

# Using simplified models to communicate the importance of prevention, detection and preparedness before a disease outbreak

B. McNab<sup>(1)</sup>, C. Dubé<sup>(2)</sup> & D. Alves<sup>(1)</sup>

(1) Animal Health and Welfare Branch, Ontario Ministry of Agriculture, Food and Rural Affairs, 1 Stone Rd, Guelph, Ontario, N1G 4Y2, Canada

(2) Canadian Food Inspection Agency, 59 Camelot, Ottawa, Ontario, K1A 0Y9, Canada

## Summary

Frontline farm workers and veterinary-policy-makers are arguably in the best positions to influence prevention, detection, and preparedness-for-control of farm animal diseases. It is important that such individuals make biologically sound decisions concerning the daily management and regulation of the health of animals. Such decisions should be based on a good understanding of key principles of disease spread and control. This paper summarises these principles, as described in previous publications, into simple models. These models may be used to communicate concepts to readers who may not have time to study more complex models. These models illustrate the relationship between the development of new disease cases (from existing cases, i.e. the reproductive ratio  $R$ ) and (i) the duration of the period during which existing cases are available as infectious, (ii) contact rates, (iii) transmission rates and (iv) susceptibility. Understanding these concepts through models has great utility, facilitating better decisions for disease prevention, detection and preparedness-for-control, before an outbreak becomes unmanageable. These basic concepts apply to all animal species, including humans.

## Keywords

Communications – Control – Detection – Disease prevention – Disease spread and control model – Preparedness – Reproductive ratio – Stakeholder understanding.

## Introduction

Animal owners and farm workers are arguably in the best position to prevent the introduction of disease, detect abnormalities, and control the spread of infectious disease among animals in their care. At another level, policy-makers in animal industries, animal health and public health are arguably in the best position to influence regulations and funding policies concerning disease prevention, detection and control, within and between their respective areas of responsibility. It is important, therefore, that all these people understand key principles of disease spread and control and the ways in which these relate to their sphere of influence. Then they may take

decisions and actions, informed by these principles, which contribute effectively to prevention, detection and control of diseases. This is true both before outbreaks begin and during their control.

Models may be used to illustrate and communicate these principles (25). Several types of disease models have been developed and reviewed by other authors (9, 19, 22, 23, 33). Examples include mathematical models (1, 10, 11, 14, 18), spatially explicit, stochastic, state-transition computer simulations (12, 16, 28, 29, 30, 32), and, more recently, network models (3, 5, 8, 9). The principal objective of this paper was to summarise key concepts from previous publications into a group of simple examples, schematic

diagrams, basic equations and figures. The aim was to communicate key principles of disease prevention and control to animal health workers and senior policy-makers, and ultimately to improve policy development from the individual farm to the international level. This paper builds slightly on a paper that was published previously by the authors in *Veterinaria Italiana* (25).

## Disease spread and control are inherently exponential

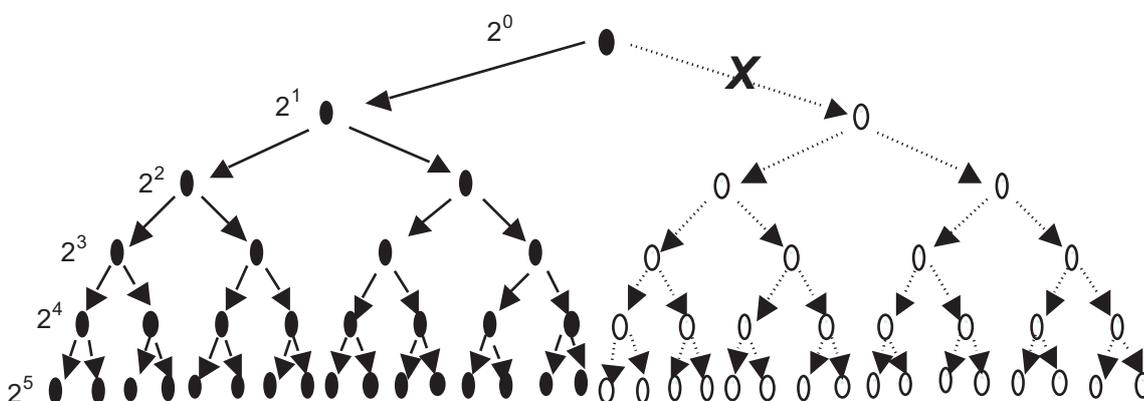
To have a clear understanding of exponential disease spread readers can simply consider their personal experience with the spread of a common cold among people in their household, workplace and community. Suppose each infected person 'gives' their cold to two other people, and each of those people then 'gives' their cold to two more people. Figure 1 is a schematic diagram of such exponential spread between units. Potential units include people (e.g. spreading a cold), or livestock farms (e.g. spreading foot and mouth disease [FMD] between farms). This diagram may also depict spread between people and various species of farmed or wild animals (e.g. some influenza viruses). The number of new cases that are generated per existing case (two in this example) is very important in determining if the outbreak expands, stabilises or decreases over time in the population. This number is known as the reproductive ratio ( $R$ ) (1).

When  $R$  is greater than one (i.e. if, on average, each infected unit infects more than one new unit), then the number of newly infected units continues to increase exponentially. In this example, with a consistent  $R$  of 2, the fifth 'generation' (after the original case) creates 32 (or  $2^5$ )

newly infected units. This brings the total number of units that have been infected to 63. This schematic diagram illustrates too that control measures and other policies can also have impacts that are exponential in nature. For example, if someone does a good job of washing their hands, such that disease is not transmitted at 'X' (Fig. 1), then the entire branch of 31 units that would otherwise have been infected are not infected. We can never know precisely who or what those units are that would otherwise have become infected, and it is true that some may become infected by other routes. Nevertheless, actions at one point can have real value to society by preventing cases far beyond that unit's immediate contacts. Figure 1 schematically illustrates the important contribution that individual people can make to society or their industry, far beyond their respective household or farm, by practising good biosecurity and contact control, to prevent spread of disease on and off their premises.

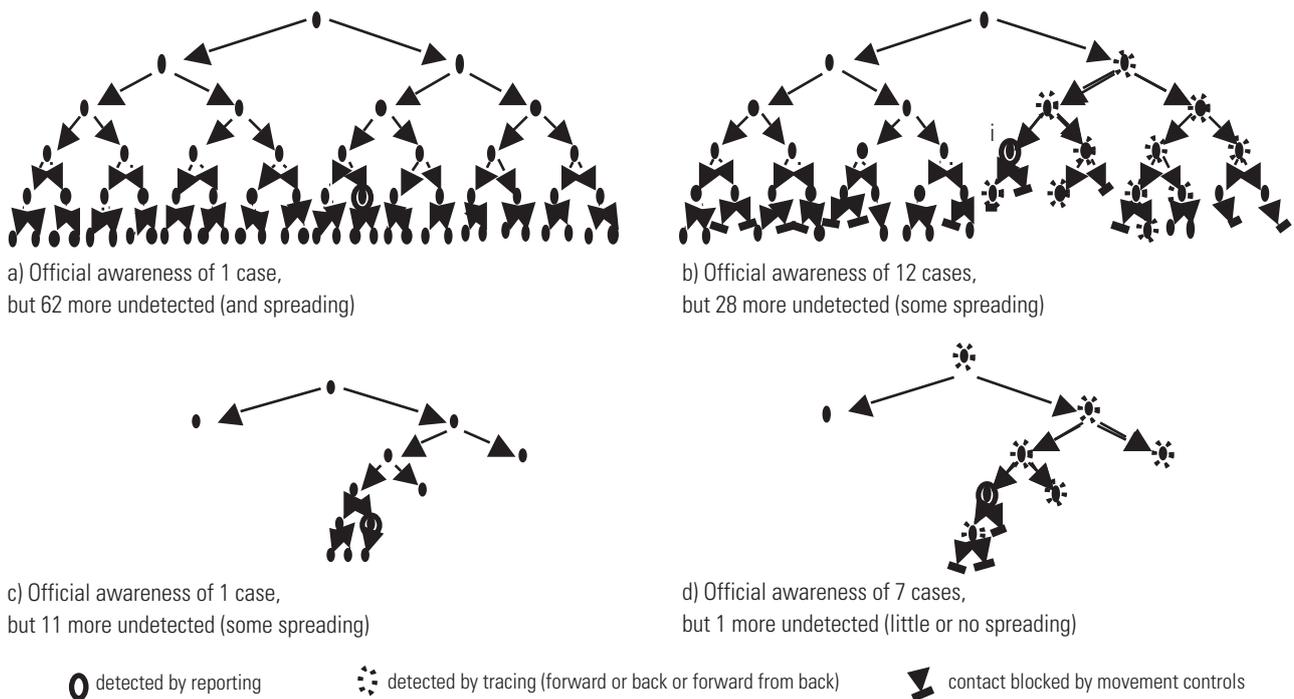
## Better biosecurity, faster detection, and better tracing

Figure 2 schematically summarises four different scenarios of an outbreak of an infectious animal disease among farm units. The scenarios vary in their levels of biosecurity, contact management, disease detection, and tracing effectiveness. Scenario 'a' represents poor biosecurity and contact management resulting in an  $R$  of 2, plus slow detection, and no tracing. This leads to initial official awareness of only one case, some time after the start of the outbreak, but 62 infected units remain as yet unknown to authorities. It can be very difficult for industry and regulatory authorities to regain control of disease spread in such situations.



**Fig. 1**  
Schematic diagram of exponential increase in number of cases if each existing case infects two new cases, i.e. reproductive ratio  $R = 2$

Note the exponential impact on overall control if transmission is blocked at 'X'



**Fig. 2**

**Schematic diagram of four scenarios**

- a) Rapid spread  $R = 2$ , slow detection, no tracing
- b) Rapid initial spread  $R = 2$ , faster detection, reasonable tracing, some prevention of spread, but still considerable spread
- c) Better initial biosecurity and contact control  $R = 1.2$ , but slow detection and no tracing
- d) Better biosecurity and contact control, initial  $R = 1.2$ , with faster detection and good tracing and subsequent movement controls

Scenario 'b' represents poor routine biosecurity with an initial  $R$  of 2, but better official detection (i.e. one generation earlier than scenario 'a', at the unit labelled 'i'), coupled with better tracing. Note the quarantine of 'i' preventing further spread from 'i', and the forward tracing and subsequent quarantine of the unit previously infected by 'i'. Also note the backward tracing to source infection units and subsequent forward tracing from backward traces; this makes it possible to identify infected units and implement quarantine measures to prevent further spread. Furthermore, note that general movement controls are having some effect, preventing transmission from some as yet unknown infected farms relative to scenario 'a'. In scenario 'b', this all leads to relatively rapid official awareness of 12 cases, but there are still 28 unknown cases in the background. There continues to be significant spread, but  $R$  has been decreased from its initial value of two.

Scenario 'c' represents better routine biosecurity and contact management, such that the early  $R$  (i.e. before officials are aware of the outbreak) is down to an average of 1.2 new cases arising from each existing case. Even with slow detection and no tracing, like that of scenario 'a', this leads to a situation of one case initially known to authorities, but 11 unknown cases still in the background.

Scenario 'd' represents good routine biosecurity and contact management, leading to an initial  $R$  of 1.2, plus earlier detection, good tracing and reporting. This results in an official awareness of seven cases and one as yet unknown case in the background. It is interesting to note that, biologically, scenario 'd' is a much better situation for industry and regulatory officials to be in than scenario 'a'. However, initially, scenario 'd' would probably be reported in the press in a manner that would sound much worse (seven known cases) than scenario 'a' (one known case), because the press would be unaware of the potential hidden cases in scenario 'a'. Over the subsequent days and weeks of scenario 'a', officials might detect most of the as yet unknown cases, only to realise how far behind they were in controlling the spread that had occurred from those infected units in the meantime.

It is important that livestock workers, owners, transporters and regulatory officials understand that the likelihood of their jurisdiction facing scenarios similar to 'a', 'b', 'c' or 'd' is influenced greatly by their individual and group efforts, before and during an outbreak. Their routine 'peacetime' practices of movement control and biosecurity greatly influence the  $R$  of disease spread, before officials become aware of an outbreak. Animal handlers' observation of signs of disease (including abnormal consumption or

production) and the speed with which they seek veterinary and laboratory diagnoses influence how quickly serious disease is detected and controls are implemented. Also, the ease with which movement and contact records, as updated by industry, can be analysed influences the speed and accuracy of traces and the precision of targeted disease-control efforts by officials. Veterinary, laboratory and emergency-response infrastructure are also important.

## A simple equation of key factors influencing disease spread and control

To better understand the reproductive rate and the way in which this affects disease spread, the reader can consider the key factors that influence the number of people to whom they transmit or ‘give’ their cold or infection. This can be thought of as their personal R, i.e. the number of new cases that they (the infectious case) create. Self-evident influential factors are summarised in Figure 3 and below. They include:

- the number of ‘infectious days’ (D), i.e. the number of days that an infectious case remains undetected (and therefore uncontrolled) and free to make contact with, and infect, other units (e.g. five days)
- the frequency of contacts they make (C) during that time (e.g. five contacts per day)
- the probability of transmission (T) per contact (e.g. 20%)
- the probability that persons they contact are susceptible (S) (e.g. 40% of contacts are with susceptible people).

Thus, a simplified equation for R (1) may be viewed as:

$$R = D \times C \times T \times S \quad \text{which in this example leads to,}$$

$$R = 5 \text{ infectious days/case} \times 5 \text{ contacts/day} \times 0.2 \text{ transmissions/contact} \times 0.4 \text{ new cases/transmission}$$

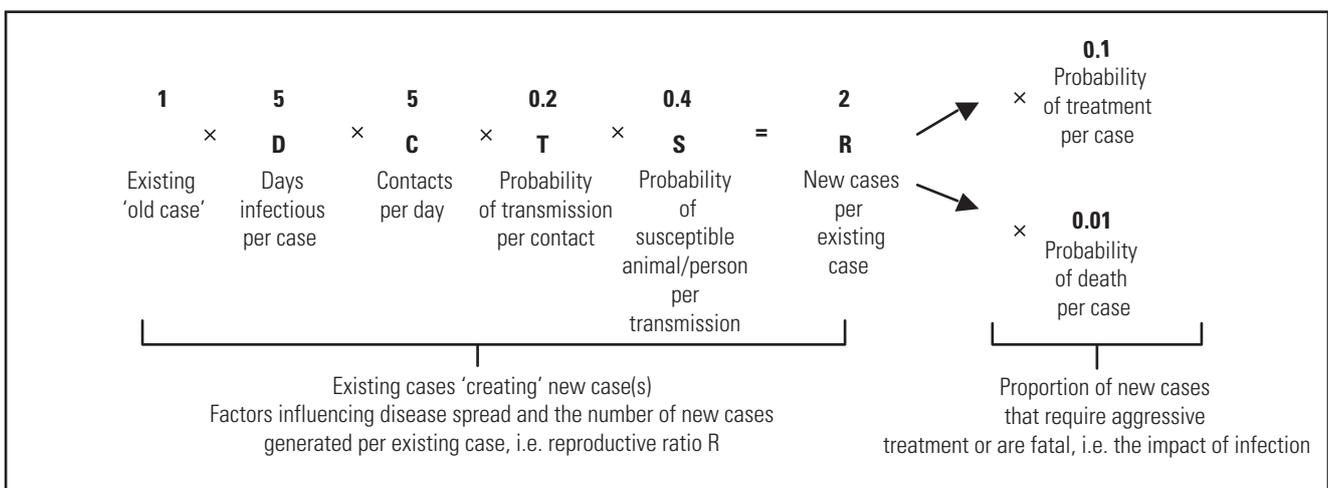
R = 2 cases/case, and is thus a ‘unit-less’ number.

The proportion of people who are susceptible (S) can also be thought of as [1 – (the proportion who are not susceptible)], where the proportion who are not susceptible include: the proportion who are already infected (I) and are naturally immune; plus the proportion who are immune through vaccination (V) for the disease in question; plus the proportion who are deliberately removed (or missing [M]) from exposure, with the intent of preventing exposure and stopping spread.

Thus, the above formula may be rewritten (replacing susceptible S), as:

$$R = D \times C \times T \times [1 - (I + V + M)]$$

This simple formula summarises key factors that influence the spread of infectious disease through a population and thus key factors or combinations of factors that must be altered to control or eradicate disease. It is important to note that R decreases as I increases, even if the other factors remain constant. This means that, all things being equal, the number of new cases produced per existing case will decrease on its own, over time, as the proportion of susceptible units decreases because the proportion of infected units has increased. Therefore, as long as susceptible units are not added to the population by immigration, births or loss of immunity, R will eventually drop below one and the outbreak will ‘burn itself out’ naturally. It is also important to note that the reproductive



**Fig. 3**  
**Schematic summary of the creation of new cases from an existing case (i.e. reproductive ratio 'R') and the impact of infection in terms of case treatment and case fatality rates**

rate R will be decreased by decreasing any combination of the: duration available as infectious (D), contact frequency (C), transmission probability (T), or proportion susceptible (S) (i.e. by increasing proportion infected [I], vaccinated [V], or missing [M]).

Some examples of actions that can be taken to decrease the number of infectious days of existing cases (D) include: staying at home to rest when you have a cold (thereby reducing the number of contacts you make and perhaps also reducing the length of time that you are actually infectious); effective disease surveillance and rapid diagnosis to reduce the duration of time during which there are no controls on infected farms (i.e. early detection, thereby reducing the length of time between the detection of disease and the introduction of quarantine measures); depopulation of animals on farms known to be infected; or pre-emptive depopulation of infected, but not yet infectious, animals (D = 0).

Examples of actions to decrease the contact frequency per infectious day (C) include:

- avoiding meeting people at work as much as possible (if you cannot stay home when you have a cold)
- routinely minimising movement on and off farms to only essential traffic
- restricting the number of contact farms and the frequency of contacts per farm
- establishing direct trading that minimises or eliminates high contact hubs (potential 'super-spreaders')
- implementing stringent quarantine and movement controls during outbreaks.

Decreasing C when infection is caused by longer-range airborne spread is more difficult.

Examples of actions to decrease the probability of transmission (T) per contact include:

- washing your hands frequently
- not shaking hands or kissing to greet people while you have a cold
- isolating additions to a herd
- all-in/all-out practices
- cleaning, disinfecting, and treating contaminated or potentially contaminated materials or equipment before allowing direct or indirect contact with susceptible animals.

Examples of actions that can be taken to decrease the proportion of the population that is susceptible (S) include:

- deliberately infecting animals (I) at a time in the production cycle when infection has less impact (e.g. porcine reproductive and respiratory syndrome in young breeding stock)
- increasing the proportion that is immune by vaccination (V)
- increasing the proportion that are missing (M) (i.e. deliberately removed), so as not to be available for infection even though they would otherwise be susceptible.

This proportion 'missing' (M) may be increased by physically removing susceptible animals by relocation, or preferential slaughter for consumption, or pre-emptive depopulation of non-infected animals at risk. Decreasing the proportion of susceptible animals by increasing immunity (through deliberate infection [I] or vaccination [V]), or removal (M), are analogous to establishing a 'fire-break', where fires are controlled by decreasing the

<b>R per existing case</b>	=	<b>Number of 'infectious days'</b>	×	<b>Contact frequency</b>	×	<b>Transmission probability</b>	×	<b>Susceptible proportion</b>
2	=	5 days	×	5 contacts/day	×	20% transmissions/contact	×	40% susceptible/transmission
Get as low as possible	by	Removing 'infectious'		Decreasing 'contact'		Increasing 'cleanliness'		Increasing 'resistance'
		↓		↓		↓		↓
		Remove what?		Allow who, what, where?		With what, where, who, how?		Select who, vaccinate with what?
Increase precision	by	Distinguishing true status surveillance		System flow ID tracing security		Ongoing hard work		Knowledge of genetics and vaccines

**Fig. 4**  
**Summary of factors that may be influenced to varying degrees of precision to help reduce the number of new cases produced per existing case (R)**  
 In this example R = 2

availability of susceptible (S) fuel. Figure 4 summarises these interactive factors contributing to R and sequentially transcribes them into important prevention, detection, and control activities (as can be seen as one moves down the rows of the figure).

## Precise scalpel versus blunt hammer

Biologically, it does not matter which combination of variables D, C, T, V, or M are altered to achieve  $R < 1$ , to bring an outbreak under control. However, the difficulty in achieving  $R < 1$  efficiently is in lowering D, C, or T with a precision that is limited to truly infected units, and with minimal restrictions to normal activities among non-infected units. Excellent surveillance, rapid diagnostic testing systems with high sensitivity and specificity, effective quarantine and movement controls of truly infected units, excellent biosecurity and effective cleaning, disinfection and treatments are all important to efficiently and effectively lower D, C, and T among truly infected units. Precision is also required to increase V or M efficiently, only among units truly at risk of becoming infected. For example, accurate knowledge of the contribution and characteristics of airborne spread in the specific outbreak will help to target the amount and location of V or M more precisely downwind, if appropriate. Similarly, accurate specific knowledge of direct and indirect movements will help to target the application of M through more precise application of pre-emptive culling or removal only among units that are truly at higher risk of infection, as identified by accurate and timely tracing.

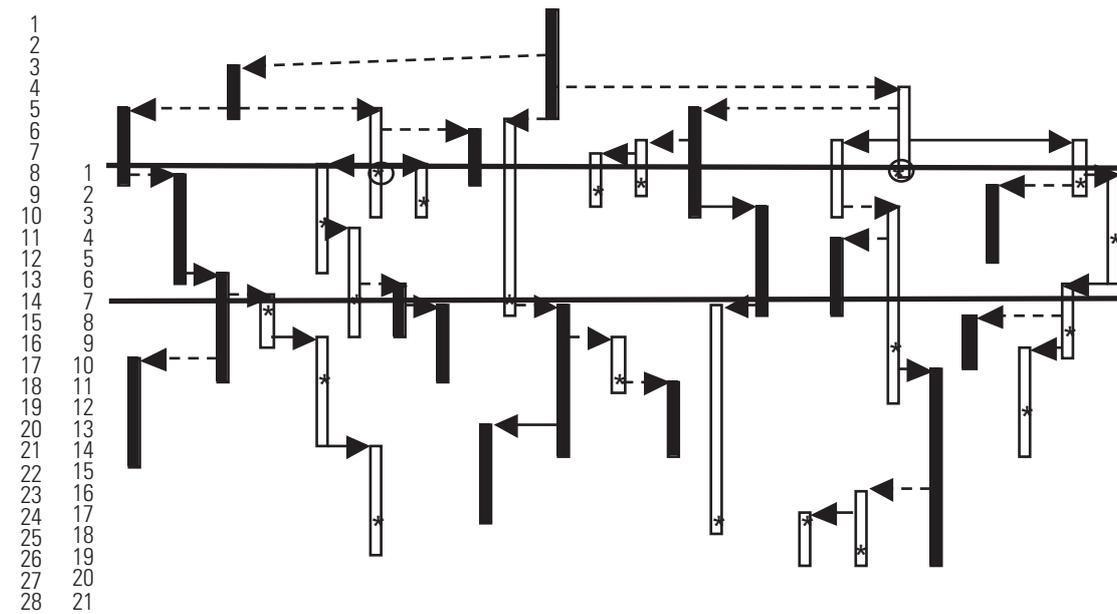
Depending on the situation, appropriately precise, timely, effective and efficient manipulation of D, C, T, V and M may not be technically or logistically feasible. Different combinations will be achievable or required under different situations. For example, if the desired disease reporting, tracing, movement restrictions and biosecurity protocols (designed to reduce D, C and T) are not being implemented appropriately by livestock owners and workers, officials may need to rely on broader, less precise, animal destruction (reducing D and increasing M) and vaccination (V) to gain control. Similarly, if airborne spread is a significant component of contact (C) and transmission (T), then even perfectly implemented movement and biosecurity restrictions may not reduce C and T sufficiently. In such cases, increased vaccination (V) and the removal of a greater proportion of susceptible animals (i.e. increasing M) may be required to reduce the reproductive ratio to below one ( $R < 1$ ). Therefore, considerable understanding, data, risk assessment and judgement are required to make appropriate decisions for

altering D, C, T, V and M effectively and efficiently, with the precision of a scalpel rather than the bluntness of a hammer (15, 17, 21).

## It may be simple but it is not easy

The concepts described above may be simple to understand. However, putting them into practice accurately and precisely during an outbreak is not easy. Figure 5 may be viewed as a more complex version of Figure 1. It schematically illustrates the temporal overlap of infected units of various generations of disease spread. It also illustrates how some cases and contacts may remain unknown to authorities for some time, or perhaps never be identified. Estimating the current or most recent R, in terms of the average number of new cases generated per infectious case, is not easy during an outbreak. This is because newly detected cases may actually be old cases, and several cases may not yet be detected. Also, the exact dates of infection and infectious periods of units are rarely known as precisely as implied in Figure 5. In addition, obtaining true measures of D, C, T, I, V, and M during an outbreak is difficult. Furthermore, since different combinations of D, C, T, I, V and M can result in the same R, it is possible to fit untrue combinations of variables to generate the observed (accurate or inaccurate) R.

Figure 5 schematically illustrates a scenario of true spread among farm units of a generic infectious animal disease. The time units (e.g. days) increase down the left-hand side of the figure. The rectangles represent truly infected herds that are either known or as yet unknown to authorities. The vertical placement and length of each rectangle represent the relative dates and duration of the combined latent and infectious periods of the respective herd unit. White rectangles represent herds known to disease control authorities as infected, found either after having been reported to authorities or by successful traces (forward or backward) from known cases. The vertical location of the asterisk within white rectangles corresponds to the day (in the left-hand time scales) when authorities became aware of the infected herd. The black rectangles represent infected herds that are as yet unknown to authorities. The arrows represent direct, indirect or airborne contacts that truly caused the spread of infection between the specific units indicated, on the date indicated. The solid arrows represent contacts known to authorities. The dotted arrows represent effective transmission contacts that remain as yet unknown to authorities. Note that the true outbreak has been going on for 28 days, but the first discoveries of infected herds (two circled asterisks) were not made until day eight, so the outbreak appears to have been going on for only 21 days.



Actual time-scale (days) of outbreak: far left. Apparent time-scale of outbreak: inner scale  
 White rectangles and solid arrows represent infected herds and contacts known to authorities  
 Asterisks illustrate the day the herd was detected  
 Circled asterisks mean initial detection of infected herd  
 Black rectangles and dotted arrows represent infected herds and contacts not yet known to authorities

**Fig. 5**  
**Schematic diagram of disease spread over time illustrating temporal overlap of herds during their respective latent and infectious periods, and the concept of herds known and not known to authorities**

Upper horizontal line suggests herds that might be included in the denominator of R (i.e. old cases) from which new cases (numerator of R) were generated, as suggested by some of the herds crossed by the lower horizontal line, one complete disease generation period later (arbitrarily set at one week in this illustrative example)

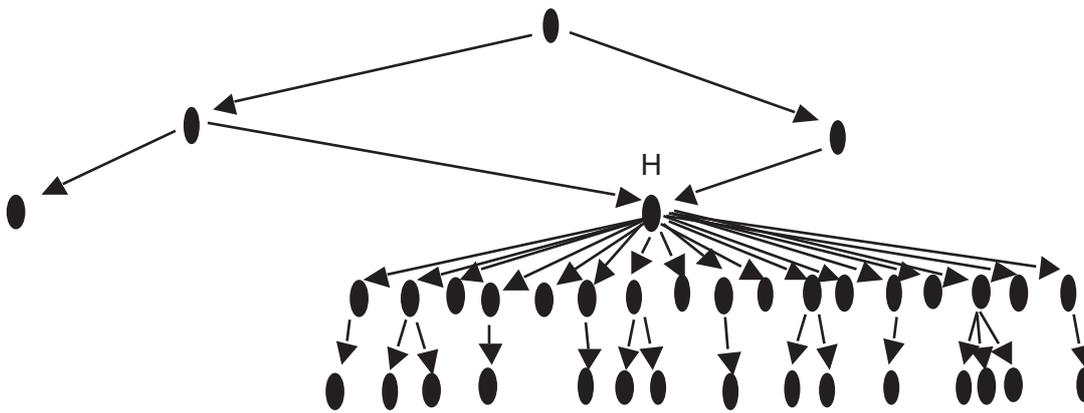
It is useful to estimate R at different points in time during an outbreak to learn if R is decreasing and if it has been reduced to less than one. However, to do so, it is crucial to know which known infected herds should be classified as new infections, and which are old infections serving as sources for the current new infections. That is, it is crucial to know which cases should be included in the numerator (some of the herds crossed by the lower horizontal line in Fig. 5) and which in the denominator (some of the herds crossed by the upper horizontal line in Fig. 5) of R. This difficulty, combined with the problem of as yet undetected old and new cases (and some herds may even be false positives), can lead to an incorrect estimate of R. Furthermore, an incorrect estimate of R can lead to inappropriate policy decisions because the outbreak is perceived as being under control (erroneous  $R < 1$ ) or out of control (erroneous  $R > 1$ ). Therefore, considerable skill, data, knowledge and judgement are required to assess outbreaks (15, 17, 21).

## Networks

The previous examples (Fig. 3) assumed equal numbers of transmission contacts by infected individuals or farms.

Recent studies of animal movements in Great Britain (5), Denmark (3) and Canada (8) have demonstrated that while most livestock operations have a few contacts, some may act as super-spreaders due to their interactions with a high number of other units (e.g. markets, dealers) in scale-free contact networks (2, 18, 20). Figure 6 schematically illustrates the impact of a hub (H) or super-spreader. Note the above average number of new cases generated from H. In this example, the average R is 1.6 (36/22), but unit H is responsible for the creation of 17 new cases. If the new cases arising from H are excluded, the average R is 0.9 (19/21), which is less than 1. From a disease control point of view, this illustrates the importance of avoiding the creation of such hubs, or ensuring extremely rapid control of infection and transmission from such hubs, during an outbreak.

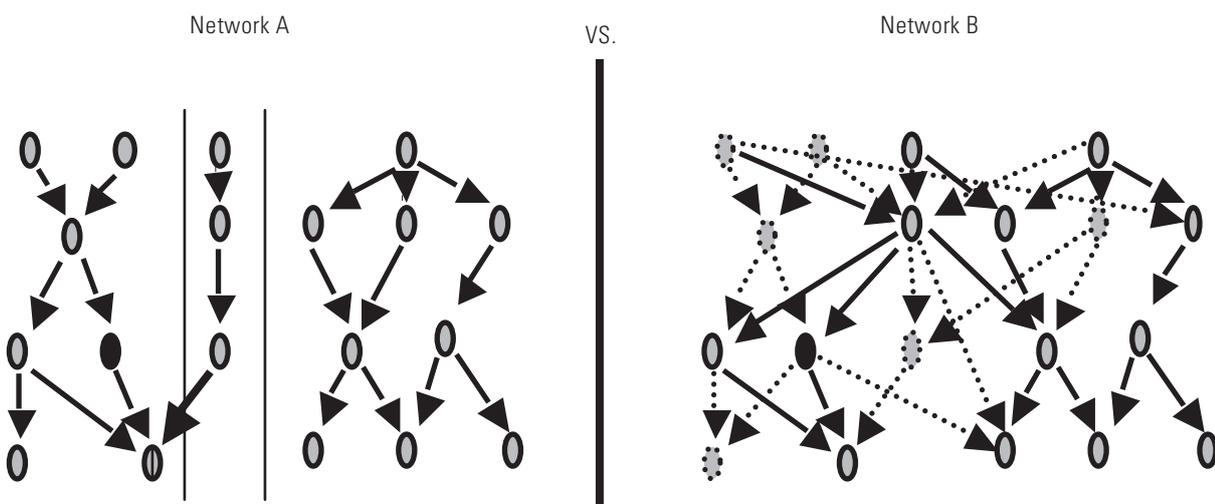
Furthermore, transmission between units rarely follows a simple system of contacts among randomly selected units, at variable frequencies. In reality, most human and business contacts form reasonably structured networks. Animal movement networks may be deliberately designed and implemented in a manner that compartmentalises components of the system, to prevent spread of disease. The OIE recognises such compartmentalisation as a method of controlling disease spread. However, more



**Fig. 6**  
**Schematic diagram of extensive disease spread from an active 'hub' (H) or 'super-spreader' in scale-free contact networks**

often, networks of animal movements evolve in ways that improve short-term economic gain but are not necessarily biologically robust. Recently, formal network analysis has been applied to livestock movements (3, 5, 8, 9). But before one can study the characteristics of a contact network using network analysis, one must have accurate data of the nodes (units) and arcs or vectors (contacts) that make up that network. Data on unique premises identification (nodes) and each movement between each specifically identified pair of premises (vectors) are required to do such network analyses. Figure 7 is a schematic representation of two different networks. In network 'A', all nodes (units or premises) and vectors (contacts or livestock movements) are known, as represented by solid-edged ovals and solid arrows. Also, three different groups of nodes (components of the

network) are relatively isolated. Disease spread, after random introduction of infection into network 'A' (e.g. at solid oval), would be limited to the smaller component of the network (on the left in this example). Furthermore, because all units (nodes) and movements (vectors) are known, tracing is rapid and precise. Therefore, control of spread is quite feasible. In contrast, network 'B' (Fig. 7) includes several premises (nodes) and contacts (vectors) that are real but unknown to authorities (represented by dotted ovals and arrows). Also, the entire network is more highly connected in 'B' than in 'A'. This makes understanding, prioritisation, tracing and control of spread much more difficult in 'B' than in 'A'. Therefore, the design and understanding of true contact networks is extremely important to the prevention and control of disease spread in populations.



**Fig. 7**  
**Schematic diagram of two networks, 'A' and 'B'**  
 In network 'A' all nodes and contacts are known and some components are relatively isolated. Rapid tracing and control are feasible, when one infected unit is detected (solid oval). In contrast, in network 'B' some true nodes and contacts remain unknown (dotted ovals and arrows), at least one hub exists and the network is more highly connected, making control more difficult

## Discussion and conclusions

The trade disruption and economic impacts caused by serious infectious diseases such as FMD are more significant in exporting countries that are normally free of such animal diseases (4, 7, 13, 24, 26, 27, 31). Farm workers and owners may perceive themselves as having little influence over the control of diseases that are usually foreign to their country. They may feel dependent on government authorities to protect them from such exotic animal diseases. Similarly, citizens may believe that they personally have little influence on the introduction and spread of disease in their communities, and erroneously assume that only public health authorities can stop the spread of disease. In addition, people, including regular citizens, animal husbandry workers, livestock owners, animal-health regulatory officials and public policy decision-makers, routinely experience competing demands for their time and attention. As a result, they do not necessarily have the time or technical skills to study and fully appreciate complex mathematical, computer simulation models or network models. Nevertheless, their decisions and actions are often vital to successful control of disease (6).

The disease spread and control concepts summarised in this paper have all been reported and reviewed previously in more detail, with greater mathematical and epidemiological rigour (1, 2, 3, 8, 9, 12, 14, 18, 20, 23, 28, 30, 33). The objective of this paper was to summarise concepts into simplified models to communicate key principles of infectious-disease control to people who do not have time to study more detailed models (25).

Many factors can influence the development of policies, practices and the actual behaviour of livestock owners, workers, buyers, sellers, transporters or regulatory officials. Changes in behaviour may, in turn, influence: the number of infectious days (D), the frequency of contact (C), the probability of transmission (T), or the proportion susceptible (S). The collateral damage that seemingly unrelated and otherwise logical policy changes can have on the spread of disease may not be fully realised until a new strain of pathogen emerges. Some examples of stimuli for change in behaviour include: market changes, technology changes, labour costs, transport costs, trade barriers, changes in consumer preferences, a general change in

public or government priorities, a re-prioritisation of resources available for veterinary inspection or diagnostic infrastructure, etc. Caution is recommended before implementing local or wide spread changes to policy. Specifically, the authors recommend that policy-makers fully think through and model the implications that proposed changes are likely to have on the spread and control of disease. Examples of questions that may be asked include, as a result of the proposed change: is the contact network likely to change to become more or less connected over all, or produce more influential or fewer hubs? Will prevention of disease spread be affected through decreased or increased frequency of direct or indirect contact (C), or the probability of transmission per contact (T)? Will susceptibility (S) or vaccine use (V) be increased or decreased? Will surveillance, detection, accessibility to diagnostics, or traceability be increased or decreased (thus affecting the number of infectious days [D] before controls are implemented)?

People may alter their behaviour according to their perception of risks and benefits. People are more likely to take constructive actions if they honestly believe their personal actions will make a positive difference to people or systems about which they sincerely care. The simple examples, schematic diagrams and equations summarised here are intended to help citizens, livestock workers, owners and policy decision-makers appreciate how their personal day-to-day decisions and actions can greatly influence the size and impact of disease outbreaks in their own households or farms, and on their industry or society as a whole. This is not only true during official responses to known outbreaks; it is also true before the outbreak is known to authorities, and even before it starts. Society will benefit if people take responsibility for their personal disease reproductive ratio (R). This will reduce the number of new cases they generate personally and it will reduce the subsequent exponential spread of disease that would have otherwise occurred in subsequent generations of cases. We hope that a broader appreciation of the key principles summarised here will compel stakeholders to act routinely in a manner that improves the prevention and control of infectious diseases.



## Les modèles simplifiés et leur utilisation en communication pour démontrer l'importance de la prévention, de la détection et de la préparation avant un foyer de maladie

B. McNab, C. Dubé & D. Alves

### Résumé

Les personnes qui, par leur activité, exercent le plus d'influence sur la prévention, la détection et la préparation à la lutte contre les maladies des animaux d'élevage sont sans conteste celles qui s'occupent des animaux dans les exploitations et celles qui sont chargées d'élaborer les politiques de santé animale. Il est essentiel que toutes leurs décisions, qu'il s'agisse de gestion quotidienne ou de réglementation de la santé animale, soient fondées biologiquement. En particulier, elles doivent reposer sur une solide connaissance des grands principes régissant la propagation et la prophylaxie des maladies animales. Les auteurs résument ces principes tels qu'ils ont été exposés dans des publications antérieures, en les présentant sous forme de modèles simples. Ces modèles peuvent servir à diffuser certains concepts parmi des lecteurs qui n'ont pas le temps d'étudier des modèles plus élaborés. Il s'agit de modèles illustrant la relation entre l'apparition de nouveaux cas d'une maladie (à partir des cas existants – donc le ratio de reproduction R), d'une part, et la durée d'infectiosité des cas existants, le taux d'exposition, le taux de transmission et la susceptibilité, d'autre part. Les modèles sont des outils précieux pour appréhender ces concepts et faciliter une prise de décisions plus efficace en vue de prévenir et détecter les maladies, mais aussi de préparer une prophylaxie adéquate avant que le foyer ne devienne ingérable. Ces concepts de base s'appliquent à toutes les maladies animales et humaines.

### Mots-clés

Communication – Détection – Modèles de propagation et de contrôle des maladies – Participation éclairée des parties prenantes – Préparation – Prévention des maladies – Prophylaxie – Ratio de reproduction.



## Utilización de modelos simplificados para explicar la importancia de la prevención y detección de brotes infecciosos y de la preparación antes de que se produzcan

B. McNab, C. Dubé & D. Alves

### Resumen

Seguramente los trabajadores que están en contacto directo con los animales en las explotaciones, así como los planificadores de políticas veterinarias, son quienes están en mejor situación para influir en la labor de prevención, detección y preparación para el control de las enfermedades de los animales de granja. Es importante que esas personas adopten decisiones correctas desde el punto de vista biológico en relación con la regulación y gestión diaria de la salud

de los animales, decisiones que deben reposar en una buena comprensión de los principios básicos de la propagación y el control de enfermedades. Esos principios, descritos en publicaciones previas, vienen sintetizados aquí en forma de modelos sencillos que pueden utilizarse para transmitir una serie de conceptos a lectores que quizá no tengan tiempo de estudiar modelos más complejos. Dichos modelos ponen de manifiesto la relación entre la aparición de nuevos casos de enfermedad (respecto del número de casos existentes, o en otras palabras: el cociente reproductivo R) y la duración del periodo en el cual los casos existentes son infecciosos, así como los índices de exposición y de transmisión y la susceptibilidad. Entender estos conceptos mediante modelos es muy útil, pues facilita la adopción de decisiones más atinadas para prevenir y detectar enfermedades y preparar la lucha contra ellas antes de que un brote escape a todo control. Estos conceptos básicos se aplican a todas las especies animales, comprendido el ser humano.

#### Palabras clave

Cociente reproductivo – Comprensión por las partes interesadas – Comunicaciones – Control – Detección – Modelos de propagación y control de enfermedades – Preparación – Prevención de enfermedades.



## References

1. Anderson R.M. & Nokes D.J. (1991). – Mathematical models of transmission and control. In Oxford textbook of public health, 2nd Ed. (W.W. Holland, R. Detels & G. Knox, eds). Vol. II, Chapter 14. Oxford Medical Publications, Oxford, 225-252.
2. Barabasi A.L. & Bonabeau A. (2003). – Scale-free networks. *Sci. Am.*, **288** (5), 60-69.
3. Bigras-Poulin M., Thompson R.A., Chriel M., Mortensen S. & Greiner M. (2006). – Network analysis of Danish cattle industry trade patterns as an evaluation of risk potential for disease spread. *Prev. vet. Med.*, **76**, 11-39.
4. Burrell A. (2002). – Outbreak, control and prevention of animal diseases: economic aspects and policy issues. AGR/CA/APM(2002)19. Working Party on Agricultural Policies and Markets, Directorate for Food, Agriculture and Fisheries, Committee for Agriculture. Organisation for Economic Co-operation and Development, Paris, 53 pp.
5. Christley R.M., Robinson S.E., Lysons R. & French N.P. (2005). – Network analysis of cattle movement in Great Britain. In Proc. Annual Meeting of the Society for Veterinary Epidemiology and Preventive Medicine, April, Inverness, 234-244.
6. Crispin S.M. (2005). – Foot-and-mouth disease: the vital need for collaboration as an aid to disease elimination. *Vet. J.*, **169**, 162-164.
7. Dijkhuizen A.A. (1989). – Epidemiological and economic evaluation of foot-and-mouth disease control strategies in the Netherlands. *Neth. J. agric. Sci.*, **37**, 1-12.
8. Dubé C., Ribble C., Kelton D. & McNab B. (2008). – Comparing network analysis measures to determine potential epidemic size of highly contagious exotic diseases in fragmented monthly networks of dairy cattle movements in Ontario, Canada. *Transbound. emerg. Dis.*, **55** (9-10), 382-392.
9. Dubé C., Ribble C., Kelton D. & McNab B. (2009). – A review of network analysis terminology and its application to foot-and-mouth disease modelling and policy development. *Transbound. emerg. Dis.*, **56** (3), 73-85.
10. Ferguson N.M., Donnelly C.A. & Anderson R.M. (2001). – The foot-and-mouth epidemic in Great Britain: pattern of spread and impact of interventions. *Science*, **292**, 1155-1160.
11. Ferguson N.M., Donnelly C.A. & Anderson R.M. (2001). – Transmission intensity and impact of control policies on the foot and mouth epidemic in Great Britain. *Nature*, **413**, 542-548.
12. Garner M.G. & Beckett S.D. (2005). – Modelling the spread of foot-and-mouth disease in Australia. *Aust. vet. J.*, **83**, 30-38.

13. Garner M.G., Fisher B.S. & Murray J.G. (2002). – Economic aspects of foot and mouth disease: perspectives of a free country, Australia. *In* Foot and mouth disease: facing the new dilemmas (G.R. Thomson, ed.). *Rev. sci. tech. Off. int. Epiz.*, **21** (3), 625-635.
14. Green L.E. & Medley G.F. (2002). – Mathematical modelling of the foot and mouth disease epidemic of 2001: strengths and weaknesses. *Res. vet. Sci.*, **73**, 201-205.
15. Guitian J. & Pfeiffer D. (2006). – Should we use models to inform policy development? *Vet. J.*, **172**, 393-395.
16. Harvey N., Reeves A., Schoenbaum M.A., Zagmutt-Vergara F.J., Dubé C., Hill A.E., Corso B.A., McNab W.B., Cartwright C.I. & Salman M.D. (2007). – The North American Animal Disease Spread Model: a simulation model to assist decision making in evaluating disease incursions. *Prev. vet. Med.*, **82**, 176-197.
17. Honhold N., Taylor N.M., Wingfield A., Einshoj P., Middlemiss C., Eppink L., Wroth R. & Mansley L.M. (2004). – Evaluation of the application of veterinary judgement in the pre-emptive cull of contiguous premises during the epidemic of foot-and-mouth disease in Cumbria in 2001. *Vet. Rec.*, **155**, 349-355.
18. Kao R.R. (2002). – The role of mathematical modelling in the control of the 2001 FMD epidemic in the UK. *Trends Microbiol.*, **10**, 279-286.
19. Keeling M.J. (2005). – Models of foot-and-mouth disease. *Proc. roy. Soc. Lond., B, biol. Sci.*, **272**, 1195-1202.
20. Kiss I.Z., Darren M.G. & Kao R.R. (2006). – Infectious disease control using contact tracing in random and scale-free networks. *J. roy. Soc., Interface*, **3**, 55-62.
21. Kitching R.P., Hutber A.M. & Thrusfield M.V. (2005). – A review of foot-and-mouth disease with special consideration for the clinical and epidemiological factors relevant to predictive modelling of the disease. *Vet. J.*, **169**, 197-209.
22. Kitching R.P., Thrusfield M.V. & Taylor N.M. (2006). – Use and abuse of mathematical models: an illustration from the 2001 foot and mouth disease epidemic in the United Kingdom. *In* Biological disasters of animal origin. The role and preparedness of veterinary and public health services (M. Hugh-Jones, ed.). *Rev. sci. tech. Off. int. Epiz.*, **25** (1), 293-311.
23. Kostova-Vassilevska T. (2004). – On the use of models to assess foot-and-mouth disease transmission and control. US Department of Homeland Security, Advanced Scientific Computing Program. UCRL-TR-205241. Lawrence Livermore National Laboratory, University of California, Livermore, California, 37 pp. Available at: [www.llnl.gov/tid/lof/documents/pdf/309485.pdf](http://www.llnl.gov/tid/lof/documents/pdf/309485.pdf) (accessed on 15 January 2007).
24. Laurence C.J. (2002). – Animal welfare consequences in England and Wales of the 2001 epidemic of foot and mouth disease. *In* Foot and mouth disease: facing the new dilemmas (G.R. Thomson, ed.). *Rev. sci. tech. Off. int. Epiz.*, **21** (3), 863-868.
25. McNab B. & Dubé C. (2007). – Simple models to assist in communicating key principles of animal disease control. *Vet. ital.*, **43** (2), 317-326.
26. Mangen M.-J.J., Burrell A.M. & Mourits M.C.M. (2004). – Epidemiological and economic modelling of classical swine fever: application to the 1997/1998 Dutch epidemic. *Agric. Syst.*, **81**, 37-54.
27. Marangon S. & Capua I. (2006). – Control of avian influenza in Italy: from stamping out to emergency and prophylactic vaccination. *In* Proc. OIE/FAO International Scientific Conference on Avian Influenza (A. Schudel & M. Lombard, eds). *Dev. Biol. (Basel)*, **124**, 109-115.
28. Morris R.S., Wilesmith J.W., Stern M.W., Sanson R.L. & Stevenson M.A. (2001). – Predictive spatial modelling of alternative control strategies for the foot-and-mouth disease epidemic in Great Britain, 2001. *Vet. Rec.*, **149**, 137-144.
29. Nielen M., Jalvingh A.W., Meuwissen M.P.M., Horst S.H. & Dijkhuizen A.A. (1999). – Spatial and stochastic simulation to evaluate the impact of events and control measures on the 1997-1998 classical swine fever epidemic in the Netherlands. II. Comparison of control strategies. *Prev. vet. Med.*, **42**, 297-317.
30. Schoenbaum M.A. & Disney W.T. (2003). – Modeling alternative mitigation strategies for a hypothetical outbreak of foot-and-mouth disease in the United States. *Prev. vet. Med.*, **58**, 25-52.
31. Scudamore J.M., Trevelyan G.M., Tas M.V., Varley E.M. & Hickman G.A.W. (2002). – Carcass disposal: lessons from Great Britain following the foot and mouth disease outbreaks of 2001. *In* Foot and mouth disease: facing the new dilemmas (G.R. Thomson, ed.). *Rev. sci. tech. Off. int. Epiz.*, **21** (3), 775-787.
32. Stevenson M.A., Sanson R.L., Stern M.W., O'Leary B.D., Mackereth G., Sujau M., Moles-Benfell N. & Morris R.S. (2006). – InterSpread Plus: a spatial and stochastic simulation model of disease in animal populations (Research project BER-60-2004). Biosecurity New Zealand Technical Paper No. 2005/NN. Ministry of Agriculture and Forestry, Biosecurity New Zealand, Wellington.
33. Taylor N. (2003). – Review of the use of models in informing disease control policy development and adjustment. A report for the Department for Environment, Food and Rural Affairs (DEFRA) Veterinary Epidemiology and Economics Research Unit, Reading, 94 pp. Available at: [www.defra.gov.uk/science/documents/publications/2003/UseofModelsInDiseaseControlPolicy.pdf](http://www.defra.gov.uk/science/documents/publications/2003/UseofModelsInDiseaseControlPolicy.pdf) (accessed on 15 January 2006).