmitting the virus; asymptomatic individuals can also transmit, but likely at a lower rate; these individuals may recover faster than symptomatic individuals. Exposed individuals are infected but not yet infectious. We consider a campus population of fixed size, so that death is only due to the disease, and there is no replacement of susceptible individuals.

We consider a baseline transmission rate β, which will be lowered by a factor $\eta < 1$ for asymptomatic transmission. A fraction p of exposed individuals will move to the asymptomatic class at rate κ, while a fraction $(1-p)$ will move to the symptomatic class. Symptomatic individuals leave at rate μ; a fraction f will recover, whereas a fraction $(1-f)$ will die. Recovery of asymptomatic individuals may be amplified by a factor $\alpha \geq 1$.

The model is given by

$$\begin{aligned} S' &= -\beta SI - \eta\beta SA \\ E' &= \beta SI + \eta\beta SA - \kappa E \\ I' &= (1-p)\kappa E - \mu I \\ A' &= p\kappa E - \alpha\mu A \\ R' &= f\mu I + \alpha\mu A \\ D' &= (1-f)\mu I. \end{aligned}$$

The model is illustrated in Figure 1.

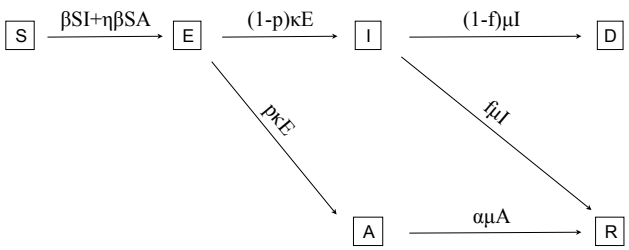

Figure 1: Flow diagram of the model

3 DATA

The fraction of the population that is asymptomatic is 17% (Mizumoto, Kagaya, Zarebski, and Chowell 2020), so $p = 0.17$. As of March 12, 2020, there had been 4970 deaths and 68,900 recovered cases (Worldometer 2020), suggesting a death rate of 6.28%. However, the campus population is young, with the bulk of individuals under 40, so we used the case-fatality rate of 0.2% for these individuals (Feng et al. 2020).

The incubation period has been reported as 3 days (range 0–24 days) or 5.2 days (range 4.1–7 days). We used the smaller value, since it was published later, so that $\kappa = 1/3$ days^{-1}. It takes 20 days (range 10–41 days) from the onset of symptoms to death (Wang, Tang, and Wei 2020) among people under 70, so $\mu = 1/20$ days^{-1}. The reproduction number has been estimated at 2.35 (range 1.15–4.77) (Kucharski et al. 2020). The total campus population is approximately $N = 42{,}000$ (uOttawa 2014).

Note: In the early stages of a fast-moving epidemic, data is often scarce or unreliable. These numbers are the most accurate that were available at the time. Furthermore, not all individuals will be on campus at any given time, and there is significant heterogeneity in the susceptible population, so N is likely an overestimate.

4 RESULTS

The disease-free equilibrium is given by

$$(S, E, I, A, R, D) = (N, 0, 0, 0, 0, 0),$$

where N is the total campus population. We used the initial conditions

$$\begin{aligned} S(0) &= N & E(0) &= 0 & I(0) &= 1 \\ A(0) &= 0 & R(0) &= 0 & D(0) &= 0. \end{aligned}$$

Using the next-generation method (van den Driessche and Watmough 2002), we have

$$F = \begin{bmatrix} 0 & \beta N & \eta\beta N \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \qquad V = \begin{bmatrix} \kappa & 0 & 0 \\ -(1-p)\kappa & \mu & 0 \\ -p\kappa & 0 & \alpha\mu \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta N(1-p)}{\mu} + \frac{\eta\beta N p}{\alpha\mu} & \frac{\beta N}{\mu} & \frac{\eta\beta S}{\alpha\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

The reproduction number is thus

$$R_0 = \frac{\beta N(1-p)}{\mu} + \frac{\eta\beta Np}{\alpha\mu}.$$

The first term consists of the contributions from symptomatic individuals, while the second term consists of contributions from asymptomatic individuals.

Substituting the data and rearranging, we have

$$\beta = \frac{2.35\alpha}{697200\alpha + 142800\eta}$$

We use several possible scenarios to calculate the transmission rate:

1. Asymptomatic individuals are identical to symptomatic individuals ($\eta = \alpha$). In this case, $\beta = 2.7976 \times 10^{-6}$.
2. Asymptomatic individuals do not infect susceptibles ($\eta = 0$). In this case, $\beta = 3.3706 \times 10^{-6}$.
3. Asymptomatic individuals are 50% as transmissible as symptomatic individuals but recover twice as fast ($\eta = 0.5, \alpha = 2$). In this case, $\beta = 3.20644 \times 10^{-6}$.

Using different values of β, we ran several simulations to determine possible long-term outcomes: (i) in the absence of interventions and (ii) if contact rates are cut by 50%.

4.1 Potential scenarios

Figure 2 illustrates the case when the transmission is high and in the absence of interventions. In this case, there is a peak of 6000 symptomatic cases on campus, culminating in 63 deaths. The first death occurs 115 days after the first symptomatic case, and the epidemic last about a year. This is the worst-case scenario.

Figure 3 illustrates the case when transmission is at the low end of the spectrum, in the absence of interventions. In this case, the peak increases to 7000 symptomatic cases and the outbreak lasts about 400 days, but the deaths decline to 58, and the first death does not occur until 150 days after the first symptomatic case. The increased number of symptomatic cases and longer timespan are due to the lowered death rate; more surviving individuals makes the disease last longer and ultimately infects more people.

Figure 4 illustrates the case when transmission is high but the number of contacts is reduced by half. In this case, the number of cases peaks at 700, with only 25 dead, but the effect is a long delay: the first death occurs after 450 days, and the epidemic lasts for 3 years.

Figure 5 illustrates the case when transmission is at the low end of the spectrum and when the number of contacts is reduced by half. In this case, the number of cases is kept below 25, with only 5 deaths. However, the outbreak period is extremely long: the first death occurs more than 5 years after the first symptomatic case, while the outbreak lasts 13 years. This is the best-case scenario, in terms of cases and deaths. It follows the reduction of contacts is critical.

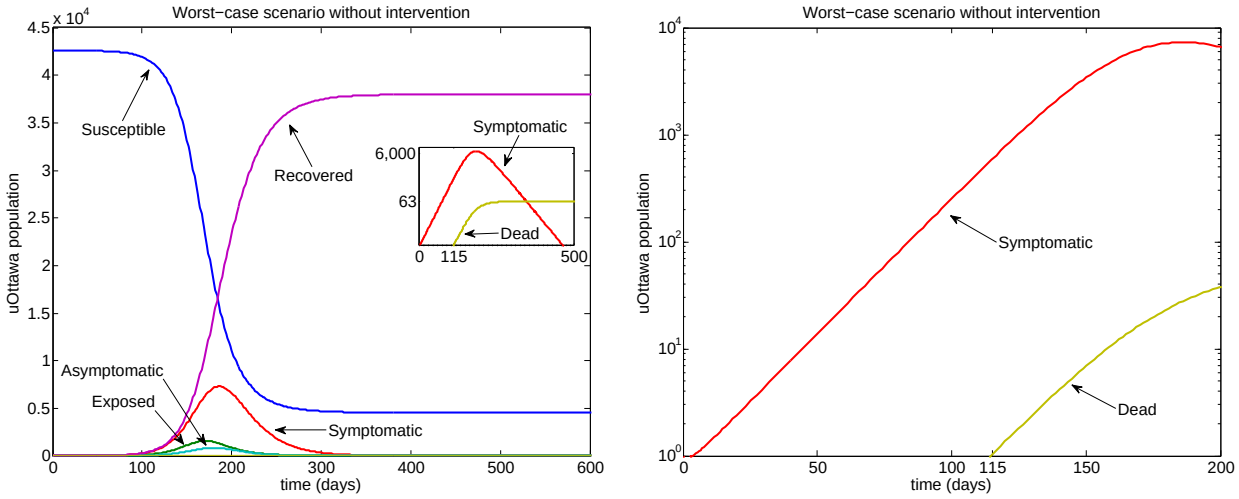

Figure 2: In the case of high transmissibility, without intervention, we predict the first death occurring 115 days after the first symptomatic individual is diagnosed, eventually leading to 63 deaths. (Note that the inset diagram and the figure on the right are both on a log scale.)

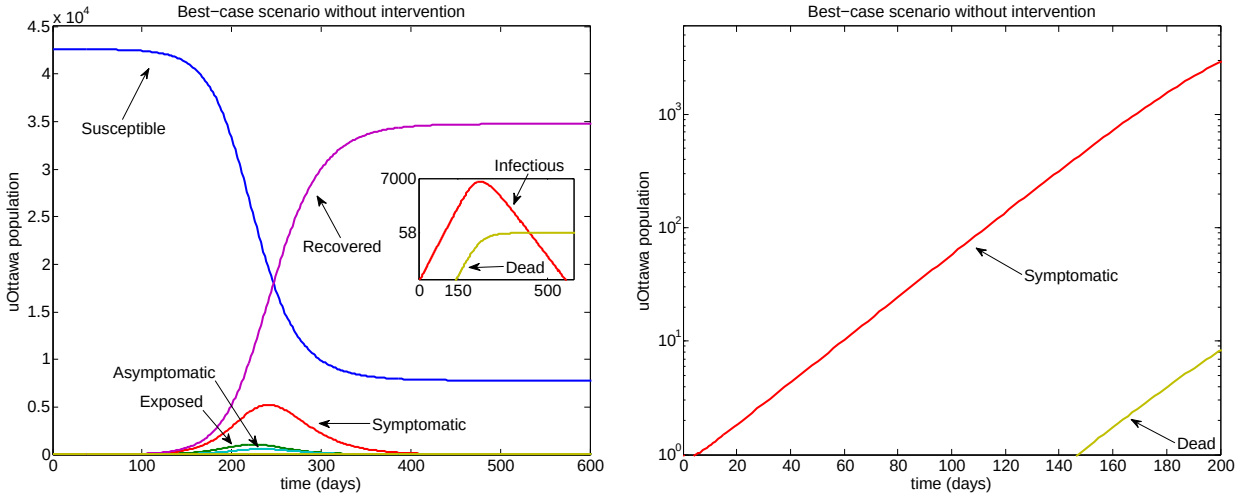

Figure 3: In the case of low transmissibility, without intervention, we predict the first death occurring 150 days after the first symptomatic individual is diagnosed, eventually leading to 58 deaths.

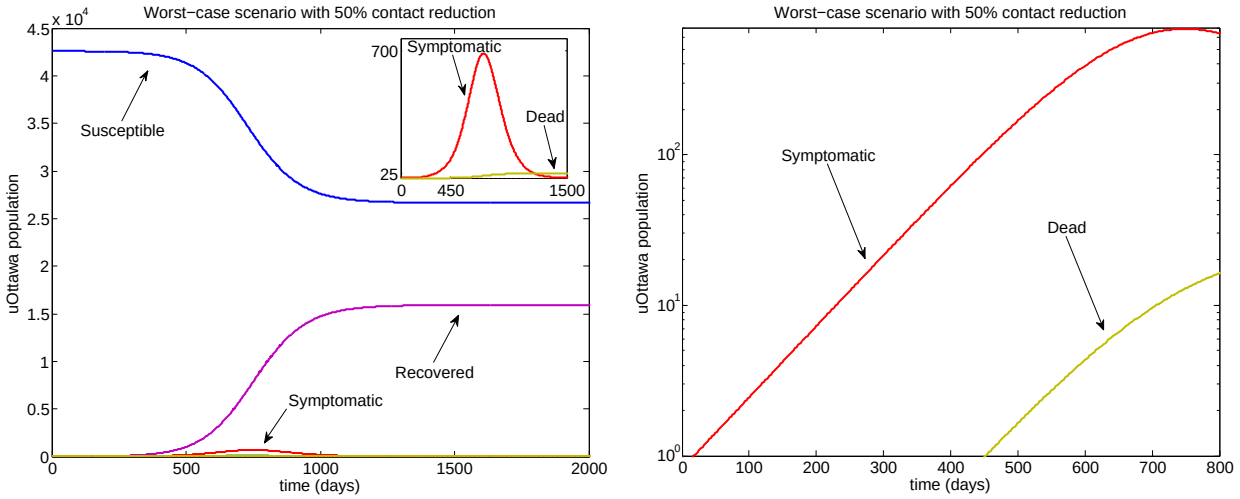

Figure 4: In the case of high transmissibility, with 50% reduction in contacts, we predict the first death occurring 450 days after the first symptomatic individual is diagnosed, eventually leading to 25 deaths.

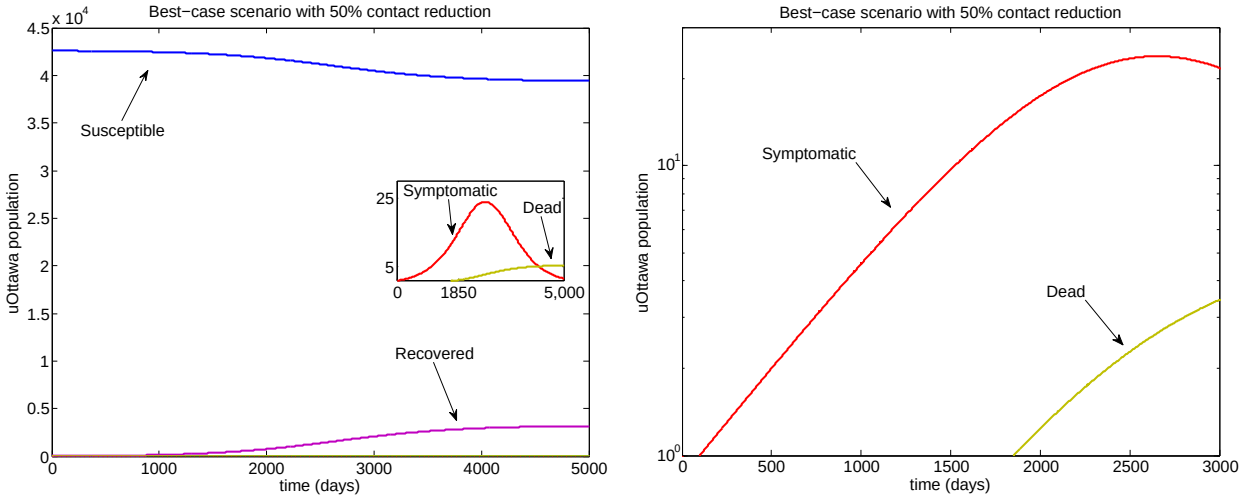

Figure 5: In the case of low transmissibility, with 50% reduction in contacts, we predict the first death occurring 1850 days after the first symptomatic individual in diagnosed, eventually leading to 5 deaths.

5 OUTCOME

On March 13, one day after receiving these results, the university closed campus entirely. It remains closed, except for essential research (e.g., labs that cannot be left unattended), at time of writing, more than three months later, with the Summer 2020, Fall 2020 and Winter 2021 semesters scheduled to be given online.

These results illustrate the interface between theoretical mathematics, numerical simulations, real-world data and human behavior. By requesting a modeling study during the early stages of a pandemic, a variety of potential scenarios could be assessed quickly, using the information available at the time. Decisions could then be made by the administration, armed with useful predictions.

6 ROADS NOT TAKEN

A great deal of information has come to light about COVID-19 since March 12. The subsequent months of global lockdown have changed the course of these predictions. While there have been no cases or deaths on campus since (due to its closure), the outbreak in Ottawa has followed the typical age distribution for COVID-19, with significant numbers of infections in hospitals, retirement homes and long-term care facilities (Ottawa Public Health, May 2020). At the time of writing (July 14, 2020), Ottawa's first wave had effectively ended, with a total of 2149 cases and 263 deaths, in a population of 1.4 million people. Sporadic cases peaked in mid to late March at 27 cases/day, but a much larger outbreak in long-term care facilities peaked at 93 cases/day on April 25 (Ottawa Public Health, June 2020).

While the model developed here was a snapshot in time, it is worth considering some further information about the disease and approaches to modeling. The assumption that symptomatic individuals had a higher transmission rate than asymptomatic individuals, while likely true for circulating individuals in the first part of 2020, would have been revised drastically after March 12. Once lockdowns began to be enforced and symptomatic individuals were requested or required to stay home or be hospitalized, their contacts would have declined significantly, offsetting their higher rate of viral shedding.

For a campus population consisting primarily of younger individuals, we matched the death rate due to COVID-19 to that of younger individuals. Correspondingly, the asymptomatic rate may also have been underestimated, since younger people are less affected by the disease. If the death rate is proportional to the symptomatic rate, this suggests the following ratio:

$$\frac{\text{population death rate}}{\text{population symptomatic rate}} = \frac{\text{campus death rate}}{\text{campus symptomatic rate}}$$

$$\frac{0.0628}{1-0.17} = \frac{0.002}{\text{campus symptomatic rate}}$$

From this relationship, we have $p = 0.9736$ and $\beta = 10.369 \times 10^{-6}$ in the case where $\alpha = 2$ and $\eta = 0.5$.

Figure 6 shows the results from Figure 2 (the worst-case scenario) using this revised asymptomatic rate. In this case, the number of exposed and asymptomatic individuals rises, while the number of symptomatic individuals is significantly lower, peaking at 300 instead of 6000. Although the first death is predicted at a similar time (127 days after the first symptomatic case, rather than 115 days), the total number of deaths is kept at 2. The other cases produce similar numbers (results not shown).

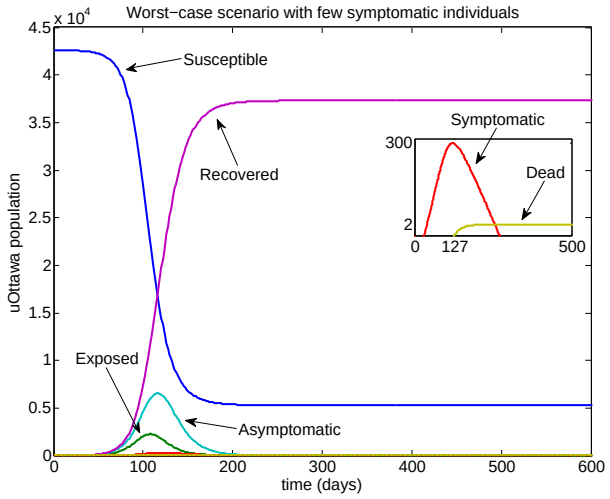

Figure 6: In the case where asymptomatic individuals on campus were matched proportionally to the campus death rate, the number of symptomatic individuals decreases significantly, and the number of exposed and asymptomatic individuals rises. In this case, we predict the first death occurring 127 days after the first symptomatic individual is diagnosed, eventually leading to 2 deaths.

7 DISCUSSION

We should stress that we are not recommending that lockdown remain in place for 13 years; it is up to political leaders to decide if 5, 25, 58 or 63 deaths on a university campus is acceptable. Furthermore, the actual contact reduction due to global lockdown was almost certainly more than 50% among university students, although this is likely to be highly heterogeneous (e.g., if students have jobs as essential workers).

The model has several limitations, which should be acknowledged. In addition to the "snapshot in time" it provides for March 12, 2020, and the concomitant limitations of available data, it should be noted that the model does not consider vital dynamics in the campus population; in particular, there is no influx of new susceptibles, and the population remains constant. In the former case, it is reasonable that the number of susceptibles will only decrease during a pandemic, but the university population may change significantly as a result of lockdown (e.g., fewer international students or an increase in distance learning via zoom). We also assumed mass-acton transmission, which assumes individuals are well-mixed; that assumption may not hold in lockdown, which not only reduces the number of contacts but also the strength of their interactions. Finally, recovered individuals were assumed to be immune to reinfection, which we now know is not true.

In summary, the benefits of this model were its rapid development, its flexibility in considering multiple cases where data was lacking and its ability to directly answer the questions posed by the university administration. We have thus given a snapshot of the role that models can play during a fast-moving pandemic, shown their power to make predictions and illustrated their ability to inform policy when linked to decision-making processes.

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AUTHOR BIOGRAPHY

ROBERT SMITH? (the question mark is part of his name) is a professor of disease modeling in the Department of Mathematics and the Faculty of Medicine at the University of Ottawa. He holds a Ph.D. in mathematics from McMaster University. His research interests lie in using differential equations to model intervention strategies and human behavior in infectious diseases. His email address is rsmith43@uottawa.ca.