DYNAMICAL ANALYSIS IN DISEASE TRANSMISSION AND FINAL EPIDEMIC SIZE

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Abstract. We propose two compartment models to study the disease transmission dynamics, then apply the models to the current COVID-19 pandemic and to explore the potential impact of the interventions, and try to provide insights into the future health care demand. Starting with an SEAIQR model by combining the effect from exposure, asymptomatic and quarantine, then extending the model to the one with ages below and above 65 years old, and classify the infectious individuals according to their severity. We focus our analysis on each model with and without vital dynamics. In the models with vital dynamics, we study the dynamical properties including the global stability of the disease free equilibrium and the existence of endemic equilibrium, with respect to the basic reproduction number. Whereas in the models without vital dynamics, we address the final epidemic size rigorously, which is one of the common but difficult questions regarding an epidemic. Finally, we apply our models to estimate the basic reproduction number and the final epidemic size of disease by using the data of COVID-19 confirmed cases in Canada and Newfoundland & Labrador province.

1. Introduction. Mathematical modelling is a powerful tool for understanding disease transmission and exploring different scenarios. To study the infectious disease epidemiology and the dynamics of disease transmission, mathematical models based on the principles of viral transmission can play a key role in providing evidence-based information to health policymakers. Applications include determining optimal control strategies against new or emergent infections, such as SARS-CoV-2, Zika, Ebola, against HIV, tuberculosis and malaria [16, 4, 15, 23, 13, 14].

Since the identification of the novel coronavirus (which is named COVID-19) in December 2019, we all know that the virus has been sweeping the globe [12, 32, 21, 20, 2]. The disease, which can trigger severe respiratory symptoms, has been reported on every continent except Antarctica and in at least 180 countries. On March 11, 2020, the World Health Organization (WHO) assessed COVID-19 as a
pandemic. The data surrounding the biology, epidemiology, and clinical characteristics of the COVID-19 virus have been growing daily. Up to June 20, 2020, there have been more than 8.8 million confirmed cases, including 465K deaths globally. At present, many researchers have made rapid responses to the current epidemic [25, 26, 30] and established mathematical models about the dynamics and transmission of COVID-19. It is well known that the basic reproduction number, which is defined by the expected number of new infective individuals infected by a typical infective individual during its entire period of infectiousness in a fully susceptible population, is an important index to measure the transmission potential of a disease. The use of mathematical models to estimate the basic reproduction number of COVID-19, ranging from 1.4 – 6.49, gives a sharp threshold property for their global dynamics [10, 24], helps to determine the potential and severity of outbreaks [28, 22].

There has been growing recognition that, different from SARS, MERS coronavirus [31, 9], there is a long incubation period of the new coronavirus (14 days on average), which reflects the importance of quarantine. Quarantine/isolation (we mix them for simplicity in this paper) is one of the commonest methods of controlling the spread of diseases. In the light of the experience of China, quarantine is considered as one of the most effective ways to prevent people from getting infected by the virus. In previous pandemics such as SARS, the number of asymptomatic or mildly symptomatic people after infection was quite low, making it easier to contact trace the infected cases, and isolate them. We do not yet have enough information as to whether this is the case with COVID-19, although a large number of facts indicate that one of the characteristics of COVID-19 is the presence of an asymptomatic infected population.

One of the common questions regarding an epidemic is its final size [1, 3, 5, 6]. The final size of an epidemic can be defined informally as the total number of people experiencing infection [11] throughout the outbreak. The probability distribution of final sizes is of particular interest to statistical epidemiologists. For a deterministic epidemic in a closed, homogeneous population, the final size equation gives the number (or frequency) of susceptible hosts at the end of the epidemic. The result in [18] shows that in most of such models, the final size relation involves only a single parameter - the basic reproduction number. With interventions such as quarantine and reduction of contact are included in the compartmental model, does this claim still hold?

There is limited information regarding the new coronavirus risk factors for severe disease. From the official site of the Centers for Disease Control and Prevention (CDC), based on currently available information and clinical expertise, we know that older adults (with age above 65) and people of any age who have serious underlying medical conditions might be at higher risk for severe illness. The majority of patients (accounting for 80% of the USA and Canada deaths from the disease) dying with COVID-19 are elderly and the large majority of the deceased may have severe underlying diseases. As the epidemic has the potential to collapse the health care system, it is essential for the effective allocation of emergency resources in areas with high levels of infection.

Given the above considerations, to improve understanding of the complex dynamics in the emerging outbreak of COVID-19, we propose two different compartmental models to study the transmission dynamics of the virus and to explore the potential impact of the quarantine interventions in the population of Canada and
Newfoundland & Labrador province within Canada, and to disentangle how transmission is affected in different age group. First, we establish a one-group SEAIQR model to investigate the dynamics of the disease transmission by combining the effect from exposure, asymptomatic, and quarantine classes. Since COVID-19 poses a significant threat to anyone (especially for older people) unfortunate enough to be hospitalized with it, and critical cases can be admitted to the intensive care unit and possibly hooked up to ventilators, we further extend and modify the one-group model to two-groups with ages below and above 65 years old, and classify the infectious individuals to mild symptoms who needs self-isolation, patient in hospital and patient in intensive care, try to provide some insights into infectious disease trends in different age group. We focus our analysis on each model with and without vital dynamics. As we know that, in a population with vital dynamics, new births can provide more susceptible individuals to the population, sustaining an epidemic or allowing new introductions to spread throughout the population. Without the implementation of strict containment measures and movement restrictions, the disease dynamics will reach a steady state. In this category, we work on the dynamical properties including the global stability of the disease-free equilibrium and the existence of endemic equilibrium, with respect to the basic reproduction number. On the other side, if the virus transmission over time is much faster than the natural birth and death rates in the population, vital dynamics (birth and death) can be ignored. Because of the rapid spreading of the COVID-19 virus, in the models without vital dynamics, we explore the final epidemic size which is one of the common but difficult questions regarding an epidemic. Finally, using the data of COVID-19 confirmed cases in Canada and Newfoundland & Labrador province, we parameterize the model to estimate the basic reproduction number and the final epidemic size of disease.

This paper is structured as follows. After the introduction, in Section 2, we propose a one-group SEAIQR model, study the dynamical properties involved in the model with vital dynamics, give the relation of the basic reproduction number to the system parameters, explore the final epidemic size when ignoring the natural birth and death rates and provide theoretical computation for the final size. With the concern of high risk in elder people, in Section 3, we modify the previous one-group model to two-groups by separating the population with age below or above 65 years, together with the consideration of the severity of infectious individuals. Parallel dynamical analysis and final epidemic size computation are carried. In Section 4, we implement the numerical simulations to supplement the global dynamics beyond the theoretical analysis and compute the final epidemic size for one- and two-group models by using the real data in Canada and Newfoundland & Labrador province. Conclusion and discussion are drawn in Section 5, where the recovery rate incorporating social healthy system and medical resources is addressed.

2. Dynamics in SEAIQR model.

2.1. Model description. We propose a deterministic “Susceptible-Exposed-Infectious without or with symptoms-Quarantined-Recovered” (SEAIQR) compartmental model based on the clinical progress and interventions of the disease, the flow diagram of the disease transmission is shown in Figure 1.

Here, we divide the total population ($N$) into six classes: susceptible ($S$), exposed ($E$), infected but not yet symptomatic ($A$), infectious with symptoms ($I$), quarantined ($Q$), and recovered ($R$), that is, $N = S + E + A + I + R + Q$. We
assume that, after exposure to the virus, the individuals will be quarantined in each class. With vital dynamics, the disease transmission is governed by the following nonlinear system of differential equations:

\[ \begin{align*}
\dot{S}(t) &= \Pi - \lambda(t)S(t) - \mu S(t), \\
\dot{E}(t) &= \lambda(t)S(t) - (q_1 + \alpha_1 + \alpha_2 + \mu)E(t), \\
\dot{A}(t) &= \alpha_1 E(t) - (q_2 + \sigma + \gamma_1 + \mu + \theta_1)A(t), \\
\dot{I}(t) &= \alpha_2 E + \sigma A(t) - (\gamma_2 + q_3 + \mu + \theta_2)I(t), \\
\dot{Q}(t) &= q_1 E(t) + q_2 A(t) + q_3 I(t) - (\gamma_3 + \mu)Q(t), \\
\dot{R}(t) &= \gamma_1 A(t) + \gamma_2 I(t) + \gamma_3 Q(t) - \mu R(t),
\end{align*} \]

where \( \lambda(t) \) is quarantine-adjusted infection rate or the force of infection:

\[ \lambda(t) = \frac{\beta_1 A(t) + \beta_2 I(t) + \beta_3 Q(t)}{S(t) + E(t) + A(t) + I(t) + R(t) + Q(t)}. \]

Here we take the term \( \beta_3 Q(t) \) into account in the infection rate, since there exists the household infection in reality. For instance, it is fairly serious at the early stage of the outbreak in Wuhan China. In addition, due to the rapid spread of COVID-19, medical resources have been extremely saturated, many patients with mild or no symptoms are put at home to isolate which inevitably may cause family infection. This situation was particularly serious in The United States at the beginning of the outbreak. Many mild patients were not hospitalized, but isolated at home, which happens to infect the family members, since the infection chain is not interrupted. The description of the parameters and their value ranges are provided in the following table (See Table 1).

2.2. Dynamical analysis. First, we show the model (2.1) is well-defined.

**Lemma 2.1.** (i) All the solutions of the model (2.1) with positive initial data will remain positive for any time \( t > 0 \);

(ii) The closed set \( \mathcal{D} = \{(S, E, A, I, R, Q) \in \mathbb{R}_+^6 : N = S + E + A + I + Q + R \leq \frac{\Pi}{\mu} \} \) is positive invariant.

**Proof.** (i) The positivity of the solution is obvious from [27].
Table 1. Parameter Description and Estimation

| Parameter | Description                                      | Values (per day) | Source |
|-----------|--------------------------------------------------|------------------|--------|
| $\Pi$     | Recruitment rate                                 | 136              | [22]   |
| $\mu$     | Natural death rate                               | 0.0078           | United Nations |
| $\beta_1$ | Effective contact rate with $A$                  | 0.491            | [9]    |
| $\beta_2$ | Effective contact rate with $I$                  | 0.391            | assumed |
| $\beta_3$ | Effective contact rate with $Q$                  | 0.291            | assumed |
| $\alpha_1$| Progression rate from $E$ to $A$                 | 0.01880857       | [25]   |
| $\alpha_2$| Progression rate from $E$ to $I$                 | 0.156986         | [22]   |
| $\sigma$  | Symptom progression rate ($A$ to $I$)            | 0.001            | assumed |
| $q_1$     | Quarantine rate for $E$                          | 0.1              | [22]   |
| $q_2$     | Quarantine rate for $A$                          | 0.11             | assumed |
| $q_3$     | Quarantine rate for $I$                          | 0.12             | assumed |
| $\gamma_1$| Recovery rate for $A$                           | 0.13978          | [25]   |
| $\gamma_2$| Recovery rate for $I$                           | 0.03521          | [22]   |
| $\gamma_3$| Recovery rate for $Q$                           | 0.042553         | [22]   |
| $\theta_1$| Disease-induced death rate for $A$               | 0.001            | assumed |
| $\theta_2$| Disease-induced death rate for $I$               | 0.04227          | [22]   |

(ii) From (2.1), we have

$$\frac{dN}{dt} = \Pi - \mu N - \theta_1 A - \theta_2 I.$$ 

Since $\frac{dN}{dt} \leq \Pi - \mu N$, it follows that $\frac{dN}{dt} \leq 0$ if $N \geq \frac{\Pi}{\mu}$. Thus, by comparison theorem, $N \leq N(0)e^{-\mu t} + \frac{\Pi}{\mu}(1 - e^{-\mu t})$. In particular, $N(t) \leq \frac{\Pi}{\mu}$ if $N(0) \leq \frac{\Pi}{\mu}$. Thus, the region $D$ is positive invariant which attracts all solutions in $\mathbb{R}_+^6$. □

We know that the disease-free equilibrium point (DFE) $M^0 = (\frac{\Pi}{\mu}, 0, 0, 0, 0, 0)$ always exists in (2.1).

In (2.1), we denote

$$m_1 = q_1 + \alpha_1 + \alpha_2 + \mu, \quad m_2 = q_2 + \sigma + \gamma_1 + \mu + \theta_1, \quad m_3 = \gamma_2 + q_3 + \mu + \theta_2, \quad m_4 = \gamma_3 + \mu.$$  \hspace{1cm}(2.2)

Applying the formula in [28], we can calculate the basic reproduction number by $R_0 = \rho(FV^{-1})$, where

$$F = \begin{bmatrix} 0 & \beta_1 & \beta_2 & \beta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} m_1 & 0 & 0 & 0 \\ -\alpha_1 & m_2 & 0 & 0 \\ -\alpha_2 & -\sigma & m_3 & 0 \\ -q_1 & -q_2 & -q_3 & m_4 \end{bmatrix}.$$ 

Thus

$$R_0 = \rho(FV^{-1}) = \beta_1 \frac{\alpha_1}{m_1 m_4^2} + \beta_2 \frac{\sigma \alpha_1 + m_2 \alpha_2}{m_1 m_2 m_3 m_4} + \beta_3 \frac{q_3 (\sigma \alpha_1 + m_2 \alpha_2) + m_3 (q_2 \alpha_1 + m_2 q_1)}{m_1 m_2 m_3 m_4}$$

$$= \beta_1 \frac{\alpha_1}{m_1 m_4^2} + \beta_2 \frac{\sigma \alpha_1 + m_2 \alpha_2}{m_1 m_2 m_3 m_4} + \beta_3 \frac{q_3 (\sigma \alpha_1 + m_2 \alpha_2) + m_3 (q_2 \alpha_1 + m_2 q_1)}{m_1 m_2 m_3 m_4}$$

$$= R_A + R_I + R_Q.$$  \hspace{1cm}(2.3)
The basic reproduction number obtained in (2.3) breaks down into three components: secondary infections generated from the infectious people without symptom, with symptom, and from the quarantined individuals respectively.

Taking the basic reproduction number \( R_0 \) as a threshold index, we can obtain the global stability of the DFE \( M^0 \).

**Theorem 2.2.** The disease-free equilibrium (DFE) of the model (2.1), given by \( M^0 = (\frac{1}{\mu},0,0,0,0) \) is globally asymptotically stable (GAS) in \( \mathcal{D} \) whenever \( R_0 < 1 \).

**Proof.** Consider the Lyapunov function: \( V_1(t) = aE(t) + bA(t) + cI(t) + Q(t) \) with the constant coefficients \( a, b, c \):

\[
V_1 = \frac{1}{m_1} \left[ \left( \frac{\beta_1 m_3 m_4 + \beta_3 m_2 m_3 \beta_3 (q_2 m_3 + q_3 \sigma)}{\beta m_2 m_3} \right) \alpha_1 + \frac{(\beta_2 m_4 + \beta_3 q_3) \alpha_2 + q_1}{\beta m_3} \right],
\]

\[
b = \frac{\beta_1 m_3 m_4 + \beta_3 m_2 m_3 + \beta_3 (q_2 m_3 + q_3 \sigma)}{\beta m_2 m_3}, \quad c = \frac{\beta_2 m_4 + \beta_3 q_3}{\beta m_3},
\]

where \( m_i \) \( (i = 1, 2, 3, 4) \) is given in (2.2). Then the derivative of the function \( V_1(t) \) along the solution of (2.1) is

\[
\dot{V}_1 = a \left( \frac{\beta_1 A + \beta_2 I + \beta_3 Q}{S + E + A + I + R + Q} S - m_1 E \right) + b \left( \alpha_1 E - m_2 A \right) \]

\[
+ c \left( \alpha_2 E + \alpha_{2} Q \right) A + (q_1 E + q_2 A + q_3 I - m_4 Q),
\]

\[
= a \left( \frac{\beta_1 A + \beta_2 I + \beta_3 Q}{S + E + A + I + R + Q} S - \frac{m_4 \beta_2 I - m_4 \beta_1 A - m_4 Q}{\beta_3} \right)
\]

\[
\leq a \left( \beta_1 A + \beta_2 I + \beta_3 Q \right) - \frac{m_4 \beta_2 I - m_4 \beta_1 A - m_4 Q}{\beta_3}
\]

\[
= \left( a - \frac{m_4}{\beta_3} \right) (\beta_1 A + \beta_2 I + \beta_3 Q) = \frac{m_4}{\beta_3} (R_0 - 1) (\beta_1 A + \beta_2 I + \beta_3 Q).
\]

Thus, \( \dot{V}_1 \leq 0 \) for \( R_0 < 1 \) with \( \dot{V}_1 = 0 \) if and only if \( I = A = Q = 0 \). So \( V_1 \) is a Lyapunov function on \( \mathcal{D} \). Therefore, if \( R_0 < 1 \), \( V_1(t) \to 0 \) as \( t \to \infty \). That is, \((E, A, I, Q) \to (0, 0, 0, 0) \) as \( t \to \infty \).

Thus, for sufficient small \( \epsilon > 0 \), there exists \( T_1, T_2, T_3, T_4 > 0 \), when \( t > \max \{T_1, T_2, T_3, T_4\} \), \( A \leq \epsilon, I \leq \epsilon, Q \leq \epsilon \) and \( R \leq (\gamma_1 + \gamma_2 + \gamma_3) \epsilon - \mu R \). Consequently \( R(t) \leq \frac{\gamma_1 + \gamma_2 + \gamma_3}{\mu} \epsilon \to 0 \) as \( t \to \infty \). From the positivity of the system, we know \( R(t) \geq 0 \). Thus, \( R(t) \to 0 \) as \( t \to \infty \). Similarly, it can be shown that \( S(t) \to \frac{\Pi}{\mu} \) as \( t \to \infty \).

Therefore, the DFE \( M^0 \) is globally asymptotically stable in \( \mathcal{D} \) when \( R_0 < 1 \). \( \square \)

What dynamical behaviour involves in the system when \( R_0 > 1 \)? For the existence of endemic equilibrium point, we have the following result.

**Lemma 2.3.** Whenever \( R_0 > 1 \) there is unique endemic equilibrium \( M^* = (S^*, E^*, A^*, I^*, Q^*, R^*) \) in the model (2.1).

**Proof.** If the endemic equilibrium point \( M^* \) exists, the right-hand side functions in (2.1) must be zero at \( M^* \). By denoting

\[
N^* = S^* + E^* + A^* + I^* + R^* + Q^*,
\]

\[
\lambda^* = \frac{\beta_1 A^* + \beta_2 I^* + \beta_3 Q^*}{S^* + E^* + A^* + I^* + R^* + Q^*} = \frac{\beta_1 A^* + \beta_2 I^* + \beta_3 Q^*}{N^*},
\]

we have

\[
S^* = \frac{\Pi}{\mu} - \frac{m_4}{\mu} E^*, \quad A^* = \xi_0 E^*, \quad I^* = \xi_1 E^*, \quad Q^* = \xi_2 E^*, \quad R^* = \xi_3 E^*, \quad (2.4)
\]
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with positive constants

\[\begin{align*}
\xi_0 &= \frac{\alpha_1}{m_2}, \\
\xi_1 &= \frac{m_2 \alpha_2 + \sigma \alpha_1}{m_2 m_3}, \\
\xi_2 &= \frac{q_3 (\sigma \alpha_1 + m_2 \alpha_2) + m_3 (q_2 \alpha_1 + m_2 q_1)}{m_2 m_3 m_4}, \\
\xi_3 &= \frac{\gamma_1 \alpha_1 m_3 + \gamma_2 m_4 (m_2 \alpha_2 + \sigma \alpha_1) + \gamma_3 [q_3 (\sigma \alpha_1 + m_2 \alpha_2) + m_3 (q_2 \alpha_1 + m_2 q_1)]}{\mu m_2 m_3 m_4}.
\end{align*}\]

Finally \(\lambda^* S^* = m_1 E^*\) results

\[
\frac{\beta_1 A^* + \beta_2 I^* + \beta_3 Q^*}{S^* + E^* + A^* + I^* + Q^* + R^*} \left( \frac{\Pi}{\mu} - \frac{m_1}{\mu} E^* \right) = m_1 E^*,
\]

which determines \(E^*\), by (2.4), as

\[
E^* = \frac{\frac{\Pi}{\mu} (R_0 - 1)}{m_1 \mu (R_0 - 1) + 1 + \xi_0 + \xi_1 + \xi_3 + \xi_2}.
\]

Thus, \(E^*\) exists whenever \(R_0 > 1\).

Followed from (2.4), we know that the endemic equilibrium point \(M^*\) exists and is unique when \(R_0 > 1\).

About the stability of the endemic equilibrium point \(M^*\), we will provide some numerical simulation results later, instead of giving tedious and complicated relation with respect to the model parameters.

2.3. The final epidemic size without vital dynamics. Due to the rapid spreading of the COVID-19 virus, we can ignore the host mortality and recruitment in the model (2.1), that is \(\Pi = 0, \mu = 0\). Then (2.1) becomes,

\[
\begin{align*}
\dot{S} &= -\frac{\beta_1 A + \beta_2 I + \beta_3 Q}{N} S, \\
\dot{E} &= \frac{\beta_1 A + \beta_2 I + \beta_3 Q}{N} S - (q_1 + \alpha_1 + \alpha_2) E, \\
\dot{A} &= \alpha_1 E - (q_2 + \sigma + \gamma_1 + \theta_1) A, \\
\dot{I} &= \alpha_2 E + \sigma A - (\gamma_2 + \gamma_3 + \theta_1) I, \\
\dot{Q} &= q_1 E + q_2 A + q_3 I - \gamma_3 Q, \\
\dot{R} &= \gamma_1 A + \gamma_2 I + \gamma_3 Q.
\end{align*}
\]

How many people will experience COVID-19 infection throughout the outbreak? From the viewpoint of mathematical modelling, it is related to the final epidemic size. While during the pandemic outbreak, the social distancing and behavioural responses can significantly reduce the spreading of the virus. Such interventions are key to flattening the epidemic curve.

For the model (2.5), it is easy to obtain the same basic reproduction number \(R_0\) as that in (2.3). According to the relationship between \(R_0\) and \(\beta_i, i = 1, 2, 3\), it is obvious that when the contact rate \(\beta_i\) decreases, the basic reproduction number \(R_0\) decreases as well.

To explore the impact of intervention strategies in model (2.1), we take the effective contact rates \(\beta_i, (i = 1, 2, 3)\) in the classes \(A, I\) and \(Q\) as the control parameters and assume that they are time-dependent and the intervention occurs
at time $T$, that is
\[
\beta_i(t) = \begin{cases} 
\hat{\beta}_i, & t < T, \\
\hat{\beta}_i, & t \geq T,
\end{cases}
\]
with $\hat{\beta}_i < \beta_i$ for $i = 1, 2, 3$. Further, we assume that the total population $N$ is constant for $t \geq T$ (by counting the disease-related death together), denoted by $N_T := N(T)$, and denote
\[
R_c := R_{eff}(T) = R_0 \frac{S_T}{N_T},
\]
as an effective reproduction number.

To compute the final epidemic size rigorously, from the model (2.5), we have
\[
(S + E)' = -(q_1 + \alpha_1 + \alpha_2)E,
\]
\[
(S + E + A)' = -(q_1 + \alpha_2)E - (q_2 + \sigma + \gamma_1 + \theta_1)A,
\]
\[
(S + E + A + I)' = -q_1E - (q_2 + \gamma_1 + \theta_1)A - (\gamma_2 + q_3 + \theta_2)I,
\]
\[
(S + E + A + I + Q)' = - (\gamma_1 + \theta_1)A - (\gamma_2 + \theta_2)I - \gamma_3Q,
\]
\[
(S + E + A + I + Q + R)' = - \theta_1A - \theta_2I.
\]
(2.7)
Since all the solutions in the model (2.5) are non-negative and bounded, from (2.7) we can deduce that, as $t \to \infty$,
\[
E \to 0, \quad A \to 0, \quad I \to 0, \quad Q \to 0, \quad S(t) \to S(\infty) \geq 0.
\]
When $t \geq T$, define the function
\[
W_1(t) = A_1 \ln S(t) + \frac{A_2}{N_T}A(t) + \frac{A_3}{N_T}I(t) + \frac{A_4}{N_T}Q(t) + \frac{A_5}{N_T}R(t),
\]
with
\[
A_1 = \alpha_1 \gamma_3(q_3 \sigma + q_2 m_4 + \gamma_2 \sigma + \gamma_2 m_3) + \alpha_2 \gamma_3 m_2(q_3 + \gamma_2) + \gamma_3 q_1 m_2 m_3,
\]
\[
A_2 = \alpha_2 [(-\tilde{\beta}_1(q_2 \gamma_3 + \gamma_2 \gamma_3) + \tilde{\beta}_2(q_2 \gamma_3 + \gamma_2 \gamma_3) + \tilde{\beta}_3(q_3 \gamma_1 - q_2 \gamma_2)]
+ q_1 [-\tilde{\beta}_1 \gamma_3 m_3 - \tilde{\beta}_2 \gamma_2 \sigma + \tilde{\beta}_3 \gamma_1 m_3 + \tilde{\beta}_3 \gamma_2 \sigma],
\]
\[
A_3 = \alpha_1 [(-\tilde{\beta}_1(q_3 \gamma_3 + \gamma_2 \gamma_3) - \tilde{\beta}_2(q_2 \gamma_3 + \gamma_2 \gamma_3) + \tilde{\beta}_3(q_3 \gamma_1 - q_2 \gamma_2)] - q_1 m_2(\tilde{\beta}_2 \gamma_3 - \tilde{\beta}_3 \gamma_2),
\]
\[
A_4 = \alpha_1 [(-\tilde{\beta}_1 \gamma_3 m_3 - \tilde{\beta}_2 \gamma_2 \sigma + \tilde{\beta}_3 \gamma_1 m_3 + \tilde{\beta}_3 \gamma_2 \sigma)] + m_2 \sigma_2 (\tilde{\beta}_2 \gamma_3 - \tilde{\beta}_3 \gamma_2),
\]
\[
A_5 = \tilde{\beta}_1 \alpha_1 m_3 m_4 + \tilde{\beta}_2 m_4 (\sigma_1 + m_2 \sigma_2) + \tilde{\beta}_3 [q_3 (\sigma_1 + m_2 \sigma_2) + m_3 (q_2 \sigma_1 + m_2 q_1)].
\]
(2.9)
We can show that $W_1(t)$ is invariant for $t \geq T$ by calculating the derivatives along with the solution of (2.5), which is $W_1'(t) = 0$. Therefore we have $W_1(T) = W_1(\infty)$.

An epidemic ends when no infections remain. So with the further assumption
\[
E_\infty = 0, \quad A_\infty = 0, \quad I_\infty = 0, \quad Q_\infty = 0,
\]
and from (2.8) we have
\[
A_1 \ln S_T + \frac{A_2}{N_T}A_T + \frac{A_3}{N_T}I_T + \frac{A_4}{N_T}Q_T + \frac{A_5}{N_T}R_T = A_1 \ln S_\infty + \frac{A_5}{N_T}R_\infty,
\]
(2.10)
where $S_T, A_T, I_T, Q_T, R_T$ represents $S(T), A(T), I(T), Q(T), R(T)$, respectively.

Since the total number of individuals is assumed to be constant when $t \geq T$, so $N_T = S_T + E_T + A_T + I_T + Q_T + R_T = S_\infty + R_\infty + N_\infty$. Substituting $R_\infty = N_T - S_\infty$ into (2.10) yields
\[
A_1 \ln S_T + \frac{A_2}{N_T}A_T + \frac{A_3}{N_T}I_T + \frac{A_4}{N_T}Q_T + \frac{A_5}{N_T}R_T = A_1 \ln S_\infty + \frac{A_5}{N_T} (N_T - S_\infty).
\]
(2.11)
Using (2.6), we find
\[
\ln \frac{S_T}{S_\infty} = \frac{R_c m_1 m_2 m_3 m_4}{A_1 S_T} (N_T - S_\infty - R_T) - \frac{A_2}{A_1 N_T} A_T - \frac{A_3}{A_1 N_T} I_T - \frac{A_4}{A_1 N_T} Q_T. \tag{2.12}
\]
(2.12) provides a final size relation after the intervention is started at time \(T\), implying that taking the intervention into account can, indeed, lead to estimate of the final epidemic size much smaller and more realistic than estimate that by ignoring the intervention. More specifically, when \(T > 0\) (for example, \(T = 10, 20, 30, \cdots\)), the numbers of susceptible \((S_T)\), infectious without or with symptoms \((A_T, I_T)\), quarantined \((Q_T)\) and recovered groups \((R_T)\) at some time \(T\) can be confirmed through the statistics of the health department, then we can use the formula (2.12) to calculate the value of \(S_\infty\), so the final size of infected people \((N - S_\infty)\) can be obtained.

When \(T = 0\), with the initial condition \(S_0 \approx N_0, A_0 \approx I_0 \approx Q_0 \approx R_0 \approx 0\), the corresponding standard final size relation becomes
\[
\ln \left( \frac{S_0}{S_\infty} \right) = \frac{R_0 m_1 m_2 m_3 m_4}{A_1} \left( 1 - \frac{S_\infty}{S_0} \right), \tag{2.13}
\]
which presents a proportion of susceptible class from the starting of disease transmission to the end. Obviously, the estimate from (2.13) is larger than that by (2.12).

It is interesting to observe that, the formula obtained in (2.13) is different from most of the results in the literature (see \([1, 3, 5, 6, 11, 18]\)). What we can see is that quarantine is involved in our model which is part of the control measure in preventing the spreading of disease, while no such compartment(class) appeared in the mentioned references. While in the existed models with quarantine, there is no final size computation to our best knowledge!

3. Dynamics in the model with two age groups.

3.1. Model description. Although diseases can make anyone sick, there is an increased risk of more severe outcomes for vulnerable populations including the people aged 65 and over. The chance that a COVID-19 patient would develop symptoms severe enough to require hospitalization, especially for respiratory support, also rises sharply with age. In \([8]\), Del Valle et al. combined age-related residual immunity with mixed effects to develop an age-structured model of disease transmission. They confirmed that reducing the number of contacts in the population can slow down the spread of the disease, and found that if vaccination is necessary, it should first be given to the most vulnerable age groups. Miller et al. found a consistent trend in the distribution of COVID-19 cases in the United States and elsewhere, with a significantly higher incidence in the older group in \([19]\). After further adjusting for demographic and uncertainty factors, it was found in \([17, 29]\) that the case fatality rate was higher in the elderly group than in the other groups. Therefore the strategies focusing specifically on protecting high-risk elderly individuals should be considered in managing the pandemic. In this section, we separate the population into two age groups, one with the age below 65 years old (denote as group-I), and another one with age 65 and over (denote as group-II). To capture the dynamics of the infectious virus more accurately, with the fact that hospitalization and intensive care unit admission rates are also higher among the elders, we further classify the infectious individuals into different stages including mild symptoms who need self-isolation, patient in hospital and patient in intensive care, according to the severity
of infection. With the interaction between these two groups, the architecture of the disease transmission flow is given in Figure 2, there the natural mortality rate is not shown in the figure for simplicity.

\[
\begin{align*}
\dot{S}_i &= \Pi_i - \lambda(t)S_i - \mu_i S_i, \\
\dot{E}_i &= \lambda(t)S_i - (q_i^2 + a_i^1 + a_i^2 + \mu_i)E_i, \\
\dot{A}_i &= a_i^1 E_i - (q_i^1 + s_i^5 + \gamma_i^1 + d_i^1 + \mu_i)A_i, \\
\dot{I}_i^1 &= a_i^2 E_i + s_i^2 A_i - (\gamma_i^3 + q_i^3 + s_i^1 + d_i^2 + \mu_i)I_i^1, \\
\dot{I}_i^2 &= s_i^1 I_i^1 + \gamma_i^5 I_i^3 - (\gamma_i^4 + s_i^2 + d_i^3 + \mu_i)I_i^2, \\
\dot{I}_i^3 &= s_i^2 I_i^2 - (\gamma_i^5 + d_i^4 + \mu_i)I_i^3, \\
\dot{Q}_i &= q_i^1 A_i + q_i^2 E_i + q_i^3 I_i^1 - (\gamma_i^2 + \mu_i)Q_i, \\
\dot{R}_i &= \gamma_i^1 A_i + \gamma_i^2 Q_i + \gamma_i^3 I_i^1 + \gamma_i^4 I_i^2 - \mu_i R_i,
\end{align*}
\] 

Figure 2. Architecture of disease transmission flow with two age groups.

Mathematically, the model can be represented by the following system of ordinary equations:

\[
\begin{align*}
\dot{S}_i &= \Pi_i - \lambda(t)S_i - \mu_i S_i, \\
\dot{E}_i &= \lambda(t)S_i - (q_i^2 + a_i^1 + a_i^2 + \mu_i)E_i, \\
\dot{A}_i &= a_i^1 E_i - (q_i^1 + s_i^5 + \gamma_i^1 + d_i^1 + \mu_i)A_i, \\
\dot{I}_i^1 &= a_i^2 E_i + s_i^2 A_i - (\gamma_i^3 + q_i^3 + s_i^1 + d_i^2 + \mu_i)I_i^1, \\
\dot{I}_i^2 &= s_i^1 I_i^1 + \gamma_i^5 I_i^3 - (\gamma_i^4 + s_i^2 + d_i^3 + \mu_i)I_i^2, \\
\dot{I}_i^3 &= s_i^2 I_i^2 - (\gamma_i^5 + d_i^4 + \mu_i)I_i^3, \\
\dot{Q}_i &= q_i^1 A_i + q_i^2 E_i + q_i^3 I_i^1 - (\gamma_i^2 + \mu_i)Q_i, \\
\dot{R}_i &= \gamma_i^1 A_i + \gamma_i^2 Q_i + \gamma_i^3 I_i^1 + \gamma_i^4 I_i^2 - \mu_i R_i,
\end{align*}
\] 

where \( i = 1, 2 \) is the index of the group, the description of all the variables is given in Table 2.

**Table 2. Variable descriptions (i=1,2)**

| Variable | Description            |
|----------|-----------------------|
| \( S_i(t) \) | Susceptible population |
| \( E_i(t) \) | Exposed population   |
| \( A_i(t) \) | Infectious population without symptoms |
| \( I_i^1(t) \) | Infectious population with mild symptoms |
| \( I_i^2(t) \) | Patient in hospital |
| \( I_i^3(t) \) | Patient in intensive care |
| \( Q_i(t) \) | Quarantined population |
| \( R_i(t) \) | Recovered population   |
The quarantine-adjusted infection rate function $\lambda(t)$ has the following form:

$$
\lambda(t) = \frac{1}{N_1 + N_2} \sum_{i=1}^{2} (\beta_{1i} A_i + \beta_{2i} I_i^1 + \beta_{3i} I_i^2 + \beta_{4i} I_i^3 + \beta_{5i} Q_i),
$$

and $N_i = S_i + E_i + A_i + Q_i + I_i^1 + I_i^2 + I_i^3 + R_i$, $(i = 1, 2)$. The description of the parameters and their value ranges are shown in Table 3. Being short of real data, most of the parameter values are chosen by assumption there.

| Parameter | Description | Values (per day) | Source |
|-----------|-------------|------------------|--------|
| $\beta_{ij}$ | Effective contact rate in each group | 0.081-0.591 | [9] |
| $\alpha_{ij}$ | Progression rate from $E_i$ to $A_i$ | 0.01881, 0.02881 | [25] |
| $\alpha_{ij}$ | Progression rate from $E_i$ to $I_i^1$ | 0.15699, 0.16699 | [22] |
| $q_{ij}$ | Quarantine rate for $A_i$ | 0.43, 0.1 | [22] |
| $q_{ij}$ | Quarantine rate for $E_i$ | 0.45, 0.12 | assumed |
| $q_{ij}$ | Quarantine rate for $I_i^1$ | 0.52, 0.13 | assumed |
| $\gamma_{ij}$ | Recovery rate for $A_i$ | 0.13978, 0.12978 | [25] |
| $\gamma_{ij}$ | Recovery rate for $Q_i$ | 0.1, 0.09 | assumed |
| $\gamma_{ij}$ | Recovery rate for $I_i^1$ | 0.08, 0.06 | assumed |
| $\gamma_{ij}$ | Recovery rate for $I_i^2$ | 0.06, 0.04 | assumed |
| $\gamma_{ij}$ | Recovery rate for $I_i^3$ | 0.04, 0.01 | assumed |
| $s_{ij}$ | Symptom progression rate from $A_i$ to $I_i^1$ | 0.05, 0.06 | assumed |
| $s_{ij}$ | Symptom progression rate from $I_i^1$ to $I_i^2$ | 0.06, 0.08 | assumed |
| $s_{ij}$ | Symptom progression rate from $I_i^2$ to $I_i^3$ | 0.07, 0.09 | assumed |
| $d_{ij}$ | Disease-induced death rate for $A_i$ | 0.02, 0.03 | assumed |
| $d_{ij}$ | Disease-induced death rate for $I_i^1$ | 0.04227, 0.05227 | [22] |
| $d_{ij}$ | Disease-induced death rate for $I_i^2$ | 0.05227, 0.08227 | assumed |
| $d_{ij}$ | Disease-induced death rate for $I_i^3$ | 0.06227, 0.1 | assumed |

3.2. Dynamical analysis. First of all, we know that the disease-free equilibrium (DFE) $M^0 = (M^0_1, M^0_2)$ always exists in the model (3.1), where $M^0 = (\Pi_1, 0, 0, 0, 0, 0, 0, 0)$, $M^0_2 = (\Pi_2, 0, 0, 0, 0, 0, 0, 0)$. With tedious computation, we can obtain the explicit relation of the basic reproduction number $R_0$ with respect to the parameters, that is,

$$
R_0 = \frac{1}{n_{\mu_1} + n_{\mu_2}} \sum_{i=1}^{2} \frac{\Pi_i}{\mu_i} v_i^0 v_i^0 v_i^0 v_i^0 \\
\times [(\beta_{1i} \alpha_{i1}^3 v_i^3 n_{i1} + \beta_{2i} n_{i1} n_{i2} + \beta_{3i} n_{i2} s_i^3 v_i^5 + \beta_{4i} n_{i2} s_i^3 s_i^3 v_i^5 + \beta_{5i} n_{i1} n_{i3})] \\
:= \frac{1}{n_{\mu_1} + n_{\mu_2}} \sum_{i=1}^{2} \frac{\Pi_i}{\mu_i} R_{ii0}
$$

with

$$
R_{ii0} = (\beta_{1i} \alpha_{i1}^3 v_i^3 n_{i1} + \beta_{2i} n_{i1} n_{i2} + \beta_{3i} n_{i2} s_i^3 v_i^5 + \beta_{4i} n_{i2} s_i^3 s_i^3 v_i^5 + \beta_{5i} n_{i1} n_{i3}) v_i^0 + \beta_{5i} n_{i1} n_{i3},
$$

where

$$
n_{i1} = v_i^4 v_i^5 - s_i^2 \gamma_i > 0, \quad n_{i2} = \alpha_{i1} s_i^3 + \alpha_{i1} v_i^2, \\
n_{i3} = \alpha_{i1} (q_i^3 v_i^3 + q_i^3 s_i^3) + v_i^7 (\alpha_{i1} q_i^3 + v_i^3 q_i^3),
$$

and $n_{i1}, n_{i2}, n_{i3}$ are the parameter values chosen by assumption there.
Theorem 3.1. The disease-free equilibrium (DFE) $M^0$ in the model (3.1) is globally asymptotically stable (GAS) in $\mathcal{D}_1$ whenever $R_{10} + R_{20} < 1$.

Proof. Consider the following Lyapunov function:

$$V_2 = \sum_{i=1}^{2} x_i E_i + y_i A_i + z_1^1 I_1^1 + z_1^2 I_2^1 + z_1^3 I_3^1 + g_i Q_i,$$

where

$$x_1 = \frac{R_{10} - R_{20}}{R_{10}}, \quad x_2 = R_{20},$$

$$y_i = \frac{\beta_1 v_i^1 v_i^0 n_{1i} + \beta_2 v_i^1 s_i^0 v_i^6 + \beta_3 s_i^1 s_i^0 v_i^6 + \beta_4 s_i^1 s_i^0 v_i^6 + \beta_5 n_{1i}(q_i^1 v_i^3 + q_i^3 v_i^3)}{v_i^1 v_i^0 n_{1i}},$$

$$z_1^1 = \frac{\beta_2 v_i^6 n_{1i} + \beta_3 v_i^1 v_i^6 + \beta_4 s_i^1 s_i^0 v_i^6 + \beta_5 q_i^3 n_{1i}}{v_i^1 v_i^0 n_{1i}}; \quad z_2^1 = \frac{\beta_3 \gamma_i + \gamma_i v_i^4}{n_{1i}}, \quad z_3^1 = \frac{\beta_5 v_i^4}{v_i^6}.$$

The Lyapunov derivative along the solution of (3.1) is

$$V_2 = \sum_{i=1}^{2} (x_i(\lambda(t)S_i - v_i^1 E_i) + y_i(v_i^1 E_i - v_i^2 A_i)) + z_1^1 (\alpha_i^2 E_i + s_i^0 A_i - v_i^3 I_3^1)$$

$$+ z_2^1 (s_i^0 I_3^1 + q_i^3 I_3^1 - v_i^1 E_i) + z_3^1 (s_i^0 I_3^1 + q_i^3 I_3^1) + g_i(q_i^1 A_i + q_i^3 I_3^1 - v_i^1 E_i))$$

$$= \sum_{i=1}^{2} x_i(\lambda(t)S_i - \beta_1 A_i - \beta_2 I_1^1 - \beta_3 I_2^1 - \beta_4 I_3^1 - \beta_5 Q_i)$$

$$= (x_1 S_1 + x_2 S_2) \lambda(t) - \sum_{i=1}^{2} (\beta_1 A_i + \beta_2 I_1^1 + \beta_3 I_2^1 + \beta_4 I_3^1 + \beta_5 Q_i)$$

$$= N_1 S_1 + N_2 S_2 \sum_{i=1}^{2} (\beta_1 A_i + \beta_2 I_1^1 + \beta_3 I_2^1 + \beta_4 I_3^1 + \beta_5 Q_i)$$

$$- \sum_{i=1}^{2} (\beta_1 A_i + \beta_2 I_1^1 + \beta_3 I_2^1 + \beta_4 I_3^1 + \beta_5 Q_i)$$

$$= \frac{S_1}{N_1 + N_2} R_{10} + \frac{S_2}{N_1 + N_2} R_{20} - 1 \sum_{i=1}^{2} (\beta_1 A_i + \beta_2 I_1^1 + \beta_3 I_2^1 + \beta_4 I_3^1 + \beta_5 Q_i).$$
\begin{eqnarray*}
< (R_{10} + R_{20} - 1) \sum_{i=1}^{2} (\beta_{1i} I_i + \beta_{2i} I_i^2 + \beta_{3i} I_i^3 + \beta_{4i} I_i^4 + \beta_{5i} Q_i).
\end{eqnarray*}

Therefore, \( \dot{V}_2 \leq 0 \) for \( R_{10} + R_{20} < 1 \) and \( \dot{V}_2 = 0 \) if and only if \( A_i = I_i = I_i^2 = I_i^3 = Q_i = 0 \) (i=1,2).

Thus \((E_i(t), A_i(t), I_i^1(t), I_i^2(t), I_i^3(t), Q_i(t)) \to (0,0,0,0,0,0) \) as \( t \to \infty \), and consequently \( R_i(t) \to 0 \) and \( S_i(t) \to \frac{\Pi_i}{\mu_i} \) as \( t \to \infty \). So the DFE \( M^0 \) is globally asymptotically stable if \( R_{10} + R_{20} < 1 \).

\[ \square \]

**Remark 1.** (i) With \( \Pi_1 = \Pi_2 \) and \( \mu_1 = \mu_2 \), this global stability condition becomes \( R_0 < \frac{1}{2} \) since \( R_0 = \frac{1}{2} (R_{10} + R_{20}) \) from (3.2).

(ii) We should mention that the condition \( R_{10} + R_{20} < 1 \) is strong, at least for the case in (i), comparing with the normal condition \( R_0 < 1 \), although it is acceptable due to the complexity of the model. How to construct an appropriate Lyapunov function or use some other methods to obtain weaker condition (e.g., \( R_0 < 1 \)) to ensure the global stability of DFE is not trivial and open to discussion.

Different from one-group model (2.1), in addition to the DFE \( M^0 \), there exists one-group disease free (or partial disease free) equilibrium in this two-group model (3.1). Regarding the high risk for the group with age 65 and over, we discuss the condition for the existence of group-I disease free equilibrium \( M^*_1 = (P^*_1, P^*_2) \), where \( P^*_1 = (\frac{\Pi_1}{\mu_1}, 0, 0, 0, 0, 0, 0) \) and \( P^*_2 = (S^*_2, E^*_2, A^*_2, I^*_2, I^*_2, Q^*_2, Q^*_2) \). That is the case where disease only exists in the group with age 65 or over.

**Lemma 3.2.** When \( R_{20} > \frac{\Pi_1 \mu_2}{\Pi_2 \mu_1} + 1 \), there is a unique group-I disease free equilibrium \( M^*_1 \) in the model (3.1).

**Proof.** By letting \((E_1, A_1, I_1^1, I_1^2, I_1^3, Q_1, R_1) \) = \((0,0,0,0,0,0,0) \), from the right-hand side functions in the model (3.1), we know the components in \( M^*_1 \) must satisfy

\begin{align*}
S^*_1 &= \frac{\Pi_1}{\mu_1}, \quad S^*_2 = \frac{\Pi_2}{\mu_2}, \quad v_1^2 v_2^3 v_2^3, \quad I^*_1 = \frac{v_1^2 v_2^3 v_2^3}{\mu_2 n_{22}}, \quad E^*_2 = \frac{v_2^2 v_2^3}{n_{22}}, \quad A^*_2 = \frac{\alpha_2^2 v_2^3}{n_{22}}, \quad I^*_2 = \frac{\beta_1^2 I_2^1}{n_{21}}, \quad Q^*_2 = \frac{n_{23}}{v_2^2 n_{22}}, \\
I^*_2 &= \frac{\beta_2^2 I_2^1}{n_{21}}, \quad I^*_2 = \frac{\beta_3^2 I_2^1}{n_{21}}, \quad Q^*_2 = \frac{n_{23}}{v_2^2 n_{22}}, \quad E^*_2 = \frac{\gamma_2^2 v_2^3}{n_{21}}, \quad \gamma_2^2 \frac{v_2^3}{n_{21}} | E^*_2. \tag{3.4}
\end{align*}

Then \( \lambda(t^*) S^*_2 = v_1^2 E^*_2 \) results,

\begin{equation}
\frac{\beta_1^2 A^*_2 + \beta_2^2 I^*_2 + \beta_3^2 I^*_2 + \beta_4^2 I^*_2 + \beta_5^2 Q^*_2}{S^*_1 + S^*_2 + E^*_2 + A^*_2 + I^*_2 + I^*_2 + I^*_2 + Q^*_2 + R^*_2} S^*_2 = v_1^2 E^*_2. \tag{3.5}
\end{equation}

Substituting (3.4) into (3.5), we obtain

\begin{equation}
R_{20} \frac{v_1^2 v_2^3 v_2^3}{n_{22}} I^*_2 S^*_2 = v_1^2 E^*_2 (S^*_1 + S^*_2 + E^*_2 + A^*_2 + I^*_2 + I^*_2 + I^*_2 + Q^*_2 + R^*_2). \tag{3.6}
\end{equation}

Further substituting (3.4) into (3.6), we can solve \( I^*_2 \) explicitly,

\begin{equation}
I^*_2 = \frac{\frac{\Pi_2}{\mu_2} (R_{20} - 1) - \frac{\Pi_1}{\mu_1}}{\frac{v_1^2 v_2^3 v_2^3}{\mu_2 n_{22}} (R_{20} - 1) + M_2},
\end{equation}
that

The final epidemic size without vital dynamics.

is asymptotically stable under certain conditions. Numerical simulation of diseased equilibrium. We find that the diseased equilibrium diseases. To compensate for the limitations of theoretical analysis, we will give a degree of illness can help to formulate strategies to combat the spread of infectious diseases. To compensate for the limitations of theoretical analysis, we will give a numerical simulation of diseased equilibrium. We find that the diseased equilibrium is asymptotically stable under certain conditions.

3.3. The final epidemic size without vital dynamics. First we notice that with \( \Pi_i = \mu_i = 0 \), \( i = 1, 2 \), the disease-free equilibrium is \( M^0 = (\tilde{P}_1, \tilde{P}_2) \) with \( \tilde{P}_i = (S^0_i, 0, 0, 0, 0, 0, 0) \), where \( S^0_i = N^0_i \) is the initial number of susceptible individuals in each group.

Consequently, we can obtain the corresponding basic reproduction number as

\[
\mathcal{R}_0 = \frac{1}{S^0_1 + S^0_2} \sum_{i=1}^{2} \frac{S^0_i}{v^0_i n_i v_i^0} \times \left[ (\beta_{13} n_i^1 v_i^1 + \beta_{25} n_i^2 v_i^2) + (\beta_{35} n_i^3 v_i^3) + (\beta_{45} n_i^4 v_i^4) + (\beta_{55} n_i^5 v_i^5) \right]
\]

\[
:= \frac{1}{S^0_1 + S^0_2} \sum_{i=1}^{2} S^0_i \mathcal{R}_{i0},
\]

(3.7)

which is different from that given in (3.2) although they have the same \( \mathcal{R}_{i0} \) in (3.3).

By using similar intervention strategies as that in the previous section for one-group model, and denote \( \beta_{ij} \) to ignore the tilde as the control parameters. Assuming that \( N_1, N_2 \) are constants for \( t \geq T \), denoted by \( N^T_1 := N_1(T) \), \( N^T_2 := N_2(T) \), and \( N_T = N^T_1 + N^T_2 \). From the model without vital dynamics, we can deduce that, as \( t \to \infty, E_i \to 0, A_i \to 0, I^1_i \to 0, I^2_i \to 0, I^3_i \to 0, Q_i \to 0, \) and \( S_i(t) \to S_i(\infty) \geq 0 \).

To find the final epidemic size relation, we define the following function:

\[
W_2(t) = \sum_{i=1}^{2} W_{2i}(t)
\]

\[
= \sum_{i=1}^{2} \left( B_{11} \ln S_i(t) + \frac{B_{12}}{N_T} A_i(t) + \frac{B_{13}}{N_T} I^1_i(t) + \frac{B_{14}}{N_T} I^2_i(t) + \frac{B_{15}}{N_T} I^3_i(t) + \frac{B_{16}}{N_T} Q_i(t) + \frac{B_{17}}{N_T} R_i(t) \right),
\]

(3.8)
where the quantities of $B_{13}$, $B_{22}$, $B_{33}$, $B_{44}$, $B_{55}$, $B_{66}$, and $B_{77}$ are given in Appendix. To show that $W_2(t)$ is invariants for $t \geq T$, we calculate,

$$W_2'(t) = - \frac{B_{13}}{N_T} \sum_{i=1}^{2} (\beta_{11} A_{i} + \beta_{22} I_{1i} + \beta_{33} I_{2i} + \beta_{44} I_{3i} + \beta_{55} Q_{i})$$

$$+ \frac{B_{22}}{N_T} (\alpha_{1} E_{i} - v_{1}^{2} A_{i}) + \frac{B_{33}}{N_T} (\alpha_{2} E_{i} + s_{i}^{2} A_{i} - v_{2}^{2} I_{1i})$$

$$+ \frac{B_{44}}{N_T} (s_{i}^{1} I_{1i} + \gamma_{i}^{5} I_{3i} - v_{4}^{3} I_{2i}^{2}) + \frac{B_{55}}{N_T} (s_{i}^{2} I_{1i}^{2} - v_{5}^{3} I_{3i}^{2})$$

$$+ \frac{B_{66}}{N_T} (q_{i}^{1} A_{i} + q_{i}^{2} E_{i} + q_{i}^{3} I_{1i}^{3} - v_{6}^{5} Q_{i})$$

$$+ \frac{B_{77}}{N_T} (\gamma_{i}^{1} A_{i} + v_{7}^{4} Q_{i} + \gamma_{i}^{3} I_{1i}^{2} + \gamma_{i}^{4} I_{2i}^{3})$$,

then $W_2'(t) = \sum_{i=1}^{2} W_2'(i) = 0$.

For convenience, we denote $S_{1}^{i} = S_{i}(T)$, $E_{1}^{i} = E_{i}(T)$, $A_{1}^{i} = A_{i}(T)$, $I_{1i}^{1} = I_{1i}^{1}(T)$, $I_{1i}^{2} = I_{1i}^{2}(T)$, $I_{1i}^{3} = I_{1i}^{3}(T)$, $Q_{1i}^{i} = Q_{i}(T)$, and $R_{1i}^{i} = R_{i}(T)$, and the effective reproduction number $R_{e} = R_{eff}(T)$ as

$$R_{e} = R_{0} \frac{S_{i}^{T} + S_{2}^{T}}{N_{T}} = \frac{S_{i}^{T} + S_{2}^{T}}{N_{T}(S_{1}^{T} + S_{2}^{T})} \sum_{i=1}^{2} S_{i}^{0} B_{i} \tau \frac{S_{i}^{T} + S_{2}^{T}}{N_{T}(S_{1}^{T} + S_{2}^{T})} \sum_{i=1}^{2} S_{i}^{0} R_{i} \tau \frac{S_{i}^{T} + S_{2}^{T}}{N_{T}(S_{1}^{T} + S_{2}^{T})} \sum_{i=1}^{2} S_{i}^{0} R_{i} \tau$$

$$= \sum_{i=1}^{2} \left( B_{11} \ln S_{i}(\infty) + \frac{B_{i}}{N_{T}} R_{i}(\infty) \right).$$

We then can explore the relation of the final epidemic size from the invariance of $W_2(t)$. Since $W_2(T) = W_2(\infty)$, that is, by (3.8),

$$\sum_{i=1}^{2} \left( B_{11} \ln S_{i}^{T} + \frac{B_{22}}{N_{T}} A_{1i}^{T} + \frac{B_{33}}{N_{T}} I_{1i}^{1T} + \frac{B_{44}}{N_{T}} I_{1i}^{2T} + \frac{B_{55}}{N_{T}} I_{1i}^{3T} + \frac{B_{66}}{N_{T}} Q_{1i}^{T} + \frac{B_{77}}{N_{T}} R_{1i}^{T} \right)$$

$$= \sum_{i=1}^{2} \left( B_{11} \ln S_{i}(\infty) + \frac{B_{i}}{N_{T}} R_{i}(\infty) \right).$$

Since the total number of individuals at time $T$ is $N_{T} = S_{1}^{T} + S_{2}^{T}$, where $N_{T} = S_{i}^{T} + E_{i}^{T} + A_{i}^{T} + I_{1i}^{1T} + I_{1i}^{2T} + I_{1i}^{3T} + Q_{1i}^{T} + R_{1i}^{T}$, $i = 1, 2$ is constant, and $(E_{1}^{i} + A_{1i}^{i} + I_{1i}^{i} + I_{1i}^{2} + I_{1i}^{3} + Q_{1i}^{i})(\infty) = 0$, we have $N_{T}^{T} = S_{i}(\infty) + R_{i}(\infty)$. Then, $R_{i}(\infty) = N_{i}^{T} - S_{i}(\infty)$.

Substituting $R_{i}(\infty)$ into (3.10) results

$$\sum_{i=1}^{2} \left( B_{11} \ln S_{i}^{T} + \frac{B_{22}}{N_{T}} A_{1i}^{T} + \frac{B_{33}}{N_{T}} I_{1i}^{1T} + \frac{B_{44}}{N_{T}} I_{1i}^{2T} + \frac{B_{55}}{N_{T}} I_{1i}^{3T} + \frac{B_{66}}{N_{T}} Q_{1i}^{T} + \frac{B_{77}}{N_{T}} R_{1i}^{T} \right)$$

$$= \sum_{i=1}^{2} \left( B_{11} \ln S_{i}(\infty) + \frac{B_{i}}{N_{T}} (N_{i}^{T} - S_{i}(\infty)) \right).$$

Using (3.9), we find

$$\sum_{i=1}^{2} B_{11} \ln \left( \frac{S_{i}^{T}}{S_{i}(\infty)} \right) = \sum_{i=1}^{2} \frac{R_{i}^{T}(S_{i}^{T} + S_{2}^{T})}{(S_{1}^{T} + S_{2}^{T}) S_{i}^{0}} v_{1}^{i} v_{2}^{i} v_{3}^{i} v_{4}^{i} n_{1}(N_{i}^{T} - S_{i}(\infty) - R_{i}^{T})$$

$$- \sum_{i=1}^{2} \left( B_{22} A_{1i}^{T} + B_{33} I_{1i}^{1T} + B_{44} I_{1i}^{2T} + B_{55} I_{1i}^{3T} + B_{66} Q_{1i}^{T} \right).$$

(3.12)
which determine the final size relation to the epidemic situation at the intervention time $T$, effective basic reproduction number and the system parameters. Although it is tedious and intricate to estimate the final size of infected people in such complicated models, the formula (3.12), at least, can provide a way to approximate the final size from $N - S_i(\infty) - S_{2i}(\infty)$. In practice, several different mechanisms, including differences in susceptibility or transmission potential between different age groups, may contribute to the variety.

When $T = 0$, assuming that $S_i^0 \approx N_i^0$, $E_i^0 \approx A_i^0 \approx I_i^1 \approx I_i^{20} \approx I_i^{30} \approx Q_i^0 \approx R_i^0 \approx 0$, the corresponding standard final size relation is

$$2 \sum_{i=1}^{2} B_{i1} \ln \left( \frac{S_i^0}{S_i(\infty)} \right) = \frac{1}{N_0} \sum_{i=1}^{2} R_{i0} S_i^0 v_i^1 v_i^2 v_i^3 v_i^6 n_{i1} \left( 1 - \frac{S_i(\infty)}{S_i^0} \right).$$

4. Numerical simulation. Previously we have done some theoretical analysis for the models (2.1) and (3.1), due to the complexity of the models, in this section, we implement some numerical simulation to explore more dynamic properties and estimate the final epidemic sizes in Canada and Newfoundland & Labrador.

I. The stability of epidemic equilibrium. For the one-group model (2.1), based on the data in Table 1, we can calculate the basic reproduction number $R_0 = 5.2515$, and know that the endemic equilibrium $M^*$ exists and is unique. When choosing different initial values, each system ((2.1) and (3.1)) approaches the same positive equilibrium point $M^*$ (see Figure 3 for the one-group model and Figure 4 for the two-group model). We can conjecture that $M^*$ may be globally asymptotically stable, while the theoretical proof is non trivial.

In the two-group model (3.1), we assume that the initial population ratio is 5:1 for the first group (under 65) and the second group (above 65). From the numerical simulation we can observe that, in Figure 4, the system approaches to a steady state with the total patients number 89 and 43 respectively, implying the ratio is about 2:1. The simulation result indicates that the older adults are more susceptible to infection.

II. The final epidemic size estimation. Through theoretical analysis, we have obtained the final epidemic size relationship of the prognosis. These analytical formulas (2.12) and (3.12) can be used to predict the total number of infected cases...
over the entire outbreak period and help us understand what might happen and possibly drive public health action to achieve the best possible outcome.

a. Data based on Canada: Since the first four confirmed COVID-19 cases were reported in Canada on January 31, 2020, the number was increased to 15 on February 29. With the spreading of the virus, the confirmed number is blowing. Up to June 20, 2020, the number of confirmed cases had raised to 101K including 8410 deaths, from www.canada.ca/en/public-health/services/diseases/2019-novel-coronavirus-infection. In a released report by Government of Canada on April 9, 2020, the projected infection number will reach from 940000 to 1879000, under different control intensities.

The total population in Canada is $N_1 = 37590000$ from the website of Statistics Canada. To apply our model (2.1), we take January 31, 2020 as the initial time, and the initial condition is $I(0) = 4, E(0) = A(0) = Q(0) = R(0) = 0$, and $S(0) = 37589996$. We assume the intervention is carried after 30 days (at Feb. 29, 2020), implying $T = 30$. Using the same parameter values given in Table 1 except $\Pi = \mu = 0, \gamma_3 = 0.51624$, we can obtain the basic reproduction number $R_0 = 1.6768$, and numerical simulation from (2.1) gives $S_T = 37589888, E_T = 25, A_T = 1, I_T = 14, Q_T = 7, R_T = 56$, here $I_T = 14$ is almost consistent with the reported confirmed cases 15 from Government of Canada from Jan. 31 to Feb. 29, 2020.

The reflection of the public intervention (school closing, work from home, wear the mask, avoid group gathering, etc.) in our model (2.1) is assumed to be the reduction of contact rate $\beta_i$ in each class. If we assume the overall contact rates are dropped to 20–80%, correspondingly we can calculate a final size of $S_\infty$ which can determine a total number of disease cases throughout the epidemic (see Table 4), and draw the relationship between the reduction of contact rate and infection cases in Canada, see Figure 5.

Obviously, the reduction of the contact rate is one of the powerful measures to combat the spread of COVID-19 across Canada and flattening the pandemic curve. In addition, we find 50-55% of the contact rate is a considerably better estimate, comparing with the real data. In our prediction, when the contact rate is reduced by about 51.3% or 52%, the associated number of infection cases is 934929 or 1919191 respectively, which is approximately consistent with the prediction in the national report: 940000, 1879000. In particular, when the contact rate is reduced by about 50.71%, the disease cases is 109652, which is roughly consistent with the reported
Table 4. Cases over time in Canada

| % of contact rate (β_i) | S∞ | Disease Cases (N1-S∞) |
|------------------------|-----|-----------------------|
| 1                      | 7911534 | 29678466 |
| 0.8                    | 13889287 | 23700713 |
| 0.6                    | 26530691 | 11059309 |
| 0.55                   | 31820452 | 5769548  |
| 0.5                    | 37585587 | 4413     |
| 0.4                    | 37589673 | 327      |
| 0.2                    | 37589851 | 149      |

Figure 5. The relationship between the reduction of contact rates and infection cases in Canada.

Cases in Canada so far. This model simulation indicates that reducing the contact rate by half is probably the current reality, comparing with the normal situation before COVID-19 time. From Figure 5, we can see there is a big jump in the number of cases around a critical percentage of contact rates, which is close to 50% as well.

From the Statistics Canada website, we know that the proportion of the population with age 65 and above is about 17.2% in Canada. When applying our two group model (3.1), we have $N^0_1 \approx 31124520$, $N^0_2 \approx 6465480$. According to news reports (see https://globalnews.ca/news), we know the first four confirmed cases were under the age of 65, so the initial condition in (3.1) is, $(31124516, 0, 0, 0, 0, 0)$ and $(6465480, 0, 0, 0, 0, 0)$ for each group. Taking the intervention time $T = 30$ again, we can obtain $S^T_1 = 31124456$, $E^T_1 = 6$, $A^T_1 = 1$, $I^T_1 = 1$, $Q^T_1 = 22$, $R^T_1 = 31$, $S^T_2 = 6465468$, $E^T_2 = 2$, $A^T_2 = 1$, $I^T_2 = 1$, $Q^T_2 = 3$, $R^T_2 = 4$ by using the parameters in Table 3, and

\[
\begin{align*}
\beta_{11} &= 0.491; \beta_{12} = 0.391; \beta_{13} = 0.32; \beta_{14} = 0.3; \beta_{15} = 0.081; \beta_{21} = 0.591; \\
\beta_{22} &= 0.491; \beta_{23} = 0.49; \beta_{24} = 0.481; \beta_{25} = 0.48; \Pi_i = 0; \mu_1 = 0.
\end{align*}
\]

Then from (3.2), we have $R_0 = 1.6679$.

Applying the final size relation (3.12), we calculate a final size of $S_1(\infty)$ and $S_2(\infty)$, giving a total number of disease cases $N^0_i - S_i(\infty)$ over the course of the epidemic, with different contact rate (see Table 5). From Table 5, we can see that 40-60% of contact rate deduction gives a better match for the real data. In our prediction, when the reduction of the contact rates is around 50.5% and 55% (see Figure 6), the corresponding number of infected cases is 943765 and 1844531, respectively, which has good agreement with the result obtained from the model.
The proportion of infected people for each group is \(1 - \frac{S_i(\infty)}{N_0i}\). The prediction is given in Table (5) confirms the factor that the proportion of infectious people in group-I was lower than that in group-II with aged over 65.

### Table 5. Cases over time in Canada for two groups

| % of \(S_{ij}\) | \(S_1(\infty)\) | \(S_2(\infty)\) | Disease cases (I) | % of \(\frac{N_0^0 - S_1(\infty)}{N_0^0}\) | Disease cases (II) | % of \(\frac{N_0^0 - S_2(\infty)}{N_0^0}\) | Total cases |
|----------------|----------------|----------------|------------------|------------------|------------------|------------------|------------|
| 1              | 9811036        | 1091182        | 21313484         | 0.68             | 5374298          | 0.83             | 26687782   |
| 0.8            | 16552499       | 1961078        | 14572030         | 0.47             | 4504402          | 0.7              | 19076432   |
| 0.6            | 20413213       | 3831763        | 711307           | 0.02             | 2633717          | 0.41             | 3345024    |
| 0.4            | 31124381       | 6465416        | 139              | 4.5 \times 10^{-6} | 64              | 9.9 \times 10^{-6} | 203        |
| 0.2            | 31124437       | 6465461        | 83               | 2.7 \times 10^{-6} | 19              | 2.9 \times 10^{-6} | 102        |

**Figure 6.** The relationship between the change of contact rates and infection cases for two groups in Canada.

b. Data based on Newfoundland & Labrador. In Canada, Newfoundland & Labrador is among the oldest provinces with respect to median age and has a high incidence of chronic disease. Due to its particular location and cluster effect at the beginning of spreading of COVID-19 virus, since March 14, 2020, the province reported the first case, the number was jumped to 152 on March 31. By April 30, the total number of confirmed cases was 258. During those days the province experienced what was at the time the biggest single outbreak in Canada, related to “the Caul’s cluster” which infected 167 people - 64 percent of the total cases in the province. The number 261 has been lasted for more than 20 days. Up to June 18, 2020, all infected people are recovered, except three death.

The total population in Newfoundland & Labrador (NL) is \(N_2 = 521542\) from Statistics Canada. Since the few cases in the province, we only do the simulation to the model (2.1). Taking March 14, 2020 as the initial time with initial condition \(I(0) = 1, E(0) = A(0) = Q(0) = R(0) = 0\) and \(S(0) = 521541\). With the same intervention time \(T = 30\), we have \(S_T = 521515, A_T = 1, I_T = 4, Q_T = 2, R_T = 14\) from (2.1). Following the final size relation (2.12), we can estimate the number of the infected case, which is shown in Table 6 and Figure 7, with different contact rates reduction. We can see that 40-50% of contact rate reduction is more reliable. Specifically, when the percentage is 48%, the disease cases is 268, which is roughly consistent with the reported cases.
Table 6. Cases over time in NL

| % of contact rate ($\beta_i$) | $S_\infty$ | Disease Cases ($N_1-S_\infty$) |
|-------------------------------|------------|-------------------------------|
| 1                             | 109772     | 411770                        |
| 0.8                           | 192707     | 328835                        |
| 0.6                           | 368064     | 153478                        |
| 0.5                           | 520569     | 973                           |
| 0.4                           | 521464     | 78                            |
| 0.2                           | 521506     | 36                            |

![Figure 7](image-url) 

Figure 7. The relationship between the reduction of contact rates and infection cases in NL.

Our model projections suggest that as long as cases are reported in any country, intervention strategies cannot be ignored. If control measures are strengthened, the number of infectious cases will be reduced. Therefore suppression interventions are key to flattening the epidemic curve.

5. Conclusion and discussion. According to the transmission mechanism of COVID-19, we have proposed two kinds of compartment models to study the transmission dynamics of the COVID-19 virus and to explore the potential impact of interventions, to disentangle how transmission is affected in different age groups. We can obtain the basic reproduction numbers and final epidemic sizes in each model, which could be used to estimate the number of infected people during the outbreak. Our model predictions are very consistent with ongoing disease data and the final epidemic size, implying that intervention strategies cannot be ignored. If control measures or population behaviors are relaxed, the second wave of infection may appear in the future [7]. We must point out that, mathematical models cannot predict what will happen exactly, but rather can help us understand what might happen.

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Appendix. The quantities of $B_{11}$, $B_{12}$, $B_{13}$, $B_{14}$, $B_{15}$, $B_{16}$, and $B_{17}$ are as follows.

$$
\sum_{i=1}^{2} B_{1i} = v_i^6 \left[ \alpha_i (\gamma_i v_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11}) \right] + \alpha_i (\gamma_i v_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11} + \gamma_i s_i n_{11}),
$$
\[ B_{12} = \alpha^2 (-\beta_1 n_{11} v_1^6 (q_1^3 + \gamma_1^5) - \beta_1 n_{12} v_1^6 (q_1^3 + \gamma_1^5) + \beta_1 n_{11} v_1^6 (q_1^3 + \gamma_1^5) - \beta_1 n_{12} v_1^6 (q_1^3 + \gamma_1^5)) + \beta_1 n_{11} v_1^6 (q_1^3 + \gamma_1^5) - \beta_1 n_{12} v_1^6 (q_1^3 + \gamma_1^5) - \beta_3 n_{11} v_1^6 (q_1^3 + \gamma_1^5) + \beta_3 n_{12} v_1^6 (q_1^3 + \gamma_1^5), \]

\[ B_{13} = -\alpha_1 \beta_3 (-q_1 n_{11} v_1^6 - q_1 n_{12} v_1^6 + q_1^5 q_1^5 q_1^5 v_1^6 - q_1^5 q_1^5 q_1^5 v_1^6) + \beta_3 (q_1^5 n_{11} + q_1^5 v_1^6), \]

\[ B_{14} = -\alpha_1 \beta_3 q_1^5 v_1^6 - \beta_3 q_1^5 q_1^5 v_1^6 + \beta_3 (q_1^5 q_1^5 q_1^5 v_1^6), \]

\[ B_{20} = -\alpha_1 \beta_3 q_1^5 q_1^5 q_1^5 v_1^6 - \beta_3 q_1^5 q_1^5 q_1^5 v_1^6 + \beta_3 (q_1^5 q_1^5 q_1^5 v_1^6), \]

\[ B_{21} = \alpha_1 \beta_3 q_1^5 q_1^5 q_1^5 v_1^6 + \beta_3 q_1^5 q_1^5 q_1^5 v_1^6 - \beta_3 q_1^5 q_1^5 q_1^5 v_1^6 + \beta_3 (q_1^5 q_1^5 q_1^5 v_1^6). \]

\[ B_{27} = (\beta_1 n_{11}^2 v_1^6 + \beta_2 n_{12}^2 v_1^6 + \beta_3 n_{13}^2 v_1^6 + \beta_4 n_{14}^2 v_1^6) v_1^6 + \beta_3 n_{12} v_1^6. \]

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