



Project Brief

TRANSPORTATION AS A DISEASE VECTOR — A MODELING APPROACH

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Texas A&M Transportation Institute
June 2020

Executive Summary

Our interconnected world, linked by transportation networks, plays a major role in the spread of a pandemic such as COVID-19. The virus originated in a single community, but due to the global nature of transportation, it spread to other parts of the world, where it impacted local communities. And the cycle repeated, many times over. In the context of disease spread, transportation can be viewed as a *disease vector* because it can spread diseases through at least the following three mechanisms:

- Infected people and goods travel to other locations and can spread the disease when they reach their destinations and along the way.
- People congregate in groups and at higher densities when using public transportation, making it more likely that infected people can infect their fellow passengers.
- The surfaces in public transportation and shared vehicles can become infected through contact by infected people, potentially infecting others who touch the same surfaces.

It is therefore vitally important to clearly understand transportation's role in the spread of disease so that informed decisions can be made to stop or at least significantly reduce the spread of disease through transportation. In this project, we developed a demonstration model to show how transportation can function as a disease vector and to show how certain policies can be effective in reducing the spread of a disease such as COVID-19. The model addresses the spreading of disease through the third mechanism outlined above.

The demonstration model uses a stochastic agent-based approach that models infections due to local person-to-person and person-to-vehicle interactions. People are arranged on a square grid with periodic boundary conditions, and only nearest neighbors can come in contact with each other. We assume that any person can come in contact with any vehicle with equal probability. People find themselves in the following states: vulnerable, infected (with a variable time-dependent infectivity), recovered, or dead. Vehicles are characterized by their variable time-dependent infectivity. The model simulates person-to-person and person-to-vehicle interactions to model the spread of a disease such as COVID-19 through the synthetic population. The model parameters that can be changed in the modeling approach are the following:

- Person-to-person encounter rate.
- Person-to-vehicle encounter rate.
- Vehicle disinfection rate.
- Person per vehicle ratio.

These variables simulate policies such as social distancing, shelter in place, disinfection of public transportation vehicles, frequency of public transportation services, occupancy rates in public transportation vehicles, etc. The model shows that the key variables are all important in controlling the spread of a disease such as COVID-19 through transportation, and some combinations of the policies are more effective than others. The outputs of the model are shown graphically as well as through simulation videos. Simulation videos for each scenario may be accessed and viewed at [the following Dropbox location](#).

The demonstration model shows that transportation can be modeled as a disease vector and that key policies such as social distancing, shelter in place, disinfection of public transportation vehicles, and limitations on the number of people per vehicle are all very effective in fighting the spread of disease through transportation. The demonstration platform has the potential to be adapted to a transit network, city, metropolitan planning organization, or region. Further, the platform has the potential to more fully inform decision makers as they develop strategies to effectively combat the spread of disease through the transportation system.

For Further Information

This project brief represents work in progress, with funding from the Texas A&M Transportation Institute's Center for Advancing Research in Transportation Emissions, Energy, and Health, a U.S. Department of Transportation University Transportation Center. The grant number is 69A3551747128.

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Introduction

Transportation plays a major role in the global spread of disease. Transportation may affect the spread of epidemics in several ways. First, transportation increases the range of movement and the spatial diversity of the infected and exposed individuals by facilitating encounters between people who do not live near each other. Second, public transportation increases the transmission rate by forcing people into prolonged contact in a confined, close environment. And third, public transportation vehicles and infrastructure can become carriers of disease that facilitate the indirect transmission of pathogens. In this study, we extend traditional epidemiological models by specifically addressing the indirect disease transmission mechanism. Infectious people deposit pathogens onto inanimate objects where the virus can remain viable and active for hours and even days. Infected objects (also referred to as *fomites*) in transportation vehicles and infrastructure, such as seat belts, ticketing booths, armrests, etc., are examples of fomites. We explore the effect of indirect transmission, that is, the infections that occur when vulnerable people become infected through contact with fomites.

COVID-19 and previous pandemics have shown us that we must take every possible precaution to minimize disease transmission, and this includes addressing transportation's role in the spread of disease. The role of transportation in the spread of disease, specifically in vehicles, has been discussed to some extent across the literature. Transportation has historically played a role in the spread of diseases, including influenza (Browne et al., 2016), severe acute respiratory syndrome (SARS) (Bowen and Laroe, 2006), and tuberculosis (Edelson and Phipers, 2011). Studies have also focused on the enhancement of person-to-person transmission in close quarters (Harris, 2020; Kucharski et al., 2020; Li et al., 2020; Riou and Althaus, 2020) and indirect transmission via fomites (Zhao et al., 2019). However, there are still gaps in understanding the impact of transportation vehicles and infrastructure across all transport modes as an additional means of disease transmission. To our knowledge, there are no models or simulation tools that attempt to quantify the role of transportation vehicles and infrastructure as disease vectors.

Highlights

- Transportation can affect the spread of disease in several ways:
 - Increases the range and diversity of infected/exposed individuals.
 - Increases transmission rate by forcing people into prolonged contact in a confined, close environment.
 - Allows indirect transmission of pathogens through surfaces in the vehicles.
- Transportation can act as a disease vector.
- We showcase a simple demonstration model of how disease can spread due to transportation with respect to person-to-person contact rates, person-to-vehicle contact rates, disinfection rates, and number of passengers per vehicle.
- Lower person-to-person contact, person-to-vehicle contact, and passengers per trip as well as higher disinfection rates are associated with lower risk of disease transmission.

Model and Simulation Approach

We developed a demonstration model using a stochastic agent-based approach that models infections due to person-to-person contacts (as in other epidemiological models), while additionally modeling infections caused indirectly (i.e., from vehicles to people). The model includes two types of agents: people and vehicles (representing any form of public transportation). Notably, the model addresses near-range contacts between people, but it does not address long-range contacts between people or person-to-person contacts inside public transportation vehicles. People are arranged on a square grid with periodic boundary conditions, and only nearest neighbors (spatially) can come in contact with each other. We assume that any person can come in contact with any vehicle with equal probability. Each person is characterized quantitatively by the vulnerability to infection (vulnerable or affected), the onset time of infection, the time-dependent level of infectivity, and the time-dependent level of activity. Vulnerable people can become infected as a result of contact with an infected person or a vehicle with infectivity I . The probability of infection in such an encounter is assumed to be $P = 1 - e^{-\lambda I}$, where the parameter λ is estimated to be 0.018 based on the number of daily contacts and the reproduction number R_0 reported in Tang et al. (2020).

The infectivity dynamics of people and vehicles are different. We assume that the infectivity of a person (shown in Figure 1a) rises linearly over a time period τ_1 , which is estimated to be 3 days (Guan et al., 2020), to a maximum of 1 and then falls linearly to 0 at time τ_2 after the onset of infection. The mean recovery time $\tau_2 - \tau_1$ is estimated to be 15 days (Aylward and Liang, 2020). The activity factor determines the rate at which the infected person comes into contact with vehicles and other people. Its dynamics mirror that of infectivity (shown in Figure 1b). Healthy people are characterized by the activity factor of 1. After the onset of infection, the activity factor decays linearly with time and reaches the minimum dictated by the severity of the particular disease instance. The severity of each infection is assumed to be random and uniformly distributed in (0,1). Vehicles are characterized quantitatively by their level of infectivity. The infectivity decays exponentially with the characteristic time scale τ_3 , which we estimate to be roughly 0.4 days based on the study of the surface stability of SARS-CoV-2 (van Doremalen et al., 2020).

Model Overview

- Two types of agents: people and vehicles.
- Each agent is characterized by a time-dependent infectivity.
- Four types of events:
 - Person-to-person encounters.
 - Person-to-vehicle encounters.
 - Vehicle disinfections.
 - Deaths.
- People are arranged on a square grid, and only nearest neighbors can interact.
- Any person is equally likely to encounter any vehicle.
- People can be infected by other people and by vehicles.
- Vehicles are infected by people.
- The model does not address long-range contacts between people or person-to-person contacts inside a public transportation vehicle.

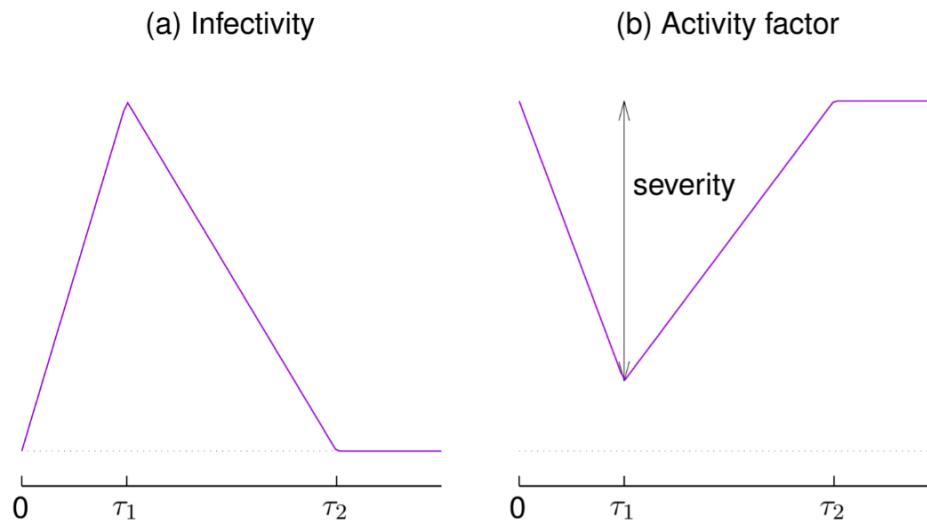


Figure 1: The infectivity (a) and the activity (b) dynamics of a person. Time 0 corresponds to the time at which a particular person became infected.

We employed the simulation methodology (Gillespie, 1976, 1977) that proceeds via stochastic sequential processing for four types of events:

- Person-to-person contacts.
- Deaths.
- Person-to-vehicle contacts.
- Vehicle disinfections.

The rate of person-to-person contacts is the total human activity A , the sum of all current activity factors scaled by the person-to-person contact rate C . When a person-to-person contact event is triggered, a random person and their nearest grid neighbor are selected with the probabilities proportional to their current activity factors. These two people come in contact. When the contact involves an infected and a vulnerable person, the vulnerable person becomes infected with the probability P introduced above. Death occurs at a rate proportional to the product of the difference of the activity factor from 1 and the death rate M , which is fixed at 0.05 to approximate the observed fraction of fatal outcomes for COVID-19. When persons are marked dead, they are excluded from further event processing.

The rate of person-to-vehicle contacts is the product of the total human activity A and the person-to-vehicle contact rate V . In this context, “vehicles” refer to public spaces (including public transportation vehicles and infrastructure), rather than personal vehicles. The person-to-vehicle contact rate does not vary with a specific person’s access to personal transportation. When a vulnerable individual comes in contact with a vehicle, the person becomes infected with probability P given above, which depends on the current vehicle infectivity. When an infected person comes into contact with a vehicle, the infectivity of that vehicle is incremented by the current infectivity of the infected person. The disinfection rate is the product of the number of vehicles N_v and the disinfection rate D . When a disinfection event is triggered, a random vehicle’s infectivity is set to 0.

The simulation proceeds until the total infectivity of all people and vehicles is 0. At the end of the simulation, we measure the fraction of the population infected, the death fraction, and the total duration of the epidemic.

Table 1 summarizes all model parameters. The parameters that can be influenced by policy include the person-to-person contact rate C , the person-to-vehicle contact rate V , the number of vehicles N_v , and the vehicle disinfection rate D . For example, decision makers may employ social distancing and/or shelter-in-place orders to limit person-to-person contact and person-to-vehicle contact. Decision makers may also regulate the number of public transportation vehicles available and/or how often vehicles are disinfected.

Table 1: Model parameters.

Parameter	Description
N_p	Number of people
N_v	Number of vehicles
τ_1	Duration from a person's infection onset to peak infectivity
τ_2	Duration from a person's infection onset to recovery
τ_3	Characteristic time scale for the exponential decay of vehicle infectivity
D	Number of disinfections per day per vehicle
V	Person-to-vehicle contacts per day per person
C	Person-to-person contacts per day per person
M	Death rate
λ	Determines the likelihood of infection during an encounter

Key Findings

The simulation starts with a single infected individual, while every other person is vulnerable. Due to the stochastic nature of transmission, there is a non-zero probability that just a few other individuals are infected before the disease runs its course. However, if the number of infected individuals passes a certain threshold, the epidemic spreads until a significant fraction of the population has been affected. Therefore, the distribution of the ultimate infected population fraction is bimodal, as shown in Figure 2. The values of the model parameters are specified in the model description except for the person-to-person contact rate C , person-to-vehicle contact rate V , vehicle disinfection rate D , and person per vehicle ratio $R = N_p/N_v$. We verified that only this ratio is relevant because the results are unchanged if both the numbers of vehicles N_v and people N_p are multiplied by the same factor.

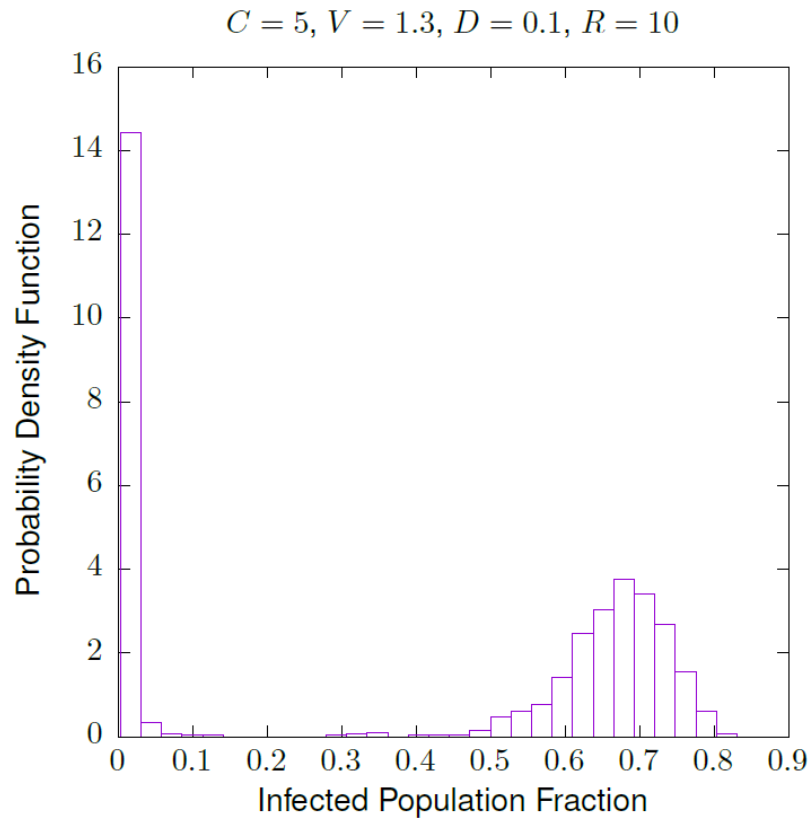


Figure 2: The distribution of the epidemic size (infected population fraction) is bimodal in general.

We quantify the epidemic by computing the probability that a significant fraction (defined as 5 percent) of the population will become infected before the epidemic runs its course. Figure 3 shows the dependence of this probability on the person-to-person contact rate C (a), person per vehicle ratio R (b), vehicle disinfection rate D (c), and person-to-vehicle contact rate V (d). The growth of the significant epidemic probability with the person-to-person contact rate C is self-evident. The qualitative trends in Figure 3b, 3c, and 3d can be understood in terms of the vehicle-to-person transmission. Vehicles serve as reservoirs of the pathogen that spread the infection to unaffected parts of the population because every person can come in contact with any vehicle with equal probability. The average vehicle infectivity declines with increasing the disinfection rate, decreasing the number of people per vehicle, and decreasing the person-to-vehicle contact rate, which leads to the behavior shown in Figure 3. A further quantification of the epidemic's severity is the expected population fraction that will be infected during a substantial epidemic. The results presented in Figure 4 exhibit the same qualitative trends and can be explained via the same mechanisms as the probability of a substantial epidemic shown in Figure 3.

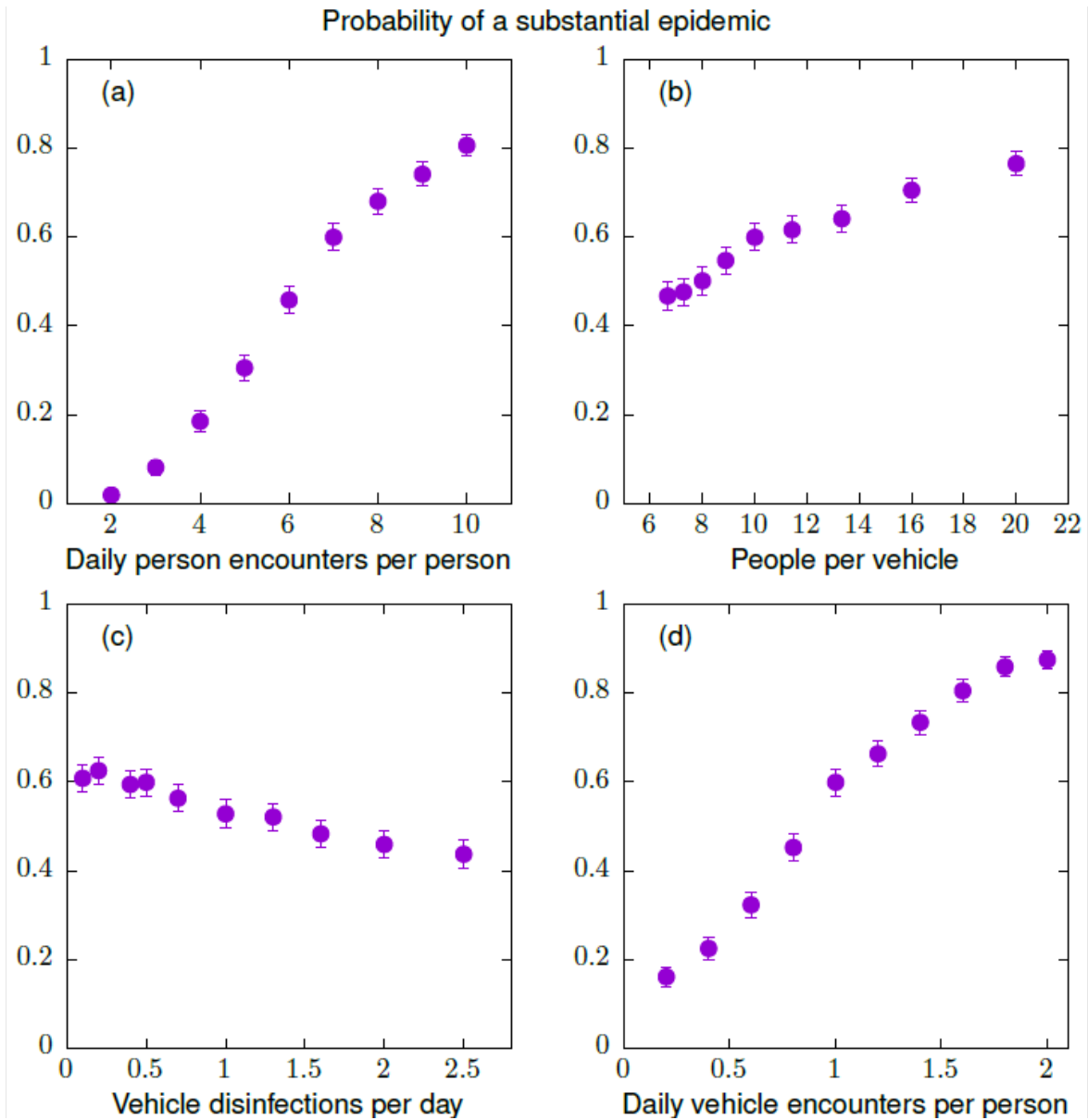


Figure 3: The probability of a substantial epidemic (in which more than 5 percent of the population is affected) as a function of the person-to-person contact rate C (a), ratio R of people per vehicle (b), vehicle disinfection rate D (c), and person-to-vehicle contact rate V (d). The values of the parameters that are not varied in each panel are $C = 7$, $V = 1$, $D = 0.5$, and $R = 10$.

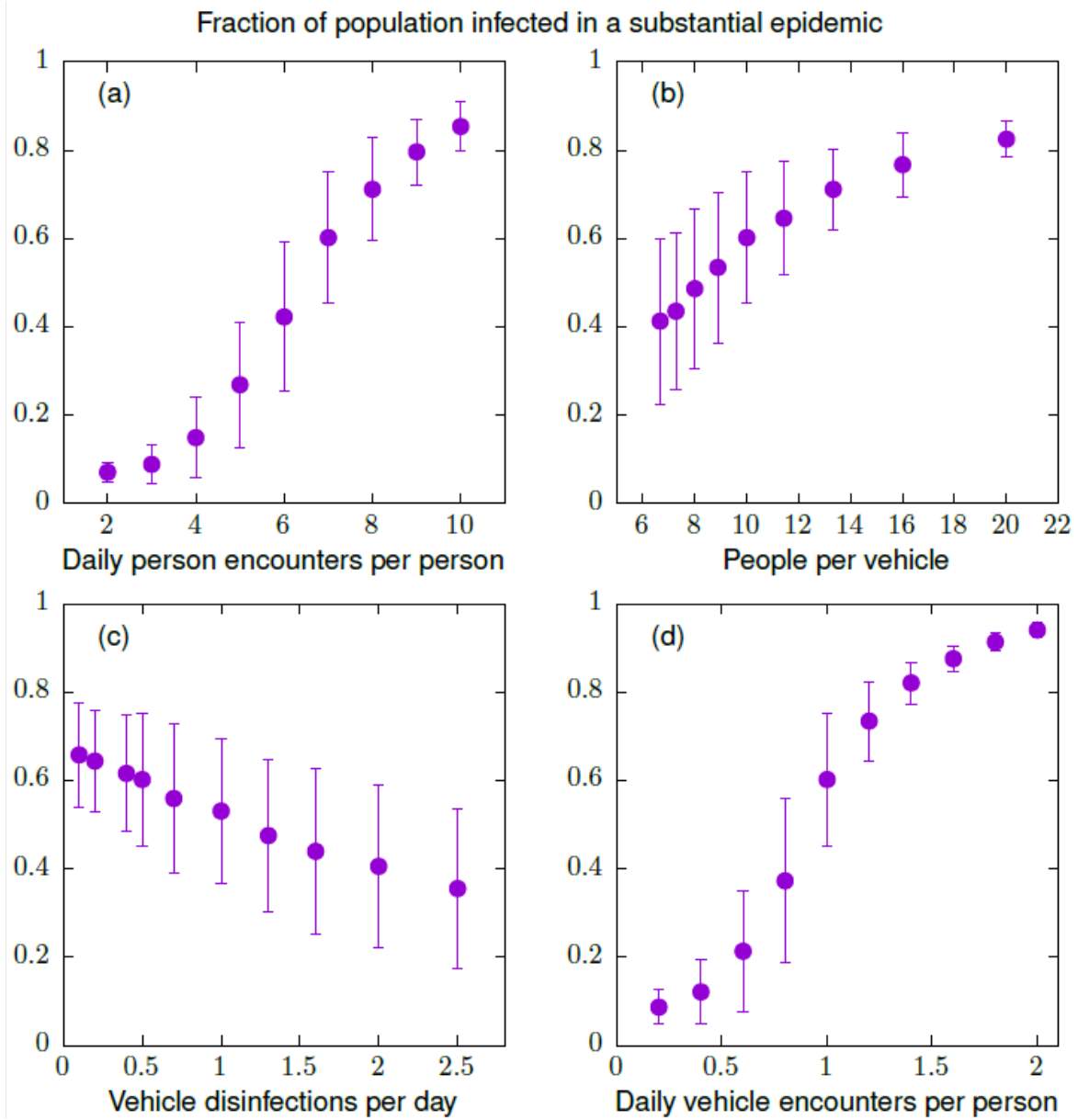


Figure 4: The average population fraction that becomes infected in a substantial epidemic. The error bars denote one standard deviation from the mean epidemic size.

Next, we present the spread of disease under different conditions graphically. Table 2 displays a link to the video of each simulation scenario. In the videos, circles represent people, and rectangles represent vehicles. The color code is explained in Figure 5. The parameters that are varied include the person-to-person contact rate C , person-to-vehicle contact rate V , number of people per vehicle R , and vehicle disinfection rate D . In the worst-case scenario, the virus quickly infects everyone, and the vehicles are highly infectious at the height of the epidemic. The rest of the videos show what happens when one or two of the variables are changed to try to mitigate the worst case. Decreasing the person-to-person contact rate to 1 person-to-person contact per day per person ($C = 1$) works (e.g., social distancing). Decreasing the person-to-vehicle contact rate to 1 person-to-vehicle contact every 10 days per person ($V = 0.1$) works (e.g., working from home). Increasing the vehicle disinfection rate to 5 disinfections per day per vehicle ($D = 5$) or decreasing the number of people per vehicle to 4 ($R = 4$) do not work by themselves, but doing both at the same time has a noticeable effect.

Table 2: Videos depicting the epidemic graphically.

Sample Video Simulations	Parameters	Outcome
Video: Worst-case scenario	10 person-to-person contacts per day per person ($C = 10$) 1 person-to-vehicle contact per day per person ($V = 1$) 1 disinfection every 10 days per vehicle ($D = 0.1$) 20 people per vehicle ($R = 20$)	<u>Timeline:</u> 156 days <u>Population:</u> 92.8% infected 88.8% recovered 4% died
Video: Decrease person-to-person contact rate (C)	1 person-to-person contact per day per person ($C = 1$) 1 person-to-vehicle contact per day per person ($V = 1$) 1 disinfection every 10 days per vehicle ($D = 0.1$) 20 people per vehicle ($R = 20$)	<u>Timeline:</u> 228 days <u>Population:</u> 8.3% infected 7.9% recovered 0.4% died
Video: Decrease person-to-vehicle contact rate (V)	10 person-to-person contacts per day per person ($C = 10$) 1 person-to-vehicle contact every 10 days per person ($V = 0.1$) 1 disinfection every 10 days per vehicle ($D = 0.1$) 20 people per vehicle ($R = 20$)	<u>Timeline:</u> 285 days <u>Population:</u> 11.4% infected 11% recovered 0.4% died
Video: Increase vehicle disinfection rate (D)	10 person-to-person contacts per day per person ($C = 10$) 1 person-to-vehicle contact per day per person ($V = 1$) 5 disinfections per day per vehicle ($D = 5$) 20 people per vehicle ($R = 20$)	<u>Timeline:</u> 212 days <u>Population:</u> 81.9% infected 78.3% recovered 3.6% died
Video: Decrease number of people per vehicle (R)	10 person-to-person contacts per day per person ($C = 10$) 1 person-to-vehicle contact per day per person ($V = 1$) 1 disinfection every 10 days per vehicle ($D = 0.1$) 4 people per vehicle ($R = 4$)	<u>Timeline:</u> 393 days <u>Population:</u> 76.9% infected 73.6% recovered 3.3% died
Video: Increase vehicle disinfection rate (D) and decrease number of people per vehicle (R)	10 person-to-person contacts per day per person ($C = 10$) 1 person-to-vehicle contact per day per person ($V = 1$) 5 disinfections per day per vehicle ($D = 5$) 4 people per vehicle ($R = 4$)	<u>Timeline:</u> 377 days <u>Population:</u> 50.9% infected 48.3% recovered 2.6% died

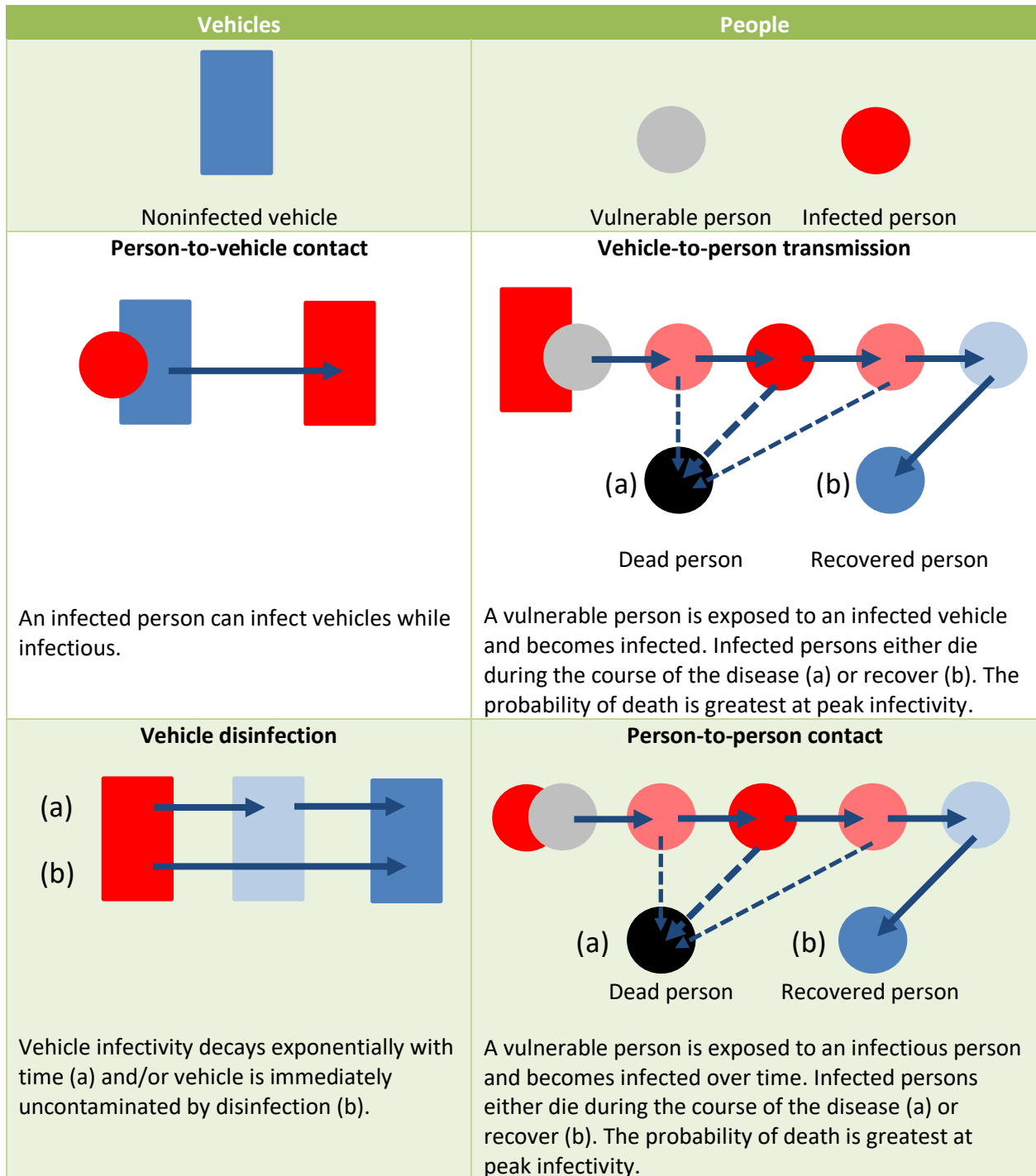


Figure 5: Explanation of the graphical representation elements and the color code. Infectivity increases from deep blue (0) to light blue to light red to deep red (>1).

Conclusions

In this project, we make a case for transportation vehicles and infrastructure as disease vectors. We present a demonstration model that shows the impact of the person-to-person contact rate, person-to-vehicle contact rate, disinfection rate, and number of people per vehicle on the spread of disease. Reducing the person-to-person contact rate or the person-to-vehicle contact rate effectively limits disease spread. Increasing the vehicle disinfection rate or decreasing the number of people per vehicle does not work alone, but doing both simultaneously can have a substantial effect on the epidemic's severity. Simplifying assumptions notwithstanding, the demonstration model shows that transportation can be modeled as a disease vector and that the person-to-person contact rate, person-to-vehicle contact rate, disinfection rate, and number of people per vehicle are important factors to consider when implementing disease control strategies in different transportation modes. Key strategies such as social distancing, shelter in place, disinfection of public transportation vehicles, and limitations on the number of people per vehicle are all very important in fighting the spread of disease through transportation.

Policy and decision makers, scientific researchers, and practitioners are encouraged to use the concepts presented in this project brief to further explore the role of transportation vehicles and infrastructure as disease vectors and to investigate strategies to limit disease spread in this capacity. Another area for further investigation includes implementing material with antimicrobial properties (e.g., copper [Grass et al., 2011]) on frequently touched transportation surfaces and personal protective equipment. The demonstration platform presented here has the potential to be adapted to a transit network, city, metropolitan planning organization, or region. There is potential for practical applications if the demonstration model is, for example, calibrated with local data such as the structure of contact networks and the patterns of usage of shared spaces in public transportation systems. The platform has the potential to more fully inform decision makers as they develop strategies to effectively combat the spread of disease through the transportation system.

The findings presented in this project brief pave the way for future research in the area of transportation as a disease vector with the goal of mitigating disease spread through transportation. Interdisciplinary work is warranted to reexamine how best to harden transportation assets to minimize the capacity to transmit disease. Transportation engineers, epidemiologists, infectious disease experts, biochemists, materials scientists, and others will need to collaborate to reimagine transportation aspects such as materials of construction and surface preparation. As the world continues to become more interconnected, it is important to continue understanding the impact transportation has on the spread of disease so that informed decisions can be made to stop or at least significantly reduce the spread of disease through transportation.

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