

# Network information analysis reveals risk perception transmission in a behaviour-influenza dynamics system

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## SUMMARY

Influenza poses a significant public health burden worldwide. Understanding how and to what extent people would change their behaviour in response to influenza outbreaks is critical for formulating public health policies. We incorporated the information-theoretic framework into a behaviour-influenza (BI) transmission dynamics system in order to understand the effects of individual behavioural change on influenza epidemics. We showed that information transmission of risk perception played a crucial role in the spread of health-seeking behaviour throughout influenza epidemics. Here a network BI model provides a new approach for understanding the risk perception spread and human behavioural change during disease outbreaks. Our study allows simultaneous consideration of epidemiological, psychological, and social factors as predictors of individual perception rates in behaviour-disease transmission systems. We suggest that a monitoring system with precise information on risk perception should be constructed to effectively promote health behaviours in preparation for emerging disease outbreaks.

**Key words:** Epidemiology, human behaviour, influenza, information theory, modelling, risk perception.

## INTRODUCTION

It is well recognized that influenza poses a significant public health burden worldwide. Generally, non-regulatory approaches to changing behaviours against influenza across individuals and populations have focused on using information-based interventions to persuade people of the infection risks they face. The potential benefits of behavioural change may result from clinics or public health campaigns such as those aimed at keeping one's distance from infected

individuals, wearing protective masks, and reducing time spent in crowded environments [1].

Understanding how and to what extent people would change their behaviour in response to an epidemic is critical for formulating public health policies to control the spread of infectious diseases [2]. Chen *et al.* [3] found that individuals who were infected at a lower rate were more likely to engage in self-protective behaviour compared to those with a higher rate of infection. Recent researches have indicated that the risk perception of the disease, the level of anxiety regarding the disease, the perceived efficacy of self-protective measures, and other factors are positively associated with people's willingness to engage in self-protective behaviour during influenza virus outbreaks [4]. Generally, risk perception can be referred to as an awareness or belief about the potential hazard and/or

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harm [5]. Risk perception may be affected by factors such as perception of a hazard, cultural and social factors or the experience or memory of a prior similar hazard, all of which may result in variation in risk perception among individuals [5]. In a behaviour-disease system, risk perception can be described by the likelihood and severity of the infectious disease [4, 6].

Previous researches have linked transmission dynamics with behavioural modelling to investigate behavioural responses to epidemics [7–12]. More recently, Wells *et al.* [13] incorporated behaviour such as perceived vaccine risks and social influences into a seasonal influenza transmission model to examine decision-making regarding vaccine uptake. Many studies have also highlighted that the social network structure could enable new health behavioural interventions to critically affect the interactions between risk perception and contagious disease transmission [4, 9, 14–16]. Despite the importance of understanding the impact of risk perception on a pandemic [1, 17], it is difficult to predict empirically the spread of health behaviour for reducing susceptibility.

Funk *et al.* [7, 8] linked a mathematical model describing the spread of risk perception in a host population and the epidemiological susceptible-infected-recovered (SIR) model for understanding the perception effects on behavioural change and susceptibility reduction. They treated the spread of risk perception or behaviour as a simple contagious disease, implying that a single contact with an infected individual is usually sufficient to transmit the risk perception or behaviour. Funk *et al.* [7] also pointed out that interaction within a disease network structure can induce health behavioural change in individuals which can feed back to alter the disease dynamics.

Generally, perception variance (noise) can be adequately described as the stochastic individual-individual variability. Experimentally, perception noise can be observed by sampling the distribution of responses by a group of infected individuals exposed to the same input sources of information regarding the accurate knowledge of influenza. Moreover, if the distribution of responses elicited by a less accurate perception overlaps with the distribution elicited by a more accurate perception, an individual whose response value falls within the overlap will not be able to distinguish with absolute certainty which perception was presented. This inability to resolve distinct risk perceptions represents a loss of information about the knowledge of influenza. In order to quantify the degree to which perception noise affects the

accurate knowledge of influenza, or specifically to determine what a behaviour-influenza (BI) transmission system can or cannot communicate accurately; it is useful to turn to information theory.

Information theory provides a mathematical framework to quantify the amount of information that can be transmitted through a noisy communication channel and thus any complex system can be reduced to a black box communication channel and analysed. The strength of this type of analysis is that only input and output measurements are required. Some studies have provided examples of disease transmission systems that have benefited from such an analysis [18].

Recently, epidemiological studies were conducted on a large scale in genetics, the network structure, and cell function by using information-theoretic methods [19]. Such ideas may provide effective means for the field of epidemiology for social network structure assessment. To discriminate the epidemic heterogeneity and network structure on the spatio-temporal pattern of the epidemic process, Colizza *et al.* [20] introduced a characterization of the epidemic pattern by using entropy. Quax *et al.* [21] inferred the structure of the sexual network by the likelihood cluster-size distribution.

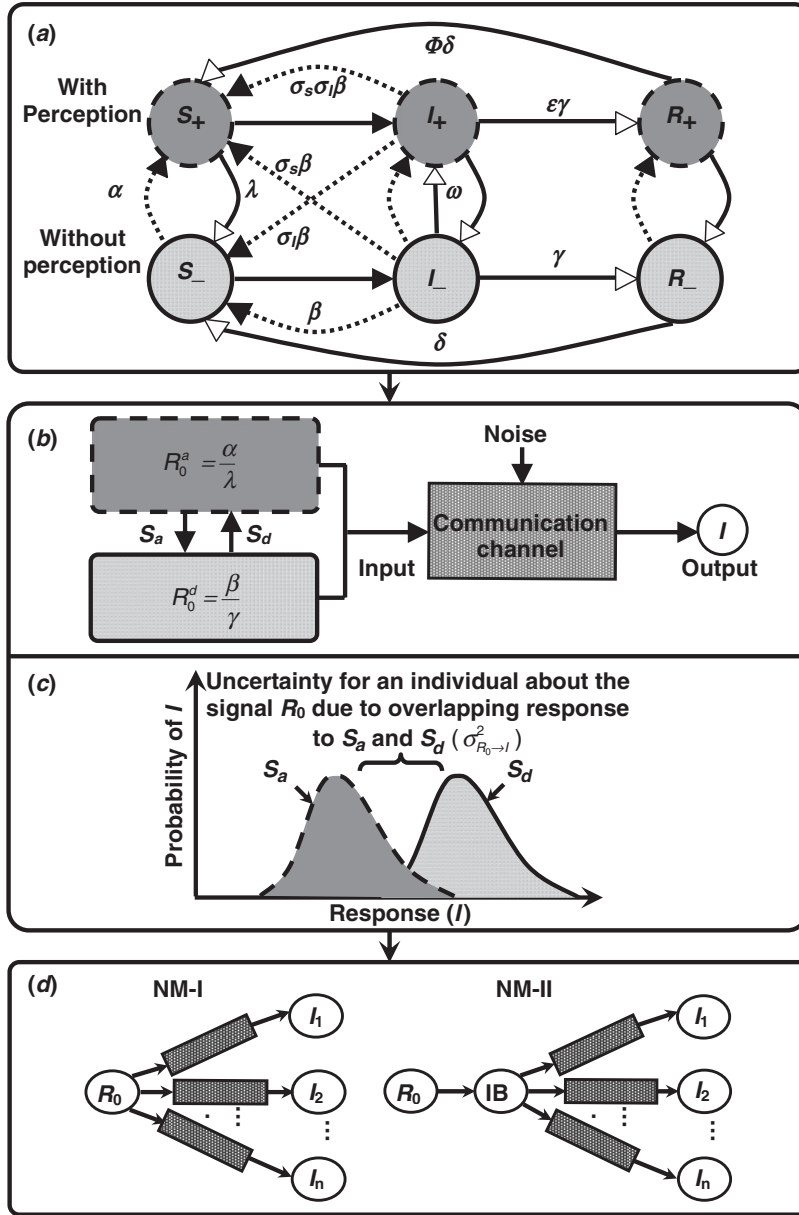
The purpose of this study is to apply information theory to a BI dynamics system for assessing risk perception transmission. In this study, we incorporated a classic SIR model into an information-theoretic framework to assess the spread of information flow of risk perception concerning influenza and the consequent behavioural changes in a single population and a network system. We provided theoretical and numerical analyses of various scenarios involved in a BI transmission model that was also implemented in a network-based information scheme.

## MATERIALS AND METHODS

### BI model

We developed a BI model by incorporating a previously established SIR-based perception model [8] into an information-theoretic framework to simulate the information flow of risk perception in response to an influenza outbreak (Fig. 1*a, b*). The mathematical descriptions of the transmission dynamics for the SIR-based perception model are listed in Table 1.

Briefly, the BI model uses six compartments to represent disease states with and without perception [8]. The model divided the population into two groups,



**Fig. 1.** Schematic representation of proposed behaviour-influenza (BI) transmission dynamics systems. (a) SIR-based perception model by Funk *et al.* [8]. (b) Information flow of  $R_0$  signals through the communication channel to link some output  $I$ . (c) Uncertainty for population about input signal  $R_0$ s due to response  $I$ . (d) Network BI models.

i.e. those with and those without perception, so that people with perception are assumed to become aware of information about the infectious disease as such information emerges in the population. Hence, people tend to assess the individual costs and benefits of behavioural change which leads to action (e.g. mask wearing, vaccination, etc.).

The number of individuals with perception would either be increased or decreased by perception spread and perception loss, respectively. The susceptible population would increase due to loss of immunity

and also decrease following infection, acquired by contact between susceptible and infected individuals, with and without perception. The infected population would increase by successful infection of susceptible individuals and decrease by recovery. The recovered population would increase and decrease by recovery and loss of immunity, respectively. The symbols, meanings, and values used in the BI model are given in Table 2.

Here we identified estimates of the basic reproduction number ( $R_0$ ), with and without perception,

Table 1. *Equations for the SIR-based perception model by Funk et al. [8]\**

Dynamic equations†	
$\frac{dS_+}{dt} = \left(\frac{S_-}{N}(S_+ + I_+ + R_+)\right)\alpha + (R_+)\varphi\delta - \left(\frac{S_+}{N}I_+\right)\sigma_S\sigma_I\beta + \left(\frac{S_+}{N}I_-\right)\sigma_S\beta - (S_+)\lambda$	(T1)
$\frac{dI_+}{dt} = \left(\frac{I_-}{N}(S_+ + I_+ + R_+)\right)\alpha + \left(\frac{S_+}{N}I_+\right)\sigma_S\sigma_I\beta + \left(\frac{S_+}{N}I_-\right)\sigma_S\beta + (I_-)\omega - (I_+)\varepsilon\gamma - (I_+)\lambda$	(T2)
$\frac{dR_+}{dt} = \left(\frac{R_-}{N}(S_+ + I_+ + R_+)\right)\alpha + (I_+)\varepsilon\gamma - (R_+)\varphi\delta - (R_+)\lambda$	(T3)
$\frac{dS_-}{dt} = (S_+)\lambda + (R_-)\delta - \left(\frac{S_-}{N}(S_+ + I_+ + R_+)\right)\alpha - \left(\frac{S_-}{N}I_-\right)\beta - \left(\frac{S_-}{N}I_+\right)\sigma_I\beta$	(T4)
$\frac{dI_-}{dt} = \left(\frac{S_-}{N}I_-\right)\beta + \left(\frac{S_-}{N}I_+\right)\sigma_I\beta + (I_+)\lambda - \left(\frac{I_-}{N}(S_+ + I_+ + R_+)\right)\alpha - (I_-)\omega - (I_-)\gamma$	(T5)
$\frac{dR_-}{dt} = (I_-)\gamma + (R_+)\lambda - \left(\frac{R_-}{N}(S_+ + I_+ + R_+)\right)\alpha - (R_-)\delta$	(T6)

\* See Figure 1a.

† See Table 2 for explanation of symbols.

based on the present BI model.  $R_0$  can be used to quantify disease infection severity, defined as the average number of secondary cases produced by an infected individual in a totally susceptible population [22]. Therefore, this study used two parameters,  $R_0^a$  and  $R_0^d$ , to describe populations with and without perception, respectively, during an influenza epidemic. Based on the BI models, there are two  $R_0$ s, i.e.  $R_0^a = \alpha/\lambda$  for the with-perception state and  $R_0^d = \beta/\gamma$  for the without-perception state, where  $\alpha$  is the rate of perception spread,  $\lambda$  is the rate of perception loss,  $\beta$  is the infection rate describing contact between infected and susceptible populations, and  $\gamma$  is the recovery rate from infected to recovered populations.

### Information flow of risk perception

In the information theory framework, we used two types of information transfer and exchange on various perception information processing within the BI scheme: (i)  $S_a \equiv R_0^a$  and (ii)  $S_d \equiv R_0^d$ , representing the  $R_0$  signals with and without perceptual states, respectively (Fig. 1b). We used the  $R_0$ 's signal as a proxy input source of risk perception information about an influenza outbreak and the infected fraction of the population ( $I$ ) as an output response to capture the infection level in the total population.

Therefore, a communication channel can be used to link an input source of information  $S_a$  or  $S_d$  to an output  $I$  (Fig. 1b). However, the signal processing may cause noise that includes noisy and incomplete surveillance data from input signal  $R_0$ s, leading to an overlap

of possible output response  $I$  (Fig. 1b, c). Thus, the uncertainty for an individual to acquire an accurate perception of influenza knowledge can be induced from the overlapping response to  $S_a$  and  $S_d$  (Fig. 1c).

### Network BI models

In view of the BI model (Fig. 1a, b), the mutual risk perception information (MI) between  $R_0$  and  $I$ , i.e.  $MI(I; R_0)$  can be expressed mathematically as the binary logarithm of the maximum number of input signal values ( $R_0$ ), whereas a signalling system can resolve in the presence of its noisy output response ( $I$ ) [23],

$$\begin{aligned}
 MI(I; R_0) &= \sum_{I, R_0} P(I, R_0) \log_2 \frac{P(I, R_0)}{P(I)P(R_0)} \\
 &= - \sum_I P(I) \log_2 P(I) \\
 &\quad - \left[ - \sum_{I, R_0} P(I, R_0) \log_2 P(I|R_0) \right] \\
 &= H(I) - H(I|R_0), \tag{1}
 \end{aligned}$$

where  $P(I, R_0)$  is a joint probability function determining the marginal probability functions  $P(I)$  and  $P(R_0)$  which can be expressed as  $P(I, R_0) = P(R_0) \times P(I|R_0)$ , in that  $P(I|R_0)$  is a conditional response distribution,  $H(I)$  is the Shannon entropy of a random variable  $I$  with a probability mass function  $P(I)$  measured in bits,  $H(I|R_0)$  is the conditional entropy for a conditional response probability  $P(I|R_0)$ , and  $\log_2$  is a binary logarithm, representing an individual may have a two-state

Table 2. Values and descriptions of input parameters used in the SIR-based perception model

Symbol	Description	Value
$S_+$	Susceptible with perception	0*
$I_+$	Infected with perception	0*
$R_+$	Recovered with perception	0*
$S_-$	Susceptible without perception	99*
$I_-$	Infected without perception	1*
$R_-$	Recovered without perception	0*
$N$	Total population	100*
$\alpha$	Perception spread rate	0.1–3*
$\Phi$	People who perceive the presence of the disease	1†
$\delta$	Immunity loss rate of recovered without perception	0.5†
$\sigma_I$	Reduced infectivity factor from infected individuals with perception to susceptible individuals without perception	0.5†
$\sigma_S$	Reduced susceptibility factor from infected individuals without perception to susceptible individuals with perception	0.5*
$\beta$	Infection rate from infected to susceptible state	1–3.5*
$\lambda$	Perception loss rate	1†
$\omega$	Rate of infected individuals becoming perceptive	1†
$\varepsilon\gamma$	Reduction in the duration of infected individuals with perception	2†
$\gamma$	Recovery rate of infected without perception	0.1†

\* Assumed in this study.

† Adopted from Funk *et al.* [8].

context of with- and without-perception information in the social contagion during an epidemic.

In particular,  $H(I)$  measures inherent uncertainty rather than how different the outcomes are, whereas  $MI(I; R_0)$  measures the reduction in the entropic uncertainty of  $I$  due to the knowledge of  $R_0$ , regardless of how their outcomes may correlate [23]. The  $R_0$  signal distribution,  $P(R_0)$ , reflects setting-specific influenza transmission potentials at which an individual perceives different  $R_0$  values.

The disease transmission is typically processed by disease networks comprising multiple channels. However, there is noise in perception acquisition, although the source of the noise is unclear. This information bottleneck (IB) might be anywhere: in the information from perception, in the ability to accumulate the incoming information over time or in an imperfect strategy that might incorrectly estimate the importance of an accurate knowledge of an epidemic.

Here we used network information theory known as the multiple access channel (MAC) model [23] to describe the effects of a disease network structure on transmission for information flow of risk perception. We considered two simple network BI models, (i) without IB: NM-I and (ii) with IB: NM-II, for transmitting a signal  $R_0$  through multiple channels to the responses  $I_1, I_2, \dots, I_n$ , under the assumption of Gaussian distribution (Fig. 1d).

The maximum MI resulting from NM-I can be calculated as [23],

$$MI_I(I_1, \dots, I_n; R_0) = \frac{1}{2} \log_2 \left( 1 + n \frac{\sigma_{R_0}^2}{\sigma_{R_0 \rightarrow I}^2} \right), \quad (2)$$

where  $n$  is the number of contacts of infectious individuals,  $\sigma_{R_0}^2$  is the variance of the  $R_0$  signal distribution, and  $\sigma_{R_0 \rightarrow I}^2$  is the variance introduced in each access channel. The ratio  $\sigma_{R_0}^2 / \sigma_{R_0 \rightarrow I}^2$  is the signal-to-noise ratio (SNR) [23].

On the other hand, the maximum MI resulting from NM-II is [23],

$$MI_{II}(I_1, \dots, I_n; R_0) = \frac{1}{2} \log_2 \left( 1 + \frac{n \left( \frac{\sigma_{R_0}^2}{\sigma_{IB \rightarrow I}^2} \right)}{1 + n \left( \frac{\sigma_{R_0 \rightarrow IB}^2}{\sigma_{IB \rightarrow I}^2} \right)} \right), \quad (3)$$

where  $\sigma_{R_0 \rightarrow IB}^2$  is the variance introduced to the IB and  $\sigma_{IB \rightarrow I}^2$  is the variance introduced in each access channel through the IB to response  $I$ .

### Variance in NM-I and NM-II

Previous studies providing valuable data related to the viral titre over time for the <18 and  $\geq 18$  years age groups on experimental human influenza data (Supplementary Tables S1 and S2), allowed us to calculate  $\sigma_{R_0}^2$ . Moreover, to assess the variance of the  $R_0$ 's

signal distribution  $\sigma_{R_0}^2$ , we utilized a previous study [24] to identify the estimates of the viral titre and  $I$ . We used a four-parameter Hill-based dose–response model to construct the relationship between  $R_0$  and viral titre,

$$R_0(V) = R_{0\min} + \frac{(R_{0\max} - R_{0\min})}{1 + (V/R_0 V_{50})^{n_H}}, \quad (4)$$

where  $V$  is the viral titre (log TCID<sub>50</sub>/ml),  $R_{0\min}$  and  $R_{0\max}$  are the minimum and maximum  $R_0$ ,  $R_0 V_{50}$  is the viral titre at 50%  $R_0$ , and  $n_H$  is the fitted Hill coefficient.

Theoretically, during the epidemic  $I$  is seen to depend only on  $R_0$  in a homogeneous and unstructured population [22],

$$I = 1 - \exp(-R_0 I). \quad (5)$$

We solved equation (5) numerically by using a non-linear regression model to best fit the profile describing the relationship between  $I$  and  $R_0$  for values of  $R_0$  ranging from 1–5 as:  $I(R_0) = 1 - \exp(1.63 - 1.66R_0)$  ( $r^2 = 0.99$ ,  $P < 0.05$ ). Finally,  $R_0$  can be estimated as a function of  $I$  only

$$R_0 = \frac{(\ln(1 - I) - 1.63)}{-1.66}. \quad (6)$$

To explore the effects of the disease network on risk perception, we used a correlation coefficient ( $\rho$ ) to associate  $R_0$  and  $I$  from the published data (see Supplementary material).  $\rho$  can be used to associate the amount of observed variability which can attribute variance to the overall biological variability and experimental noise to assess the estimates of MI affected by experimental noise. On the other hand, the overlapping percentage ( $I_O$ ) (Fig. 1c) can be used to calculate  $\sigma_{R_0 \rightarrow I}^2$ . Thus, the information-theoretic theorem with known values of  $\rho$ ,  $I_O$ , and  $\sigma_{R_0}^2$ ,  $\sigma_{R_0 \rightarrow I}^2$  can be computed as:  $(1 - \rho^2)\sigma_{R_0}^2 I_O$  [23].

To determine the variance in NM-II, we adopted the concept of influenza viral reassortment as an information exchange between viral segments [25]. First, we adopted an adaptive mutation model [26] to account for the effect of an IB on the  $R_0$ 's signal transmission in NM-II. André & Day [26] indicated that the adaptive mutation might occur by chance within a secondary infection due to a transmission bottleneck in a transmission chain of the emergence of an infectious disease.

Thus, we analogized the mutation pathogen changes to the next state as the transmission of  $R_0$ 's signal through a communication channel due to an

IB ( $R_0, R_0 \rightarrow \text{IB}$ ) which has the form [26],

$$R_{0, R_0 \rightarrow \text{IB}} = \frac{1}{1 - p \left( \frac{1 - R_0}{\mu R_0 + \mu L} \right)}, \quad (7)$$

where  $P$  is the probability of an initial state that does not change to the next stage,  $\mu$  is the probability of the chance that the mutation pathogen in the adaptive host changes to the next state, and  $L$  is the expected duration of infections caused by influenza. Thus, the variance between  $R_0$  and IB ( $\sigma_{R_0 \rightarrow \text{IB}}^2$ ) can be calculated.

On the other hand, to study the  $R_0$ 's signal transmission between IB and response  $I$ , we adopted the concept in the SIR-based pathogen genetic diversity model [27]. Thus, the information flow of risk perception through the IB to response  $I$  can be expressed as

$$R_{0, \text{IB} \rightarrow I} = \frac{b}{e + b} \left( 1 - \frac{1}{R_{0, R_0 \rightarrow \text{IB}}} \right), \quad (8)$$

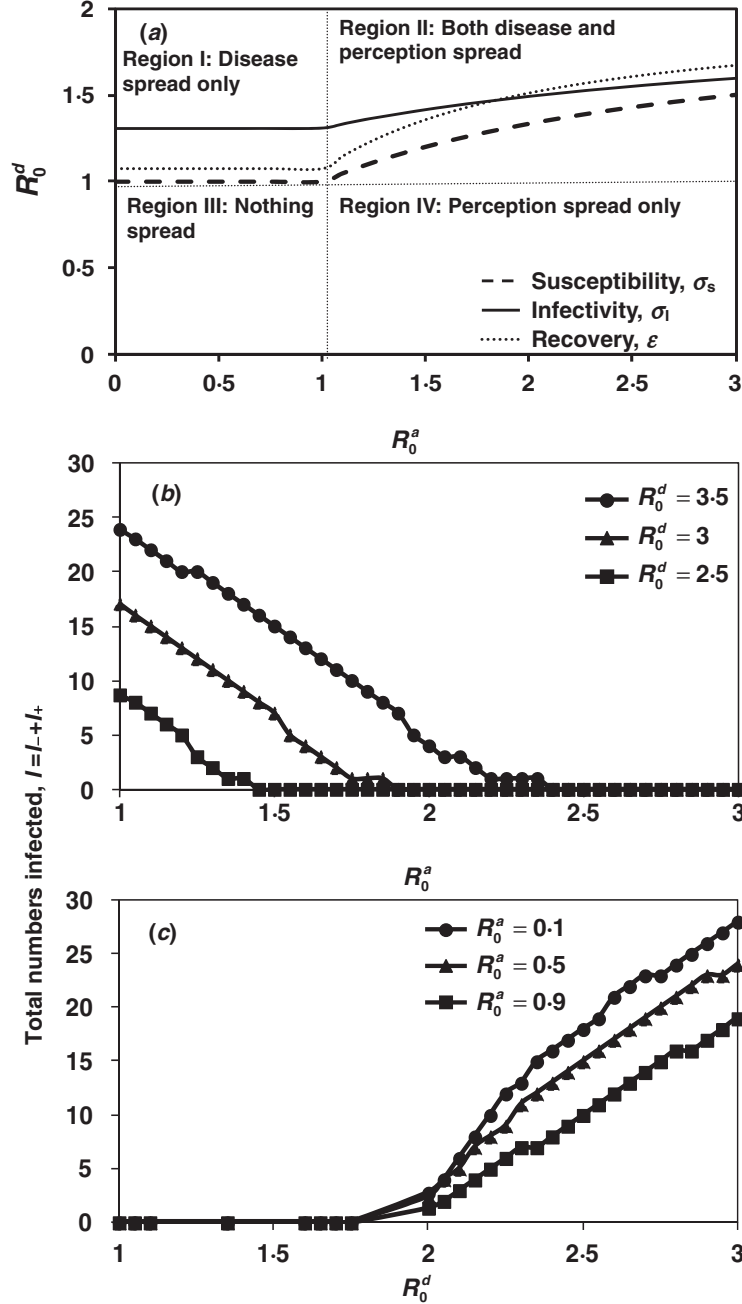
where  $e$  is the probability of an infected individual moving to the recovered state and  $b$  is the probability of any host moving to the susceptible state. The variance between IB and response  $I$  ( $\sigma_{\text{IB} \rightarrow I}^2$ ) can then be calculated by equation (8).

### Behavioural change modelling

To explore the effects of health behavioural change on BI transmission dynamics, three actions representing health behaviours as a consequence of protective behaviour adopted in a state of greater alert (e.g. awareness of carrying the disease) were adopted in an epidemic equilibrium structure [8]. (i) Reduced susceptibility: only susceptible individuals have their susceptibility reduced by a factor  $\sigma_S$ , while the other rates remain unaffected by perceiving an accurate knowledge of influenza; (ii) reduced infectivity: only infected individuals have their infectivity reduced by a factor  $\sigma_I$ , while the other rates remain unaffected by perceiving an accurate knowledge of influenza; and (iii) faster recovery: only the recovery rate depends on the perception of an accurate knowledge of influenza such that  $\varepsilon > 1$ , while all other parameters are perception-independent.

Thus, the equilibrium signal from the population without perception  $R_{0e}^d$  with a reduced susceptibility factor  $\sigma_S$  can be expressed as [8],

$$R_{0e}^d = 1 + \frac{(1 - \sigma_S)(R_{0e}^a - 1)}{1 + \sigma_S(R_{0e}^a - 1)}. \quad (9)$$



**Fig. 2.** Behaviour-influenza model simulation. (a) Four different schemes at equilibrium. (b) Total numbers infected for spread of disease only. (c) Total numbers infected for both disease and perception spread.

The equilibrium signal from a population without perception  $R_{0e}^d$  with a reduced infectivity factor  $\sigma_I$  is [8],

$$R_{0e}^d = 1 + \frac{(1 - \sigma_I) \left[ R_{0e}^a \left( 1 + \frac{\omega}{\alpha + \gamma} \right) - 1 \right]}{1 + \sigma_I \left[ R_{0e}^a \left( 1 + \frac{\omega}{\alpha + \gamma} \right) - 1 \right]}. \quad (10)$$

The equilibrium signal from a population without perception  $R_{0e}^d$  with a faster recovery factor  $\varepsilon$  has

the form [8],

$$R_{0e}^d = 1 + \frac{R_{0e}^a - 1 + \frac{\omega}{\alpha + \gamma + \omega}}{R_{0e}^a + \frac{(\varepsilon - 1)\gamma}{\alpha + \gamma + \omega}} (\varepsilon - 1). \quad (11)$$

The MI may be affected by the health behavioural change as a negative feedback. Therefore, we estimated the variance of  $R_{0e}^d$  varied with behavioural

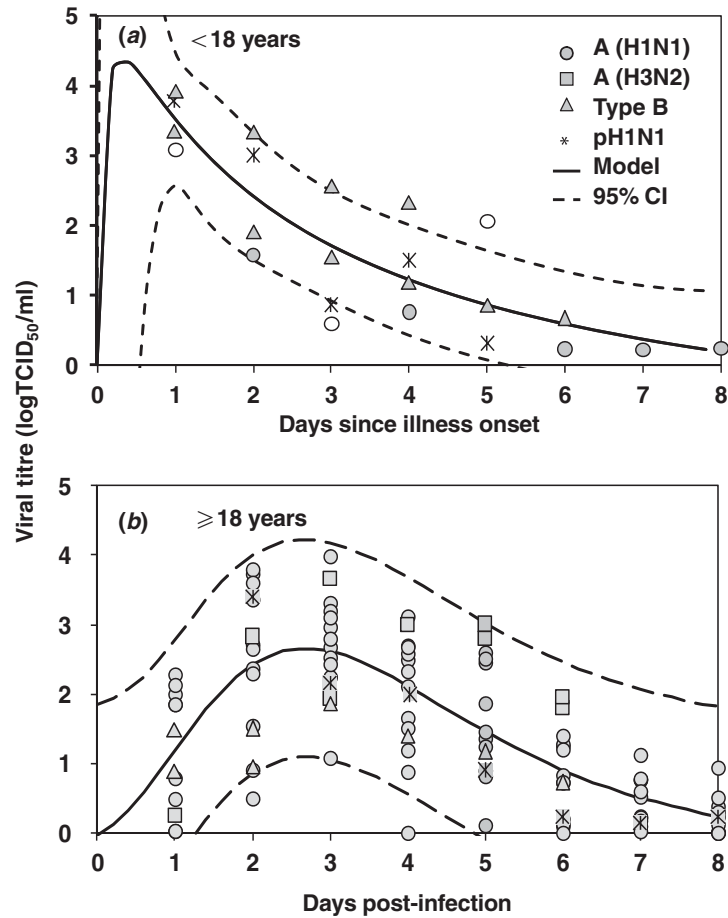


Fig. 3. Time-dependent viral titre concentrations: (a) <18 years age group, (b)  $\geq 18$  years age group, respectively.

factors to assess the MI caused by behavioural change ( $MI_b$ ) in NM-I and NM-II. We used the MI change ratio ( $MI_c$ ) defined as  $MI_c = (MI_b - MI) / MI$  to assess behavioural change effects on the network BI systems.

## RESULTS

### BI model simulation

Here we divided the equilibrium structures of the BI model into four different regions during an epidemic. (i) Region I: disease spread only; (ii) region II: both disease and perception spread; (iii) region III: nothing spread; and (iv) region IV: perception spread only (Fig. 2a).

Our results showed that the total numbers infected for disease spread only were estimated to be 0–25 given that  $R_0^d$  and  $R_0^a$  change from 2.5 to 3.5 and 1 to 3, respectively (Fig. 2b). Whereas, the total numbers infected for both disease and perception spreads were 0–35 given  $R_0^a$  and  $R_0^d$  changing from 0.1 to 0.9 and 1 to 3, respectively (Fig. 2c). Our results indicated

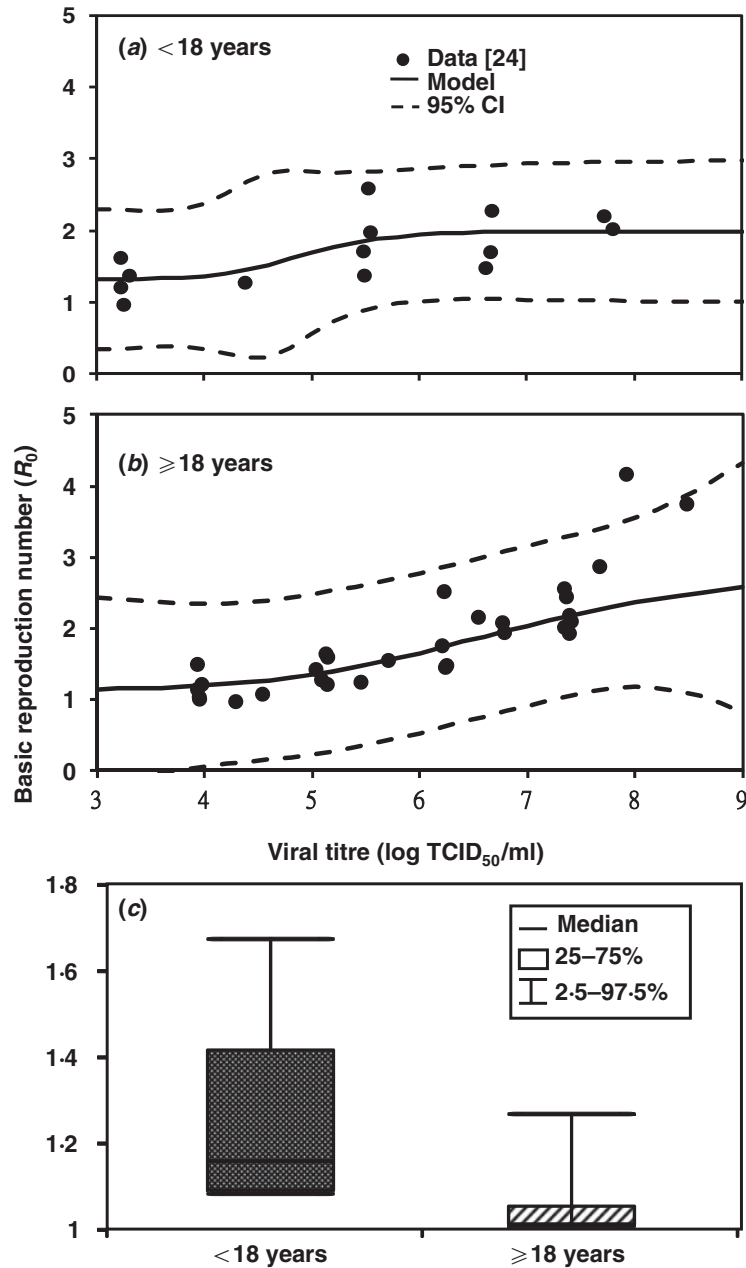
that the total numbers infected in region I was slightly lower than in region II. Moreover, we also revealed that perception spreading might suppress prevalence during the influenza outbreak.

### $R_0$ signals in different age groups

Here a lognormal [LN(geometric mean, geometric standard deviation)] model was optimally fitted to the subtype viral titre data to describe viral titres changing over time for individuals aged <18 years [LN(1.66, 1.86),  $r^2 = 0.97$ ,  $P < 0.01$ ] and  $\geq 18$  years [LN(1.54, 1.51),  $r^2 = 0.54$ ,  $P < 0.01$ ], respectively (Fig. 3). We found that viral titre peaked at day 1 with a value of 3.4 log TCID<sub>50</sub>/ml and then decreased over increasing days since illness onset for those aged <18 years (Fig. 3a). On the other hand, the peak value of viral titre in the  $\geq 18$  years age group occurred at day 3 with 4.4 log TCID<sub>50</sub>/ml (Fig. 3b).

To establish a dose–response profile describing the relationship between viral titre and  $R_0$ , varied by





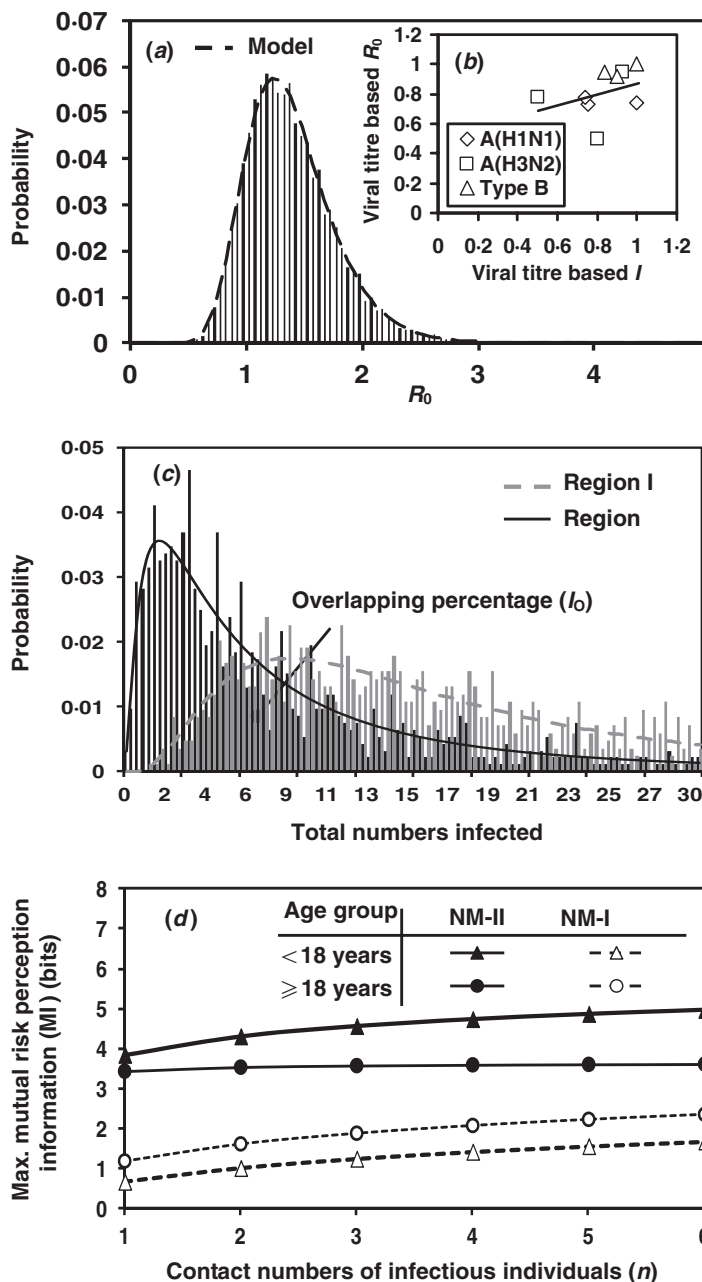
**Fig. 4.**  $R_0$  signals in different age groups. (a)  $R_0$ -viral titre relationship for the <18 years group. (b)  $R_0$ -viral titre relationship for the  $\geq 18$  years group. (c) basic reproduction number ( $R_0$ ) for the <18 and  $\geq 18$  years age groups, respectively.

age group, we used  $R_0$  estimates from published data with viral titres of (sub)type influenza virus A(H1N1), A(H3N2), and type B (see Supplementary material). We found that the Hill equation can reasonably fit the  $R_0$ -viral titre relationship with  $R_0 V_{50}$  of  $3.80 \pm 3.08$  log TCID<sub>50</sub>/ml and  $n_H$  of  $2.90 \pm 14.95$  for the <18 years age group ( $r^2=0.54$ ,  $P < 0.01$ ) (Fig. 4a) and  $R_0 V_{50} = 4.80 \pm 4.20$  log TCID<sub>50</sub>/ml and  $n_H = 4.90 \pm 19.60$  for the  $\geq 18$  year age group ( $r^2=0.42$ ,  $P < 0.01$ ) (Fig. 4b). We also found that estimated median

values of  $R_0$  were 1.16 (95% CI 1.09–1.68) and 1.01 (95% CI 1.01–1.27) for the <18 and  $\geq 18$  years age groups, respectively (Fig. 4c). We showed that  $R_0$  in the <18 years age group had higher variance than in the  $\geq 18$  years age group.

#### Network MI assessment

Figure 5a shows that the LN distribution best describes the model fitting the data, resulting in a  $R_0$



**Fig. 5.** Network maximum mutual risk perception information (MI). (a)  $R_0$  with lognormal (LN) distribution; (b) correlation coefficient ( $\rho$ ) for subtype influenza virus; (c) probability LN distributions of total numbers infected for regions I and II, respectively, (d) simulated network behaviour-influenza models relationship between the contact number of infectious individuals and maximum mutual risk perception information.

variance of LN(1.34, 1.31) ( $r^2=0.99$ ,  $P<0.05$ ). The correlation coefficient ( $\rho$ ) can be estimated from the relationship between viral titre data-based infection ratio and viral titre-based  $R_0$ , resulting in  $\rho=0.4$  for (sub)type influenza viruses (Fig. 5b). We used the optimal fitted LN function to describe probability distributions of the total numbers infected in region I [LN(6.2, 2.8),  $r^2=0.99$ ,  $P<0.01$ ] and region

II [LN(2.6, 0.8),  $r^2=0.99$ ,  $P<0.01$ ] (Fig. 5c). Moreover, the overlapping percentage ( $I_0$ ) could also be estimated as 62% (Fig. 5c).

The variance values used in NM-I and NM-II varied by age group and are listed in Table 3. The results showed that the maximum MI in NM-II were higher with estimates ranging from 3.8–5.0 and 3.4–3.6 compared to those in NM-I of 0.7–1.7 and 1.2–2.4 given

Table 3. Values of variance used in NM-I and NM-II varied by age group

Parameter	Age group	
	<18 years	$\geq 18$ years
$\sigma_{R_0}^2$	0.21	0.59
$\sigma_{R_0 \rightarrow I}^2$ *	0.14	
$\sigma_{R_0 \rightarrow IB}^2$ †	$5.5 \times 10^{-5}$	$3.7 \times 10^{-3}$
$\sigma_{IB \rightarrow I}^2$ ‡	$0.9 \times 10^{-3}$	$1.4 \times 10^{-3}$

$R_0$ , Basic reproduction number;  $I$ , infected fraction of the population; IB, information bottleneck.

\*  $\sigma_{R_0 \rightarrow I}^2 = (1 - \rho^2)\sigma_{R_0}^2 I_0$  is the variance of signal  $R_0$  to response  $I$ .

† Variances of signal  $R_0$  to IB varied by age group and are calculated by  $R_{0, R_0 \rightarrow IB} = \frac{1}{1 - p \left( \frac{1 - R_0}{\mu R_0 + \mu L} \right)}$  [26];  $P$  is the prob-

ability of initial state that does not change to the next stage;  $\mu$  is the probability of the chance that the mutation pathogen in an adaptive host changes to the next state;  $L$  is the expected duration of infection caused by influenza.

‡ Variances of IB to infected ratio  $I$  varied by age group and are calculated by  $R_{0, IB \rightarrow I} = \frac{b}{e + b} \left( 1 - \frac{1}{R_0} \right)$  [27], where  $e$  is the probability of an infected individuals moving to the recovered state and  $b$  is the probability of any host moving to the susceptible state.

the contact numbers of infectious individuals changing from 1 to 6 for age groups <18 and  $\geq 18$  years, respectively (Fig. 5d).

### Behavioural change assessment

Here we considered different age groups adopting different health behaviours (i.e. reduced susceptibility, reduced infectivity, faster recovery) as over-contact links in NM-I and NM-II. Our results indicated that the MI change ratios ( $MI_c$ ) were higher at 0.15–5.55 for those aged <18 years compared to 0.004–4.44 for those aged  $\geq 18$  years (Fig. 6). We found that the health behaviour of faster recovery could most effectively increase the  $MI_c$  ranging from 1.11 to 4.44 in the  $\geq 18$  years age group, whereas for those aged <18 years, the health behaviour of reduced infectivity increased the  $MI_c$  most, ranging from 0.70 to 5.55.

Figure 6 indicates that based on the NM-I model, the  $MI_c$ s of faster recovery altered most significantly in different age groups. However, the  $MI_c$ s of reduced susceptibility and reduced infectivity altered most

significantly for those aged  $\geq 18$  and <18 years, respectively, in the NM-II model.

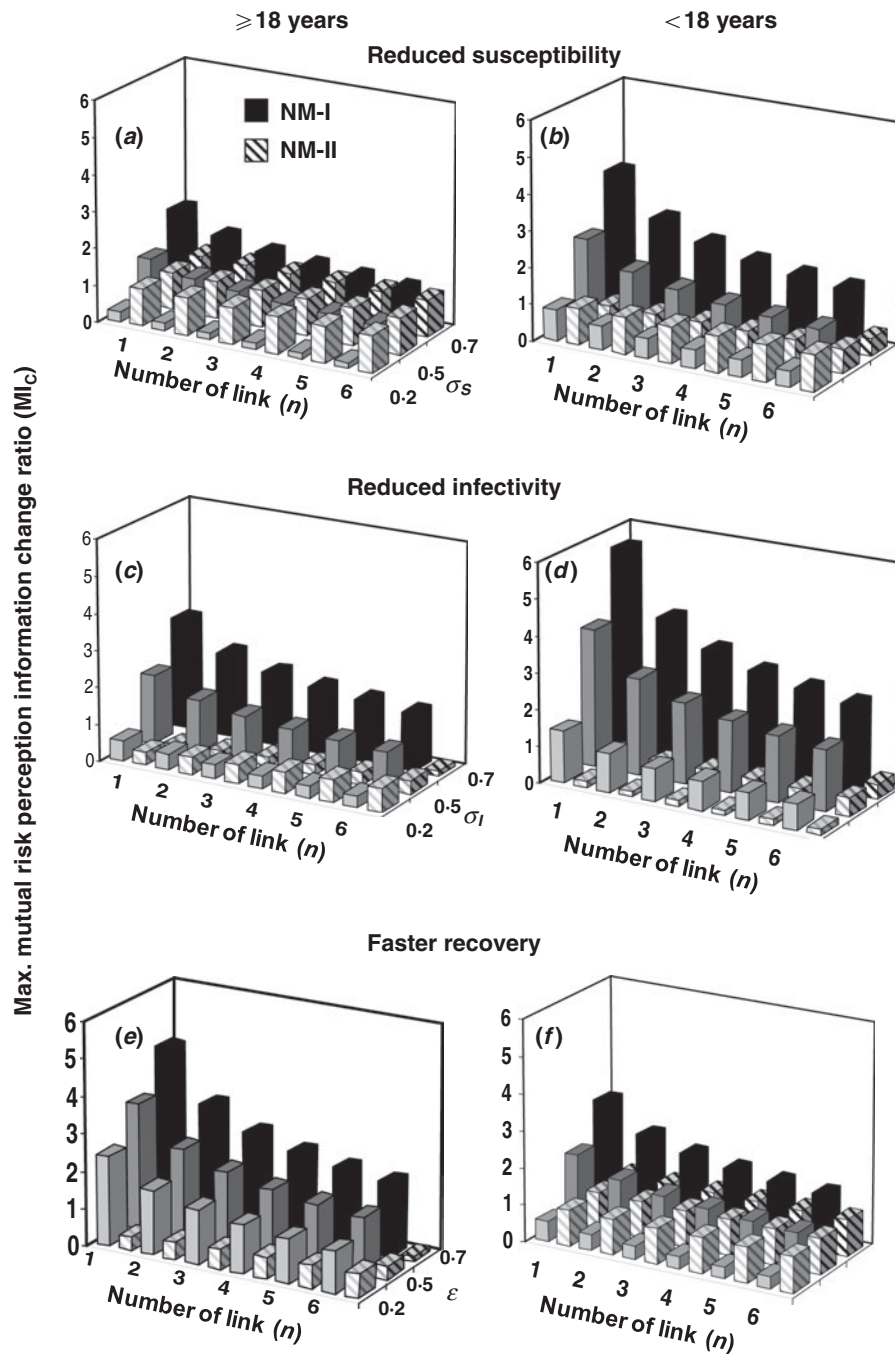
### DISCUSSION

In this study, we incorporated the information-theoretic framework into a BI transmission dynamics system in order to understand the effects of individual behavioural change on an influenza epidemic. We have shown that individuals with a perceptual state will decrease the prevalence in the disease transmission dynamics system. However, it is a challenging task to know how people acquire available sources of risk perception information and transmit it to others. These situations may cause underestimation of the amount of information flow in estimating mutual risk perception information.

The complete joint distribution between input source and output response has to be measured in order to estimate mutual information in a real-world communication channel. Thus, by carefully choosing the input source (e.g.  $R_0$ ) and output response (e.g.  $I$ ), we can apply information theory to a broad variety of behaviour-disease transmission systems. In our proposed BI model,  $P(I|R_0)$  can be readily obtained from a theoretical relationship [22]. However,  $P(R_0)$  cannot be easily estimated [28]. Hence, the amount of information corresponding to a particular signal source of accurate knowledge of disease can be difficult to assess.

Moreover, transmission capacity of mutual information of risk perception can be easily inferred by determining which  $P(R_0)$  yields the maximum amount of information. In particular, channel capacity can measure the ability of a specific behaviour-disease system to transfer maximum information and to examine how information is efficiently processed. Thus, the information theory-based initial analyses can be performed without detailed knowledge of the underlying complexity of the system, providing useful insights into what an information transmission system can or cannot achieve [29].

Several researches have suggested that the perceived risk of disease is a determinant of health-protective change on intention or behaviour. However, the different views of perceived risk may result in influences on behaviour change. For example, self-related risk (e.g. ‘I am at risk’) is a more important factor for behaviour change [30–33] than other related risk perceptions (such as an average of groups is at risk). To raise the individual’s awareness of differential risks faced may help to mitigate the epidemic.



**Fig. 6.** Behavioural change effects on the relationship between the contact number of infectious individuals and the MI change ratios for different age groups. (a, b) Reduced susceptibility; (c, d) reduced infectivity; (e, f) faster recovery.

Yet, the comparative aspect of a difference in risk perceptions remains unclear. Recently, risk information has been shown to stimulate fear, worry, anxiety, etc. Such negative emotion factors could affect perceptions and judgements, and even directly associate with a tendency to engage in protective behaviour as a mediator between perceived risk and intention [34, 35].

A minimal age structure was also considered in our model, allowing us to examine age-specific risk

perception spread rate and increase model realism [13]. Our results showed that mutual risk perception information can be affected by the ability to acquire accurate information by different age groups. Based on our results, for NM-II, it carried over 3–4 bits. We found that collective individual response can increase mutual risk perception information.

Moreover, children would have a higher mutual risk perception (NM-II) than adults. Given the higher

risk perception in children, this study suggests that an authority could focus more on interventions in children such as educational propaganda and vaccination to properly control disease spread. Indeed, children or grandchildren would have more contact with their parents or grandparents than among their peers. Therefore, as long as educational propaganda and vaccination for children is enhanced, the overall disease spread would decline because of reduced susceptibility and infectivity.

Previous studies have found that the differences of health or protective behaviours in terms of their perceived effectiveness and costs depend on varied health behaviour models such as health belief model, subjective expected utility theory, protection motivation theory, and reasoned action theory. Moreover, protective behaviours can be classified into preventive, avoidant, and management of disease behaviours [36]. Renner & Reuter [37] indicated that people need to believe they are at risk in order to take protective action. Liao *et al.* [38] demonstrated that the term of vaccination planning based on a modified theory of a planned behaviour model can be a more significant determinant of vaccination uptake than vaccination intention.

The scenarios of behaviour change were more sensitive to 'faster recovery' based on the NM-I model. Wallace [39] indicated that most active participation in medical decision-making was related to faster recovery. On the other hand, 'reduced susceptibility' was the most influential for risk perception based on the NM-II model. This result implies that a susceptible individual becomes more perceptive to disease risk according to reduced susceptibility to the medication they receive. Numerous studies have also indicated that the higher the perception rate is, the quicker individuals respond to the existence of infection, e.g. receiving vaccination for reduced susceptibility [8, 40].

In conclusion, our results show that information transmission of risk perception plays a crucial role in the spread of health-seeking behaviour throughout an influenza epidemic. This paper also shows that the network BI model provides a new approach for understanding and linking the risk perception spread and human behavioural change during an influenza epidemic. We suggest that a monitoring system associated with information flow of risk perception in the contact networks should be constructed to effectively promote health behaviours in preparation for emerging disease outbreaks.

## SUPPLEMENTARY MATERIAL

For supplementary material accompanying this paper visit <http://dx.doi.org/10.1017/S0950268814000430>.

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## DECLARATION OF INTEREST

None.

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