COVID-19 Transmission Dynamics and Final Epidemic Size

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COVID-19 Transmission Dynamics and Final Epidemic Size

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Abstract We propose two kinds of compartment models to study the transmission dynamics of COVID-19 virus and to explore the potential impact of the interventions, to disentangle how transmission is affected in different age group. Starting with an SEAIQR model by combining the effect from exposure, asymptomatic and quarantine, then extending the model to an two groups 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, using the data of COVID-19 confirmed cases in Canada and Newfoundland & Labrador province, we can parameterize the models to estimate the basic reproduction number and the final epidemic size of disease transmission.

Keywords SEAIQR · Epidemics · Disease transmission · Dynamics · Lyapunov function · Final size estimates

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1 Introduction

Since the identification of the novel coronavirus (COVID-19) in December 2019, we all know that COVID-19 virus has been sweeping the globe [1–5]. 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.

Mathematical modelling is a powerful tool for understanding disease transmission and exploring different scenarios. Mathematical models based on the principles of viral transmission can play a key role in providing evidence-based information to health policy makers and government departments. At present, many researchers have made rapid responses to the current epidemic [6–8] and established mathematical models about the dynamics and transmission of COVID-19 virus. 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 [9,10], helps to determine the potential and severity of outbreaks [11,12].

There has been growing recognition that, different from SARS, MERS coronavirus [14,15], there is a longer incubation period of COVID-19 virus (14 days in 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 the most effective way to prevent people from getting infected from the virus. Further more, 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 asymptomatic infected population.

One of the common questions regarding an epidemic is its final size [16–19]. The final size of an epidemic can be defined informally as the total number of people experiencing infection [20] over the course of 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 [21] shows that in most of such models, the final size relation involves only a single parameter - the basic reproduction
number. With intervention such as quarantine and reduction of contact are included in the compartmental model, does this claim still hold?

COVID-19 is a new virus and there is limited information regarding 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 virus, we propose two kinds of compartment 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 an one group SEAIQR model to investigate the dynamics of the disease transmission by combining the effect from exposure, asymptomatic and quarantine classes. Due to the fact that, 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, but currently there is nothing that doctors and nurses can do in the way of anti-viral treatments, 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. 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 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, related 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 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 transmission.
This paper is structured as follow. After introduction, in Section 2, we propose an one group SEAIQR model, study the dynamical properties involved in the model with vital dynamics, give the relation of the basic reproduction number with respect to the system parameters, explore the final epidemic size when ignoring the natural birth and death rates and provide the theoretical computation for the final size. With the concern of high risk in elder people, in Section 3, we modified the previous one group model to two groups by separating the population with age below or above 65 years, together with the consideration of 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 systems 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 with 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:

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

(1)

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

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

The description of the parameters and the value ranges are provided in the following table (See Table 1).

| Parameter | Description | Values (per day) | Source |
|-----------|-------------|-----------------|--------|
| \( \Pi \) | Recruitment rate | 136 | [12] |
| \( \mu \) | Natural death rate | 0.0078 | United Nations |
| \( \beta_1 \) | Effective contact rate with \( A \) | 0.491 | [15] |
| \( \beta_2 \) | 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 | [6] |
| \( \alpha_2 \) | Progression rate from \( E \) to \( I \) | 0.156986 | [12] |
| \( \sigma \) | Symptom progression rate from \( A \) to \( I \) | 0.001 | assumed |
| \( q_1 \) | Quarantine rate for \( E \) | 0.1 | [12] |
| \( q_2 \) | Quarantine rate for \( A \) | 0.11 | assumed |
| \( q_3 \) | Quarantine rate for \( I \) | 0.12 | assumed |
| \( \gamma_1 \) | Recovery rate for \( A \) | 0.13978 | [6] |
| \( \gamma_2 \) | Recovery rate for \( I \) | 0.03521 | [12] |
| \( \gamma_3 \) | Recovery rate for \( Q \) | 0.042553 | [12] |
| \( \theta_1 \) | Disease-induced death rate for \( A \) | 0.001 | assumed |
| \( \theta_2 \) | Disease-induced death rate for \( I \) | 0.04227 | [12] |

2.2 Dynamical analysis

First we show the model (1) is well-defined.

**Theorem 1**  
(i) All the solutions of the model (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 possibility of the solution is obvious from [13].

(ii) From (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 \( \mathbb{R}^+_0 \).

We know that the disease-free equilibrium point (DFE) \( M^0 = \left( \frac{\Pi}{\mu}, 0, 0, 0, 0 \right) \) always exists in (1).

In (1), we denote
\[
\begin{align*}
m_1 &= q_1 + \alpha_1 + \alpha_2 + \mu, & m_2 &= q_2 + \sigma + \gamma_1 + \mu + \theta_1, \\
m_3 &= \gamma_2 + q_3 + \mu + \theta_2, & m_4 &= \gamma_3 + \mu.
\end{align*}
\]

Applying the formula in [11], we can calculate the basic reproduction number by \( R_0 = \rho(FV^{-1}) \), where
\[
F = \begin{bmatrix} 0 & \beta_1 & \beta_2 & \beta_3 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} m_1 & 0 & 0 & 0 \\ -\alpha_1 & m_2 & 0 & 0 \\ -\alpha_2 & -\sigma & m_3 & 0 \\ -\gamma_1 & -q_2 & -q_3 & m_4 \end{bmatrix}.
\]

Thus
\[
R_0 = \rho(FV^{-1})
= \beta_1 \alpha_1 m_3 m_4 + \beta_2 m_4 (\sigma_1 + m_2 q_2) + \beta_3 [q_3 (\sigma_2 + m_2 q_2) + m_3 (q_2 m_1 + m_2 q_1)]
= \beta_1 \frac{\sigma_1}{m_1 m_2} + \beta_2 \frac{\sigma_2 + m_2 q_2}{m_1 m_2 m_3} + \beta_3 \frac{q_3 (\sigma_2 + m_2 q_2) + m_3 (q_2 m_1 + m_2 q_1)}{m_1 m_2 m_3 m_4}
= : \mathcal{R}_A + \mathcal{R}_I + \mathcal{R}_Q.
\]

The basic reproduction number obtained in (3) clearly breaks down to 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** The disease-free equilibrium (DFE) of the model (1), given by \( M^0 = \left( \frac{\Pi}{\mu}, 0, 0, 0, 0 \right) \) is globally asymptotically stable (GAS) in \( D \) whenever \( R_0 < 1 \).

Proof Consider the Lyapunov function: \( V(t) = a E(t) + b A(t) + c I(t) + Q(t) \) with the constant coefficients \( a, b, c \):

\[
\begin{align*}
a &= \frac{1}{m_4} \left[ (\beta_1 m_4 m_5 + \beta_2 m_5 + \beta_3 (q_2 m_3 + q_3 \sigma)) \alpha_1 + (\beta_2 m_4 + \beta_3 q_3) \alpha_2 \right] \\
b &= \frac{\beta_1 m_4 m_5 + \beta_2 m_5 + \beta_3 (q_2 m_3 + q_3 \sigma)}{\beta_3 m_3} \\
c &= \frac{\beta_2 m_2 + \beta_3 q_3}{\beta_3 m_3}.
\end{align*}
\]
where \( m_i \) \((i = 1, 2, 3, 4)\) is given in (2). Then the derivative of the function \( V_1(t) \) along the solution of (1) is

\[
\dot{V}_1 = a \left( \frac{\beta_1 A + \beta_2 I + \beta_3 Q}{S + E + A + I + R + Q} S - m_1 E \right) + b (\alpha_1 E - m_2 A) \\
+ c (\alpha_2 E + \sigma A - m_3 I) + (q_1 E + q_3 A + q_3 I - m_4 Q) \\
= a \frac{\beta_1 A + \beta_2 I + \beta_3 Q}{S + E + A + I + R + Q} S - m_4 \beta_2 I - \frac{m_4 \beta_1}{\beta_3} A - m_4 Q \\
\leq a (\beta_1 A + \beta_2 I + \beta_3 Q) - \frac{m_4 \beta_2}{\beta_3} I - \frac{m_4 \beta_1}{\beta_3} A - m_4 Q \\
= (a - \frac{m_4}{\beta_3}) (\beta_1 A + \beta_2 I + \beta_3 Q) \\
= \frac{m_4}{\beta_3} (R_0 - 1) (\beta_1 A + \beta_2 I + \beta_3 Q).
\]

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 \( D \). 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 \( \check{R} \leq (\gamma_1 + \gamma_2 + \gamma_3) \epsilon - \mu R \). Consequently \( R(t) \leq 2(\gamma_1 + \gamma_2 + \gamma_3) \epsilon \to 0 \) as \( t \to \infty \). As we know previously, \( R(t) \geq 0 \). Thus, \( R(t) \to 0 \) as \( t \to \infty \). Similarly, it can be shown that \( S(t) \to \frac{N}{\mu} \) as \( t \to \infty \). Therefore, the DEF \( M^0 \) is globally asymptotically stable in \( D \) when \( R_0 < 1 \).

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

**Theorem 3** Whenever \( R_0 > 1 \) there is unique endemic equilibrium \( M^* = (S^*, E^*, A^*, I^*, Q^*, R^*) \) in the model (1).

**Proof** If the endemic equilibrium point \( M^* \) exists, the right-hand side functions in (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

\[
S^* = \frac{N}{\mu} - \frac{m_1}{\mu} E^*, \quad A^* = \xi_0 E^*, \quad I^* = \xi_1 E^*, \quad Q^* = \xi_2 E^*, \quad R^* = \xi_3 E^*, \quad (4)
\]

with positive constants

\[
\xi_0 = \frac{a_1}{m_2}, \quad \xi_1 = \frac{m_2 a_2 + \sigma a_1}{m_2 m_3}, \quad \xi_2 = \frac{q_3 (\sigma a_1 + m_2 a_2) + m_3 (q_2 a_1 + m_2 q_1)}{m_2 m_3 m_4}, \quad \xi_3 = \frac{m_2 a_2 + \sigma a_1}{m_2 m_3 m_4}.
\]
Finally $\lambda^* S^* = m_1 E^*$ results
\[
\frac{\beta_1 A^* + \beta_2 I^* + \beta_3 Q^*}{S^* + E^* + A^* + I^* + Q^* + R^*} (\frac{m_1}{\mu} E^*) = m_1 E^*,
\]
which determines $E^*$, by (4), as
\[
E^* = \frac{m_1}{\mu} (R_0 - 1) (R_0 - 1) + \xi_0 + \xi_1 + \xi_3 + \xi_2.
\]
Thus, $E^*$ exists whenever $R_0 > 1$.

Followed from (4), we know that the endemic equilibrium point $M^*$ exists and is unique when $R_0 > 1$.

To discuss the stability of the endemic equilibrium point $M^*$, first we can obtain the Jacobian matrix at $M^* = (S^*, E^*, A^*, I^*, Q^*, R^*)$ as follows:
\[
J(M^*) = \begin{bmatrix}
\delta_1 - \mu & \delta_2 & \delta_3 & \delta_4 & \delta_5 & \delta_6 \\
-\delta_1 & -\delta_2 - m_1 & -\delta_3 & -\delta_4 & -\delta_5 & -\delta_6 \\
0 & \alpha_1 & -m_2 & 0 & 0 & 0 \\
0 & \alpha_2 & \sigma & -m_3 & 0 & 0 \\
0 & q_1 & q_2 & q_3 & -m_4 & 0 \\
0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu
\end{bmatrix},
\]
where
\[
\delta_1 = -\lambda(t^*) \frac{N^* - S^*}{N^*}, \quad \delta_2 = \lambda(t^*) \frac{S^*}{N^*}, \quad \delta_3 = -(\beta_1 N^* - \frac{\lambda(t^*)}{N^*}) S^*, \quad \delta_4 = -(\beta_2 N^* - \frac{\lambda(t^*)}{N^*}) S^*,
\]
\[
\delta_5 = -(\beta_3 N^* - \frac{\lambda(t^*)}{N^*}) S^*.
\]

Then the characteristic equation is a $6^{th}$-order polynomial
\[
\lambda^6 + K_1 \lambda^5 + K_2 \lambda^4 + K_3 \lambda^3 + K_4 \lambda^2 + K_5 \lambda + K_6 = 0.
\]

Here we omit the explicit forms of $K_i$ due to the long and tedious relation with respect to the model parameters.

According to the Routh-Hurwitz criterion, with the following hypothesis:
\[
(H_1) K_2 K_1 - K_3 > 0, \quad K_3 (K_2 K_1 - K_3) - K_2 (K_4 K_1 - K_5) > 0, \quad K_3 > 0, \\
(K_4 K_1 - K_3)(K_3 (K_2 K_1 - K_3) - K_2 (K_4 K_1 - K_5)) - (K_2 K_1 - K_3)(K_2 (K_3 K_1 - K_4) - K_2^2 K_1) > 0, \\
(K_3 (K_2 K_1 - K_3) - K_2 K_1)(K_2 K_1 - K_3) > 0, \\
K_2 (K_2 K_1 - K_3) - K_2 K_1 K_3 > 0, \\
K_3 (K_2 K_1 - K_3) - K_2 K_1 K_3 > 0,
\]
the endemic equilibrium point $M^*$ is locally asymptotically stable.
2.3 The final epidemic size without vital dynamics

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

Due to the rapid spreading of COVID-19 virus, we can ignore the host mortality and recruitment in the model (1), that is \( \Pi = 0, \mu = 0 \). Then (1) becomes,

\[
\begin{align*}
\dot{S} &= -\frac{\beta_1 A + \beta_2 I + \beta_3 Q}{N} S, \\
\dot{E} &= \frac{\beta_1 A + \beta_2 I + \beta_3 Q}{N} S - (q_1 + \alpha_1 + \alpha_2) E, \\
\dot{A} &= \alpha_1 E - (q_2 + \sigma + \gamma_1 + \theta_1) A, \\
\dot{I} &= \alpha_2 E + \sigma A - (\gamma_2 + q_3 + \theta_2) I, \\
\dot{Q} &= q_1 E + q_2 A + q_3 I - \gamma_3 Q, \\
\dot{R} &= \gamma_1 A + \gamma_2 I + \gamma_3 Q.
\end{align*}
\] (5)

It is easy to get the same basic reproduction number \( R_0 \) as that in (3) and obviously, \( R_0 \) is decreasing as the reduction of the values of the contact rate \( \beta_i \).

To explore the impact of intervention strategies in model (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

\[
\beta_i(t) = \begin{cases} \\
\beta_i, & t < T, \\
\tilde{\beta}_i, & t \geq T,
\end{cases}
\]

with \( \tilde{\beta}_i < \beta_i \) 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

\[
R_e := R_{eff}(T) = R_0 \frac{S_T}{N_T},
\] (6)

as an effective reproduction number.

To compute the final epidemic size rigorously, from the model (5), we have

\[
\begin{align*}
(S + E)' &= - (q_1 + \alpha_1 + \alpha_2) E, \\
(S + E + A)' &= - (q_1 + \alpha_2) E - (q_2 + \sigma + \gamma_1 + \theta_1) A, \\
(S + E + A + I)' &= - q_1 E - (q_2 + \gamma_1 + \theta_1) A - (\gamma_2 + q_3 + \theta_2) I, \\
(S + E + A + I + Q)' &= - (\gamma_1 + \theta_1) A - (\gamma_2 + \theta_2) I - \gamma_3 Q, \\
(S + E + A + I + Q + R)' &= - \theta_1 A - \theta_2 I.
\end{align*}
\] (7)
Since all the solutions in the model (5) are non-negative and bounded, from (7) we can deduce that, as $t \to \infty$,

$$E \to 0, \ A \to 0, \ I \to 0, \ Q \to 0, \ S(t) \to S(\infty) \geq 0.$$ 

When $t \geq T$, let

$$A_1 = a_1 \gamma_3 q_2 \sigma + q_2 m_3 + \gamma_2 \sigma + \gamma_2 m_3 + \alpha_2 \gamma_3 m_2 (q_1 + \gamma_2) + \gamma_3 m_2 m_3,$$

$$A_2 = a_2 [-\beta_1 (q_2 \gamma_3 + \gamma_2 \gamma_3) + \beta_2 (q_2 \gamma_3 + \gamma_1 \gamma_3) + \beta_3 (q_3 \gamma_3 + \gamma_2 \gamma_2)] + q_1 (-\gamma_1 m_3 - \beta_2 \gamma_2 \sigma + \beta_1 \gamma_1 m_3 + \beta_3 \gamma_2 \sigma),$$

$$A_3 = a_1 [\beta_1 (q_2 \gamma_3 + \gamma_2 \gamma_3) - \beta_2 (q_2 \gamma_3 + \gamma_1 \gamma_3) + \beta_3 (q_2 \gamma_2 - q_3 \gamma_1)] - q_1 m_3 (\gamma_2 \gamma_3 - \beta_3 \gamma_2),$$

$$\beta_4 = a_1 [\gamma_3 (\gamma_1 m_3 - \gamma_2 \sigma) + \gamma_3 (\beta_1 m_3 + \beta_2 \sigma)] + m_2 \gamma_2 (\beta_2 \gamma_3 - \beta_3 \gamma_2),$$

$$A_5 = \beta_1 m_3 m_4 + \beta_2 m_4 (\sigma_1 m_2 + m_2 \sigma_2) + \beta_3 (q_3 \sigma_1 + m_2 \sigma_2) + m_3 (q_2 \sigma_1 + m_2 \sigma_1).$$

After defining the function

$$W_1(t) = A_1 \ln S(t) + \frac{A_2}{N_T} A(t) + \frac{A_3}{N_T} I(t) + \frac{A_4}{N_T} Q(t) + \frac{A_5}{N_T} R(t),$$

we can show that $W_1(t)$ is invariant for $t \geq T$ by calculating the derivatives along the solutions of (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 further assumption that

$$E_\infty = 0, \ A_\infty = 0, \ I_\infty = 0, \ Q_\infty = 0,$$

and from (9) we have

$$A_1 \ln S_T + \frac{A_2}{N_T} A_T + \frac{A_3}{N_T} I_T + \frac{A_4}{N_T} Q_T + \frac{A_5}{N_T} R_T = A_1 \ln S_\infty + \frac{A_5}{N_T} R_\infty.$$  \hspace{1cm} (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 (10) yields

$$A_1 \ln S_T + \frac{A_2}{N_T} A_T + \frac{A_3}{N_T} I_T + \frac{A_4}{N_T} Q_T + \frac{A_5}{N_T} R_T = A_1 \ln S_\infty + \frac{A_5}{N_T} (N_T - S_\infty).$$  \hspace{1cm} (11)

Using (6), we find

$$\ln \frac{S_T}{S_\infty} = \frac{R_0 m_1 m_2 m_3 m_4}{A_1 S_T} (N_T - S_\infty - R_T) - \frac{A_2}{A_1 N_T} A_T - \frac{A_3}{A_1 N_T} I_T - \frac{A_4}{A_1 N_T} Q_T.$$  \hspace{1cm} (12)

(12) provides a final size relation after intervention is taken at time $T$. When $T = 0$, assuming that $S_0 \approx N_0, \ A_0 \approx I_0 \approx Q_0 \approx R_0 \approx 0$, the corresponding standard final size relation is

$$\ln \left( \frac{S_0}{S_\infty} \right) = \frac{R_0 m_1 m_2 m_3 m_4}{A_1} \left( 1 - \frac{S_\infty}{S_0} \right),$$  \hspace{1cm} (13)

which presents a proportion of susceptible class from the starting of disease transmission to the end.
It is interesting to observe that, the formula obtained in (13) is different from most of the results in the literature (see [16–21]). What we can see is that quarantine is involved in our model which is part of 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! In addition, in general, we have \( \frac{m_1 m_2 m_3 m_4}{A_1} > 1 \), implying our final size prediction is less than the one given in the literature.

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 at 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. Strategies focusing specifically on protecting high-risk elderly individuals should be considered in managing the pandemic [22]. In this section, we separate the population into two age groups, one with the age below 65 year old (denote as group-I), and another one with age 65 and over (denote as group-II). To capture the dynamics of infectious virus more accurately, we further classify the infectious individuals into different stages including mild symptoms who needs self-isolation, patient in hospital and patient in intensive care, according to the severity of infection. The architecture of the model is given in Figure 2, there the natural mortality rate is not shown in the figure for simplicity.

Fig. 2 Architecture of the model with two age groups.
Mathematically, the model can be represented by the following system of ordinary equations:

\[
\begin{align*}
\dot{S}_i &= \Pi_i - \lambda(t)S_i - \mu_i S_i, \\
\dot{E}_i &= \lambda(t)S_i - (q_1^i + \alpha_1^i + \alpha_2^i + \mu_i)E_i, \\
\dot{A}_i &= \alpha_1^i E_i - (q_1^i + s_i^a + \gamma_1^i + d_1^i + \mu_i)A_i, \\
\dot{I}_1^i &= \alpha_2^i E_i + s_i^a A_i - (\gamma_1^i + q_1^i + s_i^a + d_1^i + \mu_i)I_1^i, \\
\dot{I}_2^i &= s_1^i I_1^i + \gamma_2^i I_2^i - (\gamma_1^i + s_2^i + d_3^i + \mu_i)I_2^i, \\
\dot{I}_3^i &= s_2^i I_2^i - (\gamma_2^i + d_4^i + \mu_i)I_3^i, \\
\dot{Q}_i &= q_1^i A_i + q_2^i E_i + q_3^i I_1^i - (\gamma_2^i + \mu_i)Q_i, \\
\dot{R}_i &= \gamma_1^i A_i + \gamma_2^i Q_i + \gamma_3^i I_1^i + \gamma_4^i I_2^i - \mu_i R_i,
\end{align*}
\]

where \( i = 1, 2 \), the description of all the variables is given in Table 2, the quarantine-adjusted infection rate function \( \lambda(t) \) has the following form:

\[
\lambda(t) = \frac{1}{N_i + N_2} \sum_{i=1}^{2} (\beta_{11} A_i + \beta_{12} I_1^i + \beta_{13} I_2^i + \beta_{14} I_3^i + \beta_{15} Q_i),
\]

and \( N_i = S_i + E_i + A_i + Q_i + I_1^i + I_2^i + I_3^i + R_i, \) \( (i = 1, 2) \). 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.

### 3.2 Dynamical analysis

First of all, we know that the disease-free equilibrium (DFE) \( M^0 = (M_1^0, M_2^0) \) always exists in the model (14), where \( M_1^0 = (\frac{\Pi_1}{\mu_1}, 0, 0, 0, 0, 0, 0, 0) \), \( M_2^0 = \frac{\Pi_2}{\mu_2}, 0, 0, 0, 0, 0, 0, 0, 0 \). With tedious computation, we can obtain the relation of
Table 3 Parameter estimates (i=1,2)

| Parameter | Description | Values (per day) | Source |
|-----------|-------------|-----------------|--------|
| $\beta_{ij}$ | Effective contact rate in each group | 0.081-0.591 | [15] |
| $\alpha_{ij}$ | Progression rate from $E_i$ to $A_i$ | 0.01880857, 0.02880857 | [6] |
| $\gamma_i$ | Progression rate from $E_i$ to $I_i$ | 0.156986, 0.166086 | [12] |
| $q_{1i}$ | Quarantine rate for $A_i$ | 0.43, 0.1 | [12] |
| $q_{2i}$ | Quarantine rate for $E_i$ | 0.45, 0.12 | assumed |
| $q_{3i}$ | Quarantine rate for $I_i$ | 0.52, 0.13 | assumed |
| $\gamma_{1i}$ | Recovery rate for $A_i$ | 0.13978, 0.12978 | [6] |
| $\gamma_{2i}$ | Recovery rate for $Q_i$ | 0.1, 0.09 | assumed |
| $\gamma_{3i}$ | Recovery rate for $I_i$ | 0.08, 0.06 | assumed |
| $\gamma_{4i}$ | Recovery rate for $I_i^2$ | 0.06, 0.04 | assumed |
| $\gamma_{5i}$ | Recovery rate for $I_i^3$ | 0.04, 0.01 | assumed |
| $\delta_{1i}$ | Symptom progression rate from $A_i$ to $I_i^1$ | 0.05, 0.06 | assumed |
| $\delta_{2i}$ | Symptom progression rate from $I_i^1$ to $I_i^2$ | 0.06, 0.08 | assumed |
| $\delta_{3i}$ | Symptom progression rate from $I_i^2$ to $I_i^3$ | 0.07, 0.09 | assumed |
| $\delta_{4i}$ | Disease-induced death rate for $A_i$ | 0.02, 0.03 | assumed |
| $\delta_{5i}$ | Disease-induced death rate for $I_i^1$ | 0.04227, 0.05227 | [12] |
| $\delta_{6i}$ | Disease-induced death rate for $I_i^2$ | 0.05227, 0.08227 | assumed |
| $\delta_{7i}$ | Disease-induced death rate for $I_i^3$ | 0.06227, 0.1 | assumed |

the basic reproduction number $R_0$ with respect to the parameters, that is,

$$R_0 = \frac{1}{\mu_1 + \mu_2} \sum_{i=1}^{2} \left( \frac{\Pi_i}{\pi_i} \right) \left[ \frac{\beta_{1i} \alpha_{1i} n_{i1} + \beta_{2i} n_{i2} + \beta_{3i} s_{i1} n_{i2} + \beta_{4i} n_{i2} s_{i1}^2 n_{i1}^2 + \beta_{5i} n_{i1}^2 n_{i3}}{\mu_1 v_i^1 v_i^2 v_i^3 v_i^4 n_{i1}^4} \right]$$

$$\times \left[ (\beta_{1i} \alpha_{1i} n_{i1} + \beta_{2i} n_{i2} + \beta_{3i} n_{i2} s_{i1} + \beta_{4i} n_{i2} s_{i1}^2 + \beta_{5i} n_{i1} n_{i3}) v_i^6 + \beta_{15i} n_{i1} n_{i3} \right]$$

$$= \frac{1}{\mu_1 + \mu_2} \sum_{i=1}^{2} \left( \frac{\Pi_i}{\pi_i} R_{i0} \right)$$

with

$$R_{i0} = \frac{\beta_{1i} \alpha_{1i} n_{i1} + \beta_{2i} n_{i2} + \beta_{3i} n_{i2} s_{i1} + \beta_{4i} n_{i2} s_{i1}^2 + \beta_{5i} n_{i1} n_{i3}}{\mu_1 v_i^1 v_i^2 v_i^3 v_i^4}$$

$$n_{i1} = v_i^4 v_i^5 - s_i^2 \gamma_{i1} > 0, \quad n_{i2} = \alpha_{1i} s_i^6 + \alpha_{2i} v_i^1, \quad n_{i3} = \alpha_{1i} (q_i^1 v_i^3 + q_i^3 s_i^1) + v_i^1 (\alpha_{2i} q_i^3 + v_i^3 q_i^2),$$

and

$$v_i^1 = q_i^1 + \alpha_{1i} + \alpha_{2i} + \mu_i, \quad v_i^2 = q_i^1 + s_i^6 + q_i^1 + d_{1i} + \mu_i, \quad v_i^3 = s_i^1 + q_i^1 + \gamma_{i2} + d_{1i} + \mu_i, \quad v_i^4 = \gamma_{i2} + d_{1i} + \mu_i, \quad v_i^5 = \gamma_{i2} + \mu_i.$$  

for $i = 1, 2$. Note that, here $R_0$ in (15) is the basic reproduction number, but $R_{i0}$, $(i = 1, 2)$ in (16) are only notations used in the analysis and computation, not the basic reproduction number with respect to each group.

Similar to the previous model (1), within the closed set

$$D_1 = \left\{ \left( S_1, E_1, A_1, I_1^1, I_1^2, I_1^3, Q_1, R_1, S_2, E_2, A_2, I_2^1, I_2^2, I_2^3, Q_2, R_2 \right) \in \mathbb{R}^{10}_+ : \sum_{i=1}^{2} \left( S_i + E_i + A_i + I_i^1 + I_i^2 + I_i^3 + Q_i + R_i \right) \leq \frac{\Pi_1 + \Pi_2}{\min \{ \mu_1, \mu_2 \}} \right\}$$
we have the following global stability result at $M^0$:

**Theorem 4** The disease-free equilibrium (DFE) $M^0$ in the model (14) is globally asymptotically stable (GAS) in $D_1$ 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

$x_1 = R_{10}$, $x_2 = R_{20}$,

$y_i = \frac{\beta_1 v_i^0 s_i^0 n_i + \beta_2 v_i^0 s_i^0 n_i + \beta_3 s_i^0 v_i^0 v_i^0 + \beta_4 s_i^0 v_i^0 v_i^0 + \beta_5 n_i (q_i^0 v_i^0 + q_i^0 s_i^0)}{v_i^0 v_i^0 n_i}$,

$z_1^1 = \frac{\beta_3 v_i^0 s_i^0 n_i + \beta_3 s_i^0 v_i^0 v_i^0 + \beta_4 s_i^0 v_i^0 v_i^0 + \beta_5 q_i^0 n_i}{v_i^0 v_i^0 n_i}$,

$z_1^2 = \frac{\beta_3 v_i^0 s_i^0 n_i + \beta_4 s_i^0 s_i^0}{n_i}$,

$z_1^3 = \frac{\beta_3 v_i^0 + \beta_4 s_i^0}{n_i}$, $g_i = \frac{\beta_3}{v_i^0}$.

The Lyapunov derivative along the solution of (14) is

$$V_2 = \sum_{i=1}^{2} (x_i(\lambda(t)S_i - v_i^0 E_i) + y_i(\alpha_i^0 E_i - v_i^0 A_i) + z_1^1 (\alpha_i^0 E_i + s_i^0 A_i - v_i^0 I_1^1)$$

$$+ z_1^2 (s_i^1 I_1^1 + v_i^0 I_1^1 - v_i^0 I_2^1) + z_1^3 (s_i^2 I_2^1 - v_i^0 I_3^1) + g_i (q_i^0 A_i + q_i^0 E_i + q_i^0 I_1^1 - v_i^0 Q_i))$$

$$= \sum_{i=1}^{2} x_i \lambda(t) S_i + \sum_{i=1}^{2} \beta_{1i} A_i - \sum_{i=1}^{2} \beta_{2i} I_1^1 - \sum_{i=1}^{2} \beta_{3i} I_2^1 - \sum_{i=1}^{2} \beta_{4i} I_3^1 - \sum_{i=1}^{2} \beta_{5i} Q_i)$$

$$= (x_1 S_1 + x_2 S_2) \lambda(t) + \sum_{i=1}^{2} (\beta_{1i} A_i + \beta_{2i} I_1^1 + \beta_{3i} I_2^1 + \beta_{4i} I_3^1 + \beta_{5i} Q_i)$$

$$- \sum_{i=1}^{2} (\beta_{1i} A_i + \beta_{2i} I_1^1 + \beta_{3i} I_2^1 + \beta_{4i} I_3^1 + \beta_{5i} Q_i)$$

$$= (\frac{S_1}{N_1} + \frac{S_2}{N_2} + R_{10} + \frac{S_2}{N_1 + N_2} - R_{20} - 1) \sum_{i=1}^{2} (\beta_{1i} A_i + \beta_{2i} I_1^1 + \beta_{3i} I_2^1 + \beta_{4i} I_3^1 + \beta_{5i} Q_i)$$

$$< (R_{10} + R_{20} - 1) \sum_{i=1}^{2} (\beta_{1i} A_i + \beta_{2i} I_1^1 + \beta_{3i} I_2^1 + \beta_{4i} I_3^1 + \beta_{5i} Q_i).$$

Therefore, $V_2 \leq 0$ for $R_{10} + R_{20} < 1$ and $V_2 = 0$ if and only if $A_i = I_1^1 = I_2^1 = I_3^1 = Q_i = 0 (i=1,2)$.

Thus $(E(t), A(t), I_1^1(t), I_2^1(t), I_3^1(t), Q_i(t)) \to (0, 0, 0, 0, 0, 0)$ as $t \to \infty$, and consequently $R_i(t) \to 0$ and $S_i(t) \to \frac{Li}{N_i}$ as $t \to \infty$. So the DFE $M^0$ is globally asymptotically stable if $R_{10} + R_{20} < 1$.

**Remark 1** (i) With $I_1^1 = I_2^1$ 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 (15).
(ii) We should mention that the condition $R_{10} + R_{20} < 1$ is strong, at least for the case in (i), comparing with normal condition $R_0 < 1$. It is acceptable due to the complexity of the model. How to construct a Lyapunov function with respect to the basic reproduction number $R_0$, or use other method to obtain weaker condition to ensure the global stability of DFE is not trivial and open to discuss.

Different from one group model (1), beside the DFE $M^0$, there exists one group disease free (or partial disease free) equilibrium in this two group model (14). 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 $P^*_1 = (\frac{1}{n_1}, 0, 0, 0, 0, 0, 0)$ and $P^*_2 = (S^*_2, E^*_2, A^*_2, I^*_2, I^*_{2*}, I^*_{2*}, Q^*_2, R^*_2)$. That is the case where disease transmission only occurs in the group with age 65 or over.

**Theorem 5** 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 model (14).

**Proof** By letting $(E_1, A_1, I^*_1, I^*_{1*}, I^*_{1**}, Q_1, R_1) = (0, 0, 0, 0, 0, 0, 0)$, from the right-hand side functions in the model (14), we know the components in $M^*_1$ must satisfy

\[
S^*_1 = \frac{\Pi_1}{\mu_1}, \quad S^*_2 = \frac{\Pi_2}{\mu_2} - \frac{v^*_2 v^*_3}{\mu_1 n_22} I^*_1, \quad E^*_2 = \frac{v^*_2 v^*_3}{12} I^*_1, \quad A^*_2 = \frac{\alpha_1 v^*_3}{\mu_1 n_22}, \\
I^*_{2*} = \frac{v^*_2 s^*_1}{n_21} I^*_1, \quad I^*_{2*} = \frac{s^*_2 s^*_1}{n_21} I^*_1, \quad Q^*_2 = \frac{n_23}{v^*_2 n_22} I^*_1, \\
R^*_2 = \frac{1}{\mu_2} [\gamma^*_2 \frac{v^*_2 s^*_1}{n_22} + \gamma^*_2 \frac{n_23}{v^*_2 n_22} + \gamma^*_2 \frac{v^*_2 s^*_1}{n_21} I^*_1].
\]

Then $\lambda(t^*) S^*_2 = v^*_2 E^*_2$ results,

\[
S^*_1 + S^*_2 + E^*_2 + A^*_2 + I^*_2 + I^*_{2*} + I^*_{2*} + Q^*_2 + R^*_2 = v^*_2 E^*_2. \tag{18}
\]

Substituting (17) into (18), we obtain

\[
R_{20} \frac{v^*_2 v^*_3}{n_22} I^*_1 S^*_2 = v^*_2 E^*_2 \left( S^*_1 + S^*_2 + E^*_2 + A^*_2 + I^*_2 + I^*_{2*} + I^*_{2*} + Q^*_2 + R^*_2 \right). \tag{19}
\]

Further substituting (17) into (19), we can solve $I^*_1$ explicitly,

\[
I^*_1 = \frac{\frac{\Pi_2}{\mu_2} (R_{20} - 1) - \frac{\Pi_1}{\mu_1}}{\frac{v^*_2 v^*_3}{\mu_1 n_22} (R_{20} - 1) + M_2},
\]

where $M_2 = \frac{v^*_2 v^*_3 (\alpha_1 + v^*_3) + n_23}{v^*_2 n_22} + \frac{s^*_1 (v^*_2 + s^*_3)}{n_21} + 1 + \frac{1}{\mu_2} [\gamma^*_2 \alpha^*_1 v^*_3 n_22 + \gamma^*_2 n_23 + \gamma^*_2 + \gamma^*_2 \frac{v^*_2 s^*_1}{n_21}].$

Therefore, $M^*_1$ exists when $R_{20} \frac{\Pi_1 \mu_2}{\Pi_2 \mu_1} + 1 := \bar{R}_{20}$. 

Remark 2 Mathematically, we can obtain the corresponding condition for the existence of group-II disease free equilibrium $M^* = (Q^*_1, Q^*_2)$ with $Q^*_1 = (S^*_1, E^*_1, A^*_1, I^*_1, I^*_2, Q^*_1, R^*_1)$ and $Q^*_2 = (H_2/\mu_2, 0, 0, 0, 0, 0, 0)$ in a parallel way, which is $R_{10} > \frac{I^*_2\mu_1}{I^*_1\mu_2} + 1 := \tilde{R}_{10}$. Biologically, since $H_1 > H_2$ and $\mu_1 < \mu_2$, so in general $\tilde{R}_{20} > \tilde{R}_{10}$.

Although we can discuss the existence of positive endemic equilibrium theoretically, we neglect here due to the complexity of the model and the lengthy analysis.

3.3 The final epidemic size without vital dynamics

First we notice that with $H_i = \mu_i = 0, i = 1, 2$, the disease-free equilibrium is $M^0 = (\bar{P}_1, \bar{P}_2)$ with $\bar{P}_1 = (S^0, 0, 0, 0, 0, 0, 0)$, where $S^0$ is the initial number of susceptible individuals in each group.

Consequently we can obtain the corresponding basic reproduction number as

$$
R_0 = \frac{1}{s^0_1 + s^0_2} \sum_{i=1}^{2} s^0_i \prod_{i=1}^{2} \left( \beta_{ij} v^n_i n_i + \beta_{ij} n_i n_j \right)
$$

for $R_{10} > \frac{I^*_2\mu_1}{I^*_1\mu_2} + 1 := \tilde{R}_{10}$. Biologically, since $H_1 > H_2$ and $\mu_1 < \mu_2$, so in general $\tilde{R}_{20} > \tilde{R}_{10}$.

which is different from that given in (15) although they have same $R_{40}$ in (16).

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

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

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

$$
= \sum_{i=1}^{2} \left( B_{i1} \ln S_i(t) + \frac{B_{i2}}{N_T} A_i(t) + \frac{B_{i3}}{N_T} I^*_1(t) 
+ \frac{B_{i4}}{N_T} I^*_i(t) + \frac{B_{i5}}{N_T} I^*_i(t) + \frac{B_{i6}}{N_T} Q_i(t) + \frac{B_{i7}}{N_T} R_i(t) \right),
$$

(21)
where the quantities of $B_{i1}$, $B_{i2}$, $B_{i3}$, $B_{i4}$, $B_{i5}$, $B_{i6}$, and $B_{i7}$ are given in Appendix. To show that $W_2(t)$ is invariants for $t \geq T$, we calculate,

\[ W_2'(t) = -\frac{B_{i1}}{N_T} \sum_{i=1}^{2} (\beta_{i1}A_i + \beta_{i2}R_i + \beta_{i3}I_i + \beta_{i4}P_i + \beta_{i5}Q_i) \]

\[ + \frac{B_{i2}}{N_T} (\alpha_i E_i - v_i^3 A_i) + \frac{B_{i3}}{N_T} (\alpha_i E_i + s_i^6 A_i - v_i^3 I_i) \]

\[ + \frac{B_{i4}}{N_T} (s_i^1 I_i + \gamma_i^5 I_i - v_i^4 I_i^2) + \frac{B_{i5}}{N_T} (s_i^2 I_i^2 - v_i^5 I_i^3) \]

\[ + \frac{B_{i6}}{N_T} (q_i^2 E_i + q_i^6 I_i - v_i^6 Q_i) \]

\[ + \frac{B_{i7}}{N_T} (\gamma_i^1 A_i + v_i^8 Q_i + \gamma_i^3 I_i^3 + \gamma_i^4 I_i^4), \]

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

For convenience, we denote $S_i^T = S_i(T)$, $E_i^T = E_i(T)$, $A_i^T = A_i(T)$, $I_i^1T = I_i^1(T)$, $I_i^2T = I_i^2(T)$, $I_i^3T = I_i^3(T)$, $Q_i^T = Q_i(T)$, and $R_i^T = R_i(T)$, and the effective reproduction number $R_e = R_{e\,eff}(T)$ as

\[ R_e = R_0 \frac{S_i^T + S_i^T}{S_T} = \frac{S_i^T + S_i^T}{S_T(S_i^T + S_i^T)} \sum_{i=1}^{2} \sum_{n=1}^{2} \frac{S_i^n B_i^n}{S_i^n S_i^n} \]

\[ = \frac{S_i^T + S_i^T}{S_T S_T} \sum_{i=1}^{2} \sum_{n=1}^{2} \frac{S_i^n R_i^n}{S_i^n S_i^n} := \sum_{i=1}^{2} R_{ei}. \quad (22) \]

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 (21),

\[ \sum_{i=1}^{2} \left( B_{i1} \ln S_i^T + \frac{B_{i2}}{N_T} A_i^T + \frac{B_{i3}}{N_T} I_i^1T + \frac{B_{i4}}{N_T} I_i^2T + \frac{B_{i5}}{N_T} I_i^3T + \frac{B_{i6}}{N_T} Q_i^T + \frac{B_{i7}}{N_T} R_i^T \right) \]

\[ = \sum_{i=1}^{2} \left( B_{i1} \ln S_i(\infty) + \frac{B_{i7}}{N_T} R_i(\infty) \right). \quad (23) \]

Since the total number of individuals at time $T$ is $N_T = N_T^T + N_T^T$, where $N_T^T = S_i^T + E_i^T + A_i^T + I_i^1T + I_i^2T + I_i^3T + Q_i^T + R_i^T$, $i = 1, 2$ is constant, and $(E_i + A_i + I_i^1 + I_i^2 + I_i^3 + Q_i)(\infty) = 0$, we have $N_i^T = S_i(\infty) + R_i(\infty)$. Then, $R_i(\infty) = N_i^T - S_i(\infty)$. Substituting $R_i(\infty)$ into (23) results

\[ \sum_{i=1}^{2} \left( B_{i1} \ln S_i^T + \frac{B_{i2}}{N_T} A_i^T + \frac{B_{i3}}{N_T} I_i^1T + \frac{B_{i4}}{N_T} I_i^2T + \frac{B_{i5}}{N_T} I_i^3T + \frac{B_{i6}}{N_T} Q_i^T + \frac{B_{i7}}{N_T} R_i^T \right) \]

\[ = \sum_{i=1}^{2} \left( B_{i1} \ln S_i(\infty) + \frac{B_{i7}}{N_T}(N_i^T - S_i(\infty)) \right). \quad (24) \]
Using (22), we find
\[ \sum_{i=1}^{2} B_i \ln \left( \frac{S_i^T}{S_i(\infty)} \right) = \sum_{i=1}^{2} \frac{R_{ei}(S_i^0 + S_i^0)}{(S_i^0 + S_i^2)} S_i^0 v_i^1 v_i^2 v_i^3 n_i 1(N_T^T - S_i(\infty) - R_i^T) \]
\[ - \sum_{i=1}^{2} \left( \frac{B_{i2}}{N_T} A_i^T + \frac{B_{i3}}{N_T} I_i^T + \frac{B_{i4}}{N_T} I_i^T + \frac{B_{i5}}{N_T} I_i^T + \frac{B_{i6}}{N_T} Q_i^T \right) \]
which determine the final size relation with respect to the epidemic situation at the intervention time \( T \), effective basic reproduction number and the system parameters.

When \( T = 0 \), assuming that \( S_i^0 \approx N_i^0, E_i^0 \approx A_i^0 \approx I_i^{10} \approx I_i^{20} \approx I_i^{30} \approx Q_i^0 \approx R_i^0 \approx 0 \), the corresponding standard final size relation is
\[ \sum_{i=1}^{2} B_i \ln \left( \frac{S_i^0}{S_i(\infty)} \right) = \frac{1}{N_0} \sum_{i=1}^{2} R_{ei} S_i^0 v_i^1 v_i^2 v_i^3 n_i 1(1 - \frac{S_i(\infty)}{S_i^0}). \]

4 Numerical Simulation

Although we have done some theoretical analysis for the models (1) and (14), the results are still limited because of the complexity of the models. In this section, we implement some numerical simulation to explore more dynamical properties and estimate the final epidemic sizes in Canada and Newfoundland & Labrador.

I. The stability of epidemic equilibrium

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, the system approaches to the same positive equilibrium point \( M^* \) (see Figure 3). We can conjecture that \( M^* \) may be globally asymptotically stable, while the theoretical proof is non-trivial.

**Fig. 3** The stability of the endemic equilibrium \( M^* \) in the model (1). (a) The initial values are \((500, 0, 0, 1, 0, 0)\); (b) The initial values are \((5000, 0, 0, 1, 0, 0)\).
II. The final epidemic size estimation

Through theoretical analysis, we have obtained the final epidemic size relationship of the prognosis. These analytical formulas (12) and (25) 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 was 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 https: //www.canada.ca/en/public-health/services/diseases/2019-novel-coronavirus-infection.html. In a report released by Government of Canada [23] 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 (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 simulations from (1) give \( S_T = 37589888, E_T = 25, A_T = 1, I_T = 14, Q_T = 7, R_T = 56 \), 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 mask, avoid group gathering, etc.) in our model (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 \( S_\infty \), give a total number of disease cases over the course of the epidemic (see Table 4), and draw the relationship between the reduction of contact rate and infection cases in Canada, see Figure 4.

| % of contact rate (\( \beta_i \)) | \( S_\infty \) | Disease Cases (\( N_2 - S_\infty \)) |
|---|---|---|
| 1 | 7911334 | 29678466 |
| 0.8 | 13889287 | 23700713 |
| 0.6 | 26530691 | 11059309 |
| 0.55 | 31820452 | 5769548 |
| 0.5 | 37585587 | 4413 |
| 0.4 | 37589674 | 327 |
| 0.2 | 37589851 | 149 |
Obviously, the reduction of 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 contact rate is a considerably better estimate, comparing with the real data. In our prediction, when the contact rate is reduced about 51.3% or 52%, the associated number of infection cases is 934929 or 1919191 respectively, which is approximately consistent with the prediction in national report: 940000, 1879000 ([23]). In particular, when the contact rate is reduced about 50.71%, the disease cases is 109652, which is roughly consistent with the reported cases in Canada so far. From Figure 4, we can see there is a big jump in the number of cases around a critical percentage of contact rates. The reason for it and how to find the critical value are not known yet.

From Statistics Canada website, we know that the proportion of the population with age 65 and above is about 17.2% in Canada. When apply our two group model (14), 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 (14) is, \((31124516, 0, 0, 0, 0, 0, 0, 0)\) and \((6465480, 0, 0, 0, 0, 0, 0, 0)\) for each group. Taking the intervention time \( T = 30 \) again, we can obtain \( S_1^T = 31124456, E_1^T = 6, A_1^T = 1, I_1^T = 1, I_2^T = 1, Q_1^T = 22, R_1^T = 31, S_2^T = 6465468, E_2^T = 2, A_2^T = 1, I_1^T = 1, I_2^T = 1, I_3^T = 1, Q_2^T = 3, R_2^T = 4 \) by using the parameters in Table 3, and

\[
\begin{align*}
\beta_{11} &= 0.491; \beta_{12} = 0.391; \beta_{13} = 0.32; \beta_{14} = 0.3; \beta_{15} = 0.081; \beta_{21} = 0.591; \\
\beta_{22} &= 0.491; \beta_{23} = 0.49; \beta_{24} = 0.481; \beta_{25} = 0.48; I_1 = 0; \mu_i = 0.
\end{align*}
\]

Here we choose lower recover rate \( \gamma_2 \) and higher \( \beta_2 \) for group-II, based on the factor that elder infected people is more difficult to recover and in Canada, about 81 percent of deaths are linked to long term care facilities, although there is no exact national data released. Then from (15), we have \( R_0 = 1.6679 \).

Applying the final size relation (25), we calculate a final size of \( S_1(\infty) \) and \( S_2(\infty) \), giving a total number of disease cases \( N_0^1 - S_1(\infty) \) 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 5), the corresponding number of infected cases is 943765 and 1844531, respectively, which is approximately consistent with the prediction in [23] and has good agreement with the result obtained from the model (1). The proportion of infected people for each group is \(1 - \frac{S_i(\infty)}{N_0}\).

The prediction 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.

| % of \(\beta_{ij} \) | \(S_1(\infty)\) | \(S_2(\infty)\) | Disease cases (I) \(N_0^1 - S_1(\infty)\) | \(1 - \frac{S_1(\infty)}{N_0^1}\) | Disease cases (II) \(N_0^2 - S_2(\infty)\) | \(1 - \frac{S_2(\infty)}{N_0^2}\) | Total cases |
|-------------------|--------------|--------------|---------------------|---------------------|---------------------|---------------------|----------|
| 1                 | 9811036      | 1091182      | 2131384             | 0.68                | 5374298             | 0.83                | 26687782 |
| 0.8               | 16552490     | 1961078      | 1457290             | 0.47                | 4504402             | 0.7                 | 19076432 |
| 0.6               | 39413213     | 3831763      | 713307              | 0.02                | 2633717             | 0.41                | 3345024  |
| 0.4               | 31124381     | 6965461      | 139                 | 4.5 \times 10^{-6}  | 64                  | 9.9 \times 10^{-6}  | 265      |
| 0.2               | 31124437     | 6965461      | 83                  | 2.7 \times 10^{-6}  | 19                  | 2.9 \times 10^{-6}  | 102      |

**Fig. 5** 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 being 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 with respect to the model (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 (1). Following the final size relation (12), we can estimate the number of infected case, which is shown in Table 6 and Figure 6, 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.

| % of contact rate ($\beta_i$) | $S_\infty$ | Disease Cases ($N_1 - S_\infty$) |
|-------------------------------|------------|----------------------------------|
| 1                             | 109772     | 411770                           |
| 0.8                           | 192707     | 328835                           |
| 0.6                           | 368064     | 183478                           |
| 0.5                           | 520569     | 973                              |
| 0.4                           | 521464     | 78                               |
| 0.2                           | 521506     | 36                               |

Fig. 6 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 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 [24].

We must point out that, mathematical models cannot predict what will happen exactly, but rather can help us understand what might happen. We further mention that, in our proposed models, the recovery rates are considered as constants. This is just ideal case since the recovery of patients in hospital depends on social healthy systems and medical resources. Some research work has been done by the consideration of hospital environment, see [25–27]. Shan and Zhu [25] established a three dimensional SIR model with a nonlinear recovery rate and discussed the possible bifurcations. If we consider the recovery rate in the model (1) depends on the number of hospital beds \( (b) \) and the number of infected cases \( (I) \), for example, choose

\[
\gamma_2 = \gamma_2(b, I) = \tau_0 + (\tau_1 - \tau_0) \frac{b}{I + b},
\]

where \( \tau_0 \) is the minimum per capital recovery rate, and \( \tau_1 \) the maximum per capital recovery rate. Then the advanced model may provide more accurate estimation for public health department to control the spread of infectious diseases. However the analysis has much more involved which is out of the scope in this manuscript. Instead, we provide a comparison in the model with constant and nonlinear recovery rate, numerically.

When taking constant recovery rate, i.e., \( \tau_0 = \tau_1 = 0.03521 \) (or we can treat it as \( b = 0 \)), other parameters are same as those in Table 1, we can find, from the model (1), the number of infected people arrives the peak at \( I_{\text{max}} = 27430 \) after 89 days and gradually declined to 304, which is similar to the reported cases in NL, see Figure 7a. The nonlinear effects of limited health resources may determine the complex dynamics of the system, specifically by selecting different numbers of beds to study the impact of limited health resources on disease transmission. While if we use the nonlinear recovery rate (26) and fix \( \tau_0 = 0.03521 \) and \( \tau_1 = 0.7 \) ([27]), we can discuss the impact of limited health resources on disease transmission by selecting different numbers of hospital beds. If \( b = 3000 < I_{\text{max}} \), we can observe that the number of infected people arrives the peak (5270) after 150 days and gradually drops down to 60. With the increasing of beds number in hospitals, i.e., if \( b = 10000 \), the number of infected cases peaks at 3037 after 150 days and gradually declines to 59. If \( b = 30000 > I_{\text{max}} \), the number of infected people will reach a peak of 2559 after 150 days, and then gradually stabilizes at 58, see Figure 7b. It can be seen from the Figure 7b that when the number of beds increases from 3000 to 30000, the peak value of patients decreases greatly. We notice that, with the chosen parameter values \( \tau_0 = 0.03521 \) and \( \tau_1 = 0.7 \), the basic reproduction number \( R_0 = 3.016 \) is reduced (from 5.2515), comparing with the constant
recovery rate. Moreover, the infectious cases cannot be eliminated although it can be reduced by the increasing of the number of hospital beds.

The numerical simulation indicates that the variation of the hospital beds leads to the change of the number of patients, implying the health resources has a significant impact on the control of COVID-19 virus transmission. Specifically, the increasing of hospital beds can reduces the size of severe infection cases and delay the time of peak. Thus a sufficient number of hospital beds and other medical resources are crucial to control the spread and prevalence of the disease. The findings may help public health agencies allocate health resources rationally to prevent disease outbreaks.

\begin{align*}
\sum_{i=1}^{2} B_i &= v_i^0|\alpha_i (\gamma_i v_i^v n_{11} + \gamma_i^s s_i^s n_{11} + \gamma_i^s s_i^s v_i^v + v_i^v (s_i^v q_i + v_i^v q_i^1)) \\
&\quad + \alpha_i^2(\gamma_i v_i^v n_{11} + \gamma_i^s s_i^s v_i^v) + v_i^v q_i^4 n_{11}),
\end{align*}

\begin{align*}
B_{12} &= \alpha_i^2(-\beta_1 n_{11} v_i^v (q_i^1 + \gamma_i) - \beta_1 \gamma_i^s s_i^s v_i^v + \beta_2 n_{11} v_i^v (\gamma_i^1 + q_i^1) \\
&\quad + \beta_3 v_i^v s_i^s (q_i^1 + \gamma_i^1) - \beta_5 n_{11} (\gamma_i^1 q_i^1 - \gamma_i^1 q_i^4)) - \beta_1 v_i^v n_{11} v_i^v q_i^4 - \beta_2 s_i^s n_{11} v_i^v q_i^4),
\end{align*}

\begin{align*}
B_{13} &= -\alpha_i^2[\beta_1 (\gamma_i^1 n_{11} v_i^v - \gamma_i^s s_i^s v_i^v + \gamma_i^s s_i^s v_i^v - q_i^1 v_i^v v_i^v) + \beta_2 (\gamma_i^1 n_{11} + v_i^v q_i^4 n_{11}) \\
&\quad + \beta_3 v_i^v s_i^s (\gamma_i^1 + q_i^1) + \beta_4 n_{11} s_i^s (\gamma_i^1 + q_i^1) + \beta_5 (\gamma_i^1 q_i^1 n_{11} - \gamma_i^1 q_i^4 n_{11} - \gamma_i^1 q_i^4 n_{11})] \\
&\quad - \beta_2 n_{11} v_i^v v_i^v q_i^4 - \beta_3 s_i^s v_i^v v_i^v q_i^4 - \beta_4 s_i^s v_i^v v_i^v q_i^4 + \beta_5 (\gamma_i^1 q_i^1 n_{11} + \gamma_i^1 q_i^4 n_{11}),
\end{align*}

\begin{align*}
B_{14} &= \alpha_i^2[-\beta_1 n_{11} v_i^v (\gamma_i^1 q_i^1 + v_i^v) + \beta_2 n_{11} v_i^v (\gamma_i^1 q_i^1 + v_i^v) - \beta_3 s_i^s v_i^v v_i^v q_i^4 + \gamma_i^s s_i^s v_i^v v_i^v q_i^4 (\gamma_i^1 + q_i^1)) (\beta_3 v_i^v + \beta_4 s_i^s q_i^4) \\
&\quad \beta_3 v_i^v (\gamma_i^1 q_i^1 + v_i^v) - \alpha_i^2[\beta_2 n_{11} v_i^v (\gamma_i^1 q_i^1 + v_i^v) - \beta_3 s_i^s v_i^v v_i^v q_i^4 + \gamma_i^s s_i^s v_i^v v_i^v q_i^4 (\gamma_i^1 + q_i^1)) (\beta_3 v_i^v + \beta_4 s_i^s q_i^4) \\
&\quad - \beta_5 (\gamma_i^1 q_i^1 + v_i^v)] - \alpha_i^2[-\beta_3 s_i^s v_i^v v_i^v q_i^4 + \gamma_i^s s_i^s v_i^v v_i^v q_i^4 (\gamma_i^1 + q_i^1)) (\beta_3 v_i^v + \beta_4 s_i^s q_i^4) \\
&\quad - \beta_5 (\gamma_i^1 q_i^1 + v_i^v) - \beta_3 s_i^s v_i^v v_i^v q_i^4 - \beta_4 s_i^s v_i^v v_i^v q_i^4 - \beta_5 (\gamma_i^1 q_i^1 + v_i^v),
\end{align*}

Fig. 7 The infectious population with symptoms when initial values are (521541, 0, 0, 1, 0, 0).

**Appendix**

The quantities of $B_{11}$, $B_{12}$, $B_{13}$, $B_{14}$, $B_{15}$, $B_{16}$, and $B_{17}$ are as follows.
\[
B_{15} = -\alpha_1^i \left[ -\beta_2 v_i^4 \gamma_i^7 s_i^7 v_i^6 - \beta_3 v_i^4 \gamma_i^7 s_i^7 (s_i^7 + q_i^7) + v_i^4 (\gamma_i^7 + q_i^7) \right] + \alpha_2 v_i^4 (\gamma_i^7 + q_i^7) + \beta_4 v_i^4 (s_i^7 v_i^4 (\gamma_i^7 + q_i^7) \\
+ v_i^4 (\gamma_i^7 + q_i^7) + \gamma_i^7 s_i^7 v_i^6 - \beta_5 v_i^7 \gamma_i^7 s_i^7 v_i^6 \right] - \alpha_2 v_i^4 (\gamma_i^7 + q_i^7) - \beta_5 v_i^7 \gamma_i^7 s_i^7 v_i^6 \\
+ \beta_3 v_i^7 v_i^4 (\gamma_i^7 + q_i^7) + \beta_4 v_i^4 v_i^3 (\gamma_i^7 + q_i^7) + \gamma_i^7 s_i^7 v_i^6 \\
- \beta_3 v_i^7 v_i^4 v_i^3 \gamma_i^7 v_i^6 - \beta_4 v_i^4 v_i^3 v_i^2 \gamma_i^7 v_i^6 + \beta_5 v_i^7 \gamma_i^7 v_i^6 v_i^6,
\]

\[
B_{16} = \alpha_1^i \left[ \beta_1 v_i^7 v_i^7 n_i + \beta_2 s_i^7 v_i^7 n_i + \beta_3 s_i^7 s_i^7 v_i^7 v_i^6 + \beta_4 s_i^7 s_i^7 s_i^7 v_i^7 v_i^6 - \beta_5 (s_i^7 v_i^7 n_i + s_i^7 n_i + v_i^7 n_i) \\
+ \gamma_i^7 s_i^7 v_i^7 v_i^6 \right] + \alpha_2 v_i^4 (\gamma_i^7 + q_i^7) - \beta_2 v_i^7 v_i^6 + \beta_3 s_i^7 v_i^7 v_i^6 - \beta_4 s_i^7 s_i^7 v_i^7 v_i^6 - \beta_5 (s_i^7 v_i^7 n_i + s_i^7 n_i + v_i^7 n_i),
\]

\[
B_{17} = \beta_1 v_i^7 v_i^7 n_i + \beta_2 n_i + \beta_3 n_i + \beta_4 n_i + \beta_5 n_i.
\]

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**Figures**

![Flow diagram of the model for studying COVID-19 transmission.](image1)

**Figure 1**

Flow diagram of the model for studying COVID-19 transmission.

![Architecture of the model with two age groups](image2)

**Figure 2**

Architecture of the model with two age groups

![The stability of the endemic equilibrium \( M^\ast \) in the model (1). (a) The initial values are \( (500, 0, 0, 1, 0, 0) \); (b) The initial values are \( (5000, 0, 0, 1, 0, 0) \).](image3)

**Figure 3**

The stability of the endemic equilibrium \( M^\ast \) in the model (1). (a) The initial values are \( (500, 0, 0, 1, 0, 0) \); (b) The initial values are \( (5000, 0, 0, 1, 0, 0) \).
Figure 4

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

Figure 5

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

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

Figure 7

The infectious population with symptoms when initial values are (521541, 0, 0, 1, 0, 0).