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Atmospheric dispersion modelling of bioaerosols that are pathogenic to humans and livestock – A review to inform risk assessment studies

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ABSTRACT

In this review we discuss studies that applied atmospheric dispersion models (ADM) to bioaerosols that are pathogenic to humans and livestock in the context of risk assessment studies. Traditionally, ADMs have been developed to describe the atmospheric transport of chemical pollutants, radioactive matter, dust, and particulate matter. However, they have also enabled researchers to simulate bioaerosol dispersion.

To inform risk assessment, the aims of this review were fourfold, namely (1) to describe the most important physical processes related to ADMs and pathogen transport, (2) to discuss studies that focused on the application of ADMs to pathogenic bioaerosols, (3) to discuss emission and inactivation rate parameterisations, and (4) to discuss methods for conversion of concentrations to infection probabilities (concerning quantitative microbial risk assessment).

The studies included human, livestock, and industrial sources. Important factors for dispersion included wind speed, atmospheric stability, topographic effects, and deposition. Inactivation was mainly governed by humidity, temperature, and ultraviolet radiation.

A majority of the reviewed studies, however, lacked quantitative analyses and application of full quantitative microbial risk assessments (QMRA). Qualitative conclusions based on geographical dispersion maps and threshold doses were encountered frequently. Thus, to improve risk assessment for future outbreaks and releases, we recommended determining well-quantified emission and inactivation rates and applying dosimetry and dose–response models to estimate infection probabilities in the population at risk.

1. Introduction

1.1. Perspective on bioaerosols

Aerobiology is the research area focusing on the generation and transport of *bioaerosols*. Bioaerosols are small, airborne particles consisting of biological material (from bacteria, viruses, spores, fungi, algae, protozoa, and pollen) either attached to particulate matter or not (Bovallius and Roffey, 1987; Després et al., 2012; Dungan, 2010; Gilbert and Duchaine, 2009; Griffin, 2007; Stärk, 1999; Wéry, 2014). Large amounts of bioaerosols are produced each year by sources including the natural environment and livestock farms (Cambra-López et al., 2010; Viana et al., 2008).

Pathogenic or *infectious* bioaerosols possibly cause respiratory infections after penetration into the respiratory system of humans or animals (Stärk, 1999; Stuart and Wilkening, 2005; Wéry, 2014). The pathogenicity to cause disease is dependent on the pathogen's infectivity, and its ability to be transported and to survive (Anderson and Bokor, 2012; Kersh et al., 2013; La Scola and Raoult, 2001; Rousset et al., 2009). After being emitted or aerosolised from its source, dispersion to the surrounding environment (nearby residents, livestock, etc.) may occur. However, large-scale measurements are not generally available, time-consuming and expensive, thus complicating pathogen quantification. Also, pathogens may be inactivated in the air as well (e.g., by temperature or humidity), (Després et al., 2012; Griffiths and DeCosemo, 1994; Verreault et al., 2008).

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1.2. Atmospheric dispersion models

Atmospheric dispersion models (ADMs) may be helpful to describe the dispersion of pathogenic bioaerosols. ADMs are mechanistic models describing the transport of gases and particles – including chemical pollutants, radioactive matter, particulate matter, and dust – in the atmosphere in space and time (Holmes and Morawska, 2006; Markiewicz, 2012; Potempski et al., 2008). ADMs are widely used in the risk assessment of hazardous effects of air pollution on humans and the environment (e.g., Schaap et al., 2013). Sources are classified as either continuous (e.g., air and odour quality monitoring of emissions from industry or animal housing) or instantaneous (e.g., release of hazardous material from large fires in industrial buildings).

The advantage of mechanistic models is that they incorporate physical processes describing dispersion and that they are able to predict the dispersion process based on measurements (Kuparinen, 2006). Furthermore, most measurements are point samples in space and/or time, but ADMs can predict concentrations at high spatial and temporal resolutions. During an outbreak they may efficiently provide information, either to inform sampling or for the benefit of other response functions, such as vaccination or distribution of antibiotics (Stuart and Wilkening, 2005).

Historically, ADMs were often based on the Gaussian dispersion equation (see Section 2.4; Markiewicz, 2012, Millner, 2009) to calculate concentrations at local scales (<30 km) in a three-dimensional frame. Nowadays, most ADMs include important atmospheric processes related to fluid dynamics (e.g., turbulence) as well (Nathan et al., 2005; Upper and Hirano, 1991). Some also simulate trajectories of backward and forward spatial motions, or dispersion of so-called pollutant puffs. Furthermore, increased computer power has stimulated the development of models based on computational fluid dynamics (CFD) that include landscape features such as buildings and trees (Nathan et al., 2005; Westbrook and Isard, 1999).

Although ADMs were initially developed to simulate chemical pollutant dispersion, they have enabled researchers to simulate dispersion of bioaerosols at different spatial and temporal scales and resolutions (Després et al., 2012). Moreover, by using quantitative estimates of emission rates (i.e. the amount of pathogen emitted per unit of time, see Section 2.2), airborne concentrations (representing exposure) can be converted to doses using dosimetry models (see Section 3) to subsequently perform a quantitative microbial risk assessment (QMRA) (Paez-Rubio et al., 2007; Upper and Hirano, 1991).

ADMs are useful to address concerns about public health risks related to exposure from, for instance, livestock sources and sources related to biosafety agents (e.g., *Bacillus anthracis* and *Coxiella burnetii*) (Anderson and Bokor, 2012; Smit et al., 2012). In addition, ADMs are particularly useful in case of future outbreaks or releases. Knowledge of pathogen emission, host-susceptibility, and complex atmospheric processes may help professionals to assess and to reduce airborne infection risks (Westbrook and Isard, 1999).

1.3. Aim and outline

The objectives of this review were to present an overview of:

- the most important physical processes related to atmospheric dispersion modelling and pathogen transport (Section 2),
- studies that focused on the application of ADMs to simulate airborne transmission of pathogenic bioaerosols (Section 4),
- parameterisations regarding emission and inactivation in these ADM studies (Section 5), and
- methods for conversion of concentrations to infection probabilities applied (concerning quantitative microbial risk assessment) in the ADM studies (Sections 3 and 6),

and to place these in the context of risk assessment modelling. We focused on pathogenic bioaerosols transmitted in the outdoor environment causing airborne infections in humans and livestock. Our focus was not on direct human–human or animal–animal transmission. We used the word *pathogen* in the context of pathogenic bioaerosols. Tables 1 and 2 list respectively all abbreviations and parameters used in this review. Table 3 lists all atmospheric dispersion models discussed in this review. Appendix A lists all studies reviewed in Section 4.

Table 1
List of abbreviations.

Pathogens	
AIV	Avian influenza virus
FMDV	Foot-and-mouth-disease virus
PRV	Pseudorabies virus
SARS	Severe Acute Respiratory Syndrome
Atmospheric dispersion models	
ADMS	Atmospheric Dispersion Modelling System
AERMOD	AMS/EPA Regulatory Model
ALOHA	Areal Locations of Hazardous Atmospheres
CALPUFF	Californian Puff model
DERMA	Danish Emergency Response Model of the Atmosphere
DREAM	Dust Regional Atmospheric Model
GIADA	Guida Interattiva ad Applicazione per la Dispersione Atmosferica
HPAC	Hazard Prediction and Assessment Capability
HYSPLIT	Hybrid Single-Particle Lagrangian Integrated Trajectory model
INPUFF	Integrated PUFF model
LODI	Lagrangian Operational Dispersion Integrator
MLCD	Modèle Lagrangien Courte Distance
NAME	Numerical Atmospheric-dispersion Modelling Environment
OMEGA	Operational Multiscale Environment Model with Grid Adaptivity
OPS-ST	Operational Priority Substances Short Term model
RIMPUFF	Risø Mesoscale PUFF model
Meteorological models	
ECMWF	European Centre for Medium-Range Weather Forecasts
HiRLAM	High Resolution Limited Area Model
LAPS	Limited Area Prediction System (ABM)
MM5	Fifth-generation Penn State/NCAR Mesoscale Model
NCEP/NCAR	Numerical Weather Prediction model of NCEP and NCAR
Institutes	
ABM	Australian Bureau of Meteorology (Australia)
AMS	American Meteorological Society (USA)
DWD	German Weather Service (Deutsche Wetter Dienst) (Germany)
EPA	Environmental Protection Agency (USA)
KMAA	Korean Meteorological Administration Agency (South-Korea)
KNMI	Royal Netherlands Meteorological Institute (The Netherlands)
NCAR	National Center for Atmospheric Research (USA)
NCEP	National Centers for Environmental Prediction (USA)
NMI	Norwegian Meteorological Institute (Norway)
NOAA	National Oceanic and Atmospheric Administration (USA)
Other	
CFD	Computational Fluid Dynamics
CFU	Colony forming units
DR	Dose–response
GDAS	Global Data Assimilation System
ID_{50}	Median infectious dose
IU	Infectious unit
LD_{50}	Median lethal dose
NWP	Numerical Weather Prediction (model)
PSD	Particle size distribution
QMRA	Quantitative microbial risk assessment
SIR	Susceptible-Infected-Recovered
$TCID_{50}$	Median tissue culture infectious dose
WWTP	Wastewater treatment plant

2. Atmospheric dispersion models

2.1. Physical processes

Five major processes are related to the number of infections caused by airborne pathogens:

(1) The amount of pathogen released per unit of time (emission rate), being a function of pathogen availability and the aerosolisation rate (Shao, 2008; Viana et al., 2008; see Section 2.2).

Table 2
List of parameters discussed in this review.

Parameter	Explanation	Unit	Equation(s)
a	Age	[years]	(10), (11)
A	Cross-sectional area	[m^2]	(3), (4)
c_1, c_2	Shape parameters	[m^3 years g^{-1}]	(11)
C	Concentration	[g m^3]	(1)–(5)
D	Mean pathogen dose	[g m^3]	(7)–(12)
h	Plume height	[m]	(5)
H	Emission height	[m]	(2)
K_x	Eddy diffusion coefficient in x direction	[m^2 s^{-1}]	(1)
K_y	Eddy diffusion coefficient in y direction	[m^2 s^{-1}]	(1)
K_z	Eddy diffusion coefficient in z direction	[m^2 s^{-1}]	(1)
n	Number of pathogens	[#]	(6)
P_{inf}	Probability of infection	[dimensionless]	(6)–(12)
Q	Emission rate	[g s^{-1}]	(1)–(5)
r	Single-hit probability	[dimensionless]	(6), (7)
t	Time	[s]	(1), (5)
u	Wind speed in the x direction	[m s^{-1}]	(1)
U	Wind speed in the downwind direction of a source	[m s^{-1}]	(2), (3), (5)
v	Wind speed in the y direction	[m s^{-1}]	(1)
w	Wind speed in the z direction	[m s^{-1}]	(1)
W	Width of the plume column	[m]	(5)
x	Coordinate	[m]	(1), (2)
y	Coordinate	[m]	(1), (2)
z	Coordinate	[m]	(1), (2)
z_0	Roughness length	[m]	(5)
α	Parameter in the hyper-geometric and Poisson dose–response models	[dimensionless]	(8), (9)
β	Parameter in the hyper-geometric and Poisson dose–response models	[dimensionless]	(8), (9)
γ	Shape parameter	[dimensionless]	(10)
Δ	Shape parameter	[dimensionless]	(10)
ϵ	Shape parameter	[dimensionless]	(10)
ζ	Shape parameter	[dimensionless]	(10)
η	Shape parameter	[dimensionless]	(12)
θ	Angle between wind direction and field edge	[deg]	(5)
κ	Shape parameter	[dimensionless]	(12)
Λ	Inactivation rate	[s^{-1}]	(2)
$\sigma_y(x)$	Diffusion factor in y direction	[m]	(2)
$\sigma_z(x)$	Diffusion factor in z direction	[m]	(2)
ϕ	Flow rate	[m^3 s^{-1}]	(4)

(2) Meteorological effects, such as wind speed, wind direction, turbulence, and deposition (Stull, 2000). Mechanical turbulence is generated by wind speed variation in height; convective turbulence is related to the stability of the atmosphere. A mix of different wind conditions and solar radiation results into three basic states of the atmosphere – unstable, stable, and neutral – that largely influence the surface layer concentrations (Jacob, 1999). Unstable atmosphere are characterised by vertical (buoyant) motions induced by thermal convection that lifts particles up to higher altitudes, thus leading to decreased surface concentrations. A stable atmosphere leads to higher surface concentrations as low wind speeds and limited solar radiation limit vertical mixing. Neutral atmospheres are characterised by mainly horizontal (advective) turbulent motions with moderate vertical mixing and high horizontal plume extent.
Deposition is subdivided in wet deposition (the removal of particles by cloud and rain droplets) and dry deposition (the dust flux from the atmosphere to the surface through molecular and turbulent diffusion and gravitational settling) (Flossmann et al., 1985; Petroff et al., 2008; Shao, 2008). The dry deposition rate is a function of particle size: very large particles (±70 μm) deposit about 10,000 times faster than ultrafine (≤0.1 μm) particles (Lin et al., 1994). The *particle size distribution* – the relative amount of particles as a function of mass or number – is therefore an important predictor for the distance covered (Blatny et al., 2011).

(3) Inactivation (Griffin, 2007), expressed as a function of time or meteorological conditions, such as temperature and humidity (Zhao et al., 2014; see Section 2.5). Large differences in inactivation rates are observed among bioaerosols (Zhao et al., 2014; see Section 5.2): viruses and vegetative bacteria may be inactivated within minutes to hours or days, while spores are generally highly persistent (Dungan, 2010; Griffiths and DeCosemo, 1994; Jones and Harrison, 2004; Stuart and Wilkening, 2005). Note that growth of microorganisms can also occur (Harrison et al., 2005).

(4) The amount of pathogens inhaled, with breathing rate, lung volume, and particle size being important factors (Després et al., 2012; Rostami, 2009; Wilkinson et al., 2012; see Section 3). With respect to particle size, the inhalable (≤100 μm: particles breathed in), thoracic (≤10 μm: particles entering the lung's airways), and respirable fraction (<5 μm: particles penetrating the terminal bronchioles) are distinguished (Millner, 2009).

(5) The host's health response as a function of inhaled dose (Teunis and Havelaar, 2000; see Section 3).

Thus, a full risk assessment would comprise the chain of emission quantification, atmospheric dispersion modelling, dose estimation, and estimating the probability of infection using dose–response models (Section 3).

To run ADMs, meteorological data (observed, modelled, or predicted) are required. Observational data comprise in situ measurements, data from (local) weather stations, or data from the *Global Data Assimilation System* (GDAS), which is a worldwide weather observation database (NOAA 2014). Modelled data are generally retrieved from numerical weather prediction models using meteorological observations from GDAS. For local/regional dispersion studies (up to several kilometres) observational data from a nearby meteorological station are sufficient or high-resolution weather data (2.5 km) could be used (Van der Plas et al., 2012). For very local dispersion studies, where the effect of local landscape features is relevant, in situ measurements are required describing the local micrometeorology.

2.2. Emission rates

An emission rate is defined as an amount released per unit of time. Emission rates for pathogens depend on source type (pigs, poultry, industrial, humans, etc.), source characteristics (e.g., stable construction or animal activity), excretion route (e.g., exhaled air or faeces), pathogen species or strain, particle size, etcetera. For a full quantitative risk assessment, quantified emission rates are required.

2.3. Eulerian and Lagrangian ADMs

ADMs are either Gaussian (Section 2.4) or Eulerian or Lagrangian. The Eulerian model is based on a fixed grid in space where the concentration as a function of time is described for an observer at a specific location. The vertical dimension (z) is generally expressed as height [m] or pressure [Pa]. Most Eulerian models are based on the advection–diffusion equation, being a simplification of the more

Table 3
Atmospheric dispersion models discussed in this review (∗ = unknown).

Abbreviation	Developer	Gaussian plume	Eulerian		Lagrangian			Deposition[a]	PSD[b]	Reference
			Advection-diffusion	CFD	Gaussian puff	Particle mode	Trajectory			
ADMS	Cambridge Environmental Research Consultants, Met Office, INNOGY Holdings plc, University of Surrey (UK)	x	–	–	–	–	–	d, w	yes	(Carruthers et al., 1994)
AERMOD	AMS (USA); EPA (USA)	x	–	–	–	–	–	d, w	yes	(Cimorelli et al., 2004; EPA, 2004)
ALOHA	NOAA (USA)	x	–	–	–	–	–	–	no	(Jones et al., 2013)
CALPUFF	EPA (USA)	–	–	–	x	–	–	d, w	yes	(Scire et al., 2000)
DERMA	Danish Meteorological Institute (Denmark)	–	–	–	x	–	–	d, w	yes	(Sørensen et al., 2007)
DREAM	University of Malta	–	x	–	–	–	–	d, w	yes	(Nickovic et al., 2001)
Fluent	ANSYS (USA)	–	–	x	–	–	–	∗	∗	(ANSYS, 2014)
GIADA	Italian Environmental Protection Agency	x	–	–	–	–	–	∗	∗	∗
HPAC	Defense Threat Reduction Agency (USA)	–	–	–	x	–	–	∗	∗	∗
HYSPLIT	NOAA (USA); ABM (Australia)	–	–	–	x	x	x	d, w	yes	(Draxler and Hess, 2014)
ICAIR	∗	–	–	–	x	–	–	∗	∗	∗
INPUFF	EPA (USA)	–	–	–	x	–	–	d	yes	(Petersen and Lavdas, 1986)
LODI	Department of Energy (USA), University of California (USA)	–	–	–	–	x	–	d, w	yes	(Leone et al., 2001)
MLCD	University of Alberta (Canada); Canadian Meteorological Centre (Canada)	–	–	–	x	–	–	d, w	no	(Flesch et al., 2002)
NAME	Met Office (UK)	–	–	–	x	x	x	d, w	yes	(Borrego and Norman, 2007, chapter 62)
OMEGA	Center for Atmospheric Physics, Science Applications International Corporation (USA)	–	x	–	x	x	–	d, w	yes	(Bacon et al., 2000)
OPS–ST	National Institute for Public Health and the Environment (RIVM) (The Netherlands)	x	–	–	–	–	–	d, w	yes	(Van Jaarsveld, 2004)
RIMPUFF	Risø National Laboratory (Denmark)	–	–	–	x	–	–	d, w	yes	(Thykier-Nielsen et al., 1999)
SCREEN3	EPA (USA)	x	–	–	–	–	–	–	no	(EPA, 1995)

[a] Deposition included (d = dry, w = wet).
[b] Particle Size Distribution (PSD) included.

complex Navier–Stokes equations for fluid dynamics (Kim and Moin, 1985; Stull, 2000). The advection–diffusion equation describes the movement of particles influenced by the wind and turbulent diffusion:

$$\frac{\partial C}{dt} = Q - u\frac{\partial C}{\partial x} - v\frac{\partial C}{\partial y} - w\frac{\partial C}{\partial z} + \frac{\partial}{\partial x}\left[K_x\frac{\partial C}{\partial x}\right] + \frac{\partial}{\partial y}\left[K_y\frac{\partial C}{\partial y}\right] + \frac{\partial}{\partial z}\left[K_z\frac{\partial C}{\partial z}\right] \quad (1)$$

all in units of number of pathogens per m^3 per unit of time. The factor on the left-hand side describes the local change in concentration (C) in time; Q is the emission rate; the next three factors on the right-hand side describe the transport (advection) by the mean wind speed (u,v,w) in directions [x,y,z]; the final three terms describe the transport by turbulent motions where K is the (eddy) diffusion coefficient [$m^2\ s^{-1}$].

Lagrangian models also solve the advection-diffusion equation, but they simulate particle or air transport relative to a frame moving with the mean flow as if an observer moves with a particle (Jacob, 1999). Lagrangian models are able to create backward and forward trajectories to visualise the origin and destination of particles or air.

Eulerian and Lagrangian models are suited for simulation of homogeneous and steady-state conditions, as well as for heterogeneous and non-steady state conditions, and for flat surfaces as well as for terrains with much topography (Holmes and Morawska, 2006). Their general spatial resolution is in the order of several kilometres to thousands of kilometres. A particular form of Eulerian and Lagrangian models are those based on Computational Fluid Dynamics (CFD) that numerically solve the Navier–Stokes equations. CFD-models are very useful in complex terrains, such as mountains or urban environments, where the spatial scales of interest are close to the scales of landscape features. A disadvantage of CFD models is the large amount

of detailed information (including meteorological) required for simulation.

2.4. The Gaussian dispersion equation

A relatively simple solution of the advection–diffusion equation is the Gaussian dispersion equation (Nickovic et al., 2001; Pasquill, 1974) traditionally used in atmospheric dispersion studies:

$$C(x,y,z) = \frac{Q}{2\pi \cdot U \cdot \sigma_y(x) \cdot \sigma_z(x)} \cdot \exp\left[-\frac{1}{2}\cdot\left(\frac{y}{\sigma_y(x)}\right)^2\right] \cdot\left[\exp\left[-\frac{1}{2}\cdot\left(\frac{z-H}{\sigma_z(x)}\right)^2\right] + \exp\left[-\frac{1}{2}\cdot\left(\frac{z+H}{\sigma_z(x)}\right)^2\right]\right] \cdot \exp\left[-\lambda\cdot\frac{x}{U}\right] \quad (2)$$

where Q is again the emission rate, U is the wind speed [m/s], H is the emission height [m], and $\sigma_y(x)$ and $\sigma_z(x)$ are the diffusion factors in the y and z directions [m] (EPA, 1995). The second factor of Eq. (2) describes the crosswind dispersion (in the y-direction). The third and fourth describe vertical dispersion in the z-direction without and with reflections from the surface (by assuming a virtual source at $-H$ m height). The last factor describes inactivation with rate λ [s^{-1}] (Lighthart and Frisch, 1976; see Section 2.5).

The equation predicts the concentration at any location downwind of a source and assumes a Gaussian distribution of the particles in the crosswind (y) and vertical (z) planes (Dungan, 2010). The plume axes are always projected with respect to the wind direction with transport in the downwind direction [x] solely caused by advection by the wind. Eq. (2) assumes steady-state approximations, i.e. no parameter is time-dependent (Holmes and Morawska, 2006).

A *Gaussian plume model* includes Eq. (2) in a fixed frame, whereas a *Gaussian puff model* includes the equation nested in a Lagrangian or trajectory model. Puff models split a continuous plume in discrete particle packets, each individually transported, dispersed and evolving in size (Scire et al., 2000). The model determines the contribution of a puff to the concentration at a receptor.

2.5. Inactivation rate

Inactivation is the process of death or elimination of pathogens due to certain environmental or meteorological conditions (Jones and Harrison, 2004; Pica and Bouvier, 2012; Stärk, 1999), such as high temperatures (generally increasing inactivation), ultraviolet radiation (increasing), and humidity (both decreasing and increasing) (Al-Dagal and Fung, 1990; Jones and Harrison, 2004; Zhao et al., 2014). Dust and droplets may protect pathogens to fluctuations in meteorological conditions, such as dehydration (Mandrioli, 1998; Zhao et al., 2014). In case of re-aerosolisation, survival conditions in soil and water should also be considered (Franz et al., 2014; La Scola and Raoult, 2001; Rzezutka and Cook, 2004; Weber and Stilianakis, 2008).

3. Dosimetry and dose–response models

The next step after having calculated exposure levels using an ADM is to calculate the dose individuals are exposed to. A dose can be expressed as the number of pathogens, infectious units (IU), or colony forming units (CFUs) per unit of volume.

The simplest way to retrieve a dose is to assume the dose is equal to the modelled concentration. However, since not all pathogens are inhaled, variables like inhalation rate and exposure duration might better be included for a more detailed dose estimation (Casal et al., 1997; Dungan, 2014; Li et al., 2013; Low et al., 2007; Schley et al., 2009; Sørenson et al., 2000, 2001, Ssematimba et al., 2012; Stellacci et al., 2010). Dosimetry models can also include particle size distribution and deposition (Isukapalli et al., 2008). Examples of such models include those of Anjilvel and Asgharian (1995), Georgopoulos et al. (2005), and Rostami (2009). Once the dose is calculated, a probability of infection can be determined.

Threshold doses are, however, equal to a binary step function and are therefore generally considered inadequate for risk assessment (Teunis and Havelaar, 2000). In contrary, dose–response (DR) models describe the probability of infection (or another health outcome) given a specific dose (EPA, 2012) and are a key ingredient for quantitative microbial risk assessments (QMRA) (Dungan, 2014; Teunis and Havelaar, 2000). The *binomial, exponential, hyper-geometric*, and *beta-Poisson* models are most used (EPA 2012; Haas, 2002; Teunis and Havelaar, 2000):

$$-\ \text{Binomial}: \quad P_{inf}(n,r) = 1-(1-r)^n \quad (3)$$

$$-\ \text{Exponential}: \quad P_{inf}(D,r) = 1-\exp(-r\cdot D) \quad (4)$$

$$-\ \text{Hyper}-\text{geometric}: \quad P_{inf}(D,\alpha,\beta) = 1-{}_1F_1(\alpha,\alpha+\beta,-D) \quad (5)$$

$$-\ \text{Beta}-\text{Poisson}: \quad P_{inf}(D,\alpha,\beta) = 1-\left(1+\frac{D}{\beta}\right)^{-\alpha} \quad (6)$$

where P_{inf} is the probability of infection, n is the number of pathogens, r is the probability that ingestion of a single pathogen results in infection (single-hit), ${}_1F_1()$ is the Kummer confluent hypergeometric function, D is the mean pathogen dose [g m^3], and α and β correspond to the beta distribution parameters for specific ranges (EPA 2012; Teunis and Havelaar, 2000).

The binomial model is a single-hit model for discrete doses describing the infection probability as a function of the complement of the probability of absence of infection. The exponential model, also a single-hit model, describes the probability of infection given ingestion with a Poisson-distributed dose with mean D. The hypergeometric model is an integrated version of the exponential model for population averaged doses, allowing for variation in the single-hit probability between individual pathogens and/or between hosts (Teunis and Havelaar, 2000). The beta-Poisson model is an approximation of the hyper-geometric model.

DR-models have been developed for several pathogens, including FMDV (exponential and beta-Poisson) (French et al., 2002), *B. anthracis* (mainly exponential) (Bartrand et al., 2008; Huang and Haas, 2009; Toth et al., 2013), the avian influenza virus (time-dependent exponential and beta-Poisson) (Kitajima et al., 2011), *Legionella pneumophila* (exponential and beta-Poisson) (Armstrong and Haas, 2008; Bouwknegt et al., 2013), and *C. burnetii* (exponential and beta-Poisson) (Tamrakar et al., 2010). With respect to *C. burnetii*, Brooke et al. (2013) and Jones et al. (2006) described the *C. burnetii* single-hit probability being 0.44 or 0.9, respectively; the ID_{50} was estimated to be 1.18 organisms, and the median dose for illness was estimated to be 5.58 bacteria (Brooke et al., 2013). Berendt et al. (1980) showed that the ID_{50} for *L. pneumophila* was <129 organisms and the LD_{50} (median lethal dose) was 1.4×10^5 organisms.

4. Pathogenic bioaerosol studies using atmospheric dispersion models

This section discusses studies using ADMs to simulate dispersion of pathogenic bioaerosols. Our literature search query (see Appendix B) included keywords regarding ADMs, general pathogen keywords (e.g., 'bioaerosols' and 'pathogens'), and pathogens and diseases from a list of the European Centre for Disease Control (ECDC, 2010), from several bioaerosol reviews (Al-Dagal and Fung, 1990; Després et al., 2012; Dungan, 2010; Gilbert and Duchaine, 2009; Griffin, 2007; Griffiths and DeCosemo, 1994; Jones and Harrison, 2004; Monn and Koren, 1999) and from a report on emerging zoonoses in

the Netherlands (Van der Giessen et al., 2010). All non-airborne microorganisms and diseases related to non-airborne microorganisms from the ECDC list have been filtered out as a result of the combination of keywords regarding the ADMs.

In this chapter we reviewed the foot-and-mouth disease virus (Section 4.1), *B. anthracis* (Section 4.2), the avian influenza A virus (Section 4.3), *L. pneumophila* (Section 4.4), *C. burnetii* (Section 4.5), and the Pseudorabies virus (Section 4.6). Section 4.7 comprises other pathogens.

4.1. Foot-and-mouth disease virus

The foot-and-mouth disease virus (FMDV) affects cloven-hoofed animals, mainly cattle, sheep, and pigs, and is highly infectious (Kitching et al., 2005). Major transmission routes include airborne spread from infected farms, movement of infected livestock or contaminated persons, objects and animal products, and excretion of urine, faeces, semen and tissues (Alexandersen et al., 2003; Cottam et al., 2008; Donaldson, 1997; Kitching et al., 2005; Klein, 2009; Pharo, 2002). The incubation period varies from four to fourteen days (Sellers and Forman, 1973). Large outbreaks occurred in countries including Canada, the United Kingdom, France, Germany, Denmark, Spain, the Netherlands, and South Korea (Alexandersen et al., 2003; Bouma et al., 2003; Carrillo et al., 1990; Cottam et al., 2008; Donaldson et al., 1982; Kritana et al., 2014, Sellers and Daggupaty, 1990; Sellers and Forman, 1973; Sørenson et al., 2000; Valarcher et al., 2009). We identified four main analysis techniques for ADM simulation. Several studies simply reconstructed the geographical virus spread. Some subsequently applied a threshold dose to identify the farms at risk. Thirdly, a few applied a dose–response function to calculate a probability of infection or a sensitivity and specificity rate.

4.1.1. United Kingdom, 1966–1968

In 1966–1968, four districts in the UK were affected by FMDV (Gloster et al., 2005b; Henderson, 1969; Sellers and Forman, 1973; Smith and Hugh-Jones, 1969). Daily dosages at surrounding farms were calculated with a simplified version of the Gaussian dispersion equation (Blackall and Gloster, 1981; Gloster et al., 1981). A threshold dose of 1 *infectious unit* (IU) (basically 1 virus particle) was applied to conclude there was a "very close agreement" between predictions and observations. Note that currently more advanced dose–response models are available (Section 3) that supersede the infectious dose paradigm.

The epizootic was reconstructed later in several other studies of varying quality. Although developed for gas dispersion modelling, (Casal et al., 1997, 1995) used the Areal Locations of Hazardous Atmospheres (ALOHA) model and applied a threshold dose to predict the farms' infectivity status. They simply concluded that the predictions "agreed relatively well to the observations" and that the predicted doses per receptor farm were uncertain due to uncertain emission rates and the possible existence of other transmission routes. In other words, predicted concentrations were compared to the observations at farms in a qualitative way, and estimated infection risks using dose–response models were not calculated to support the hypothesis. (Gloster et al., 2005b) used the Atmospheric Dispersion Modelling System (ADMS) and concluded that all farms could have been exposed (given the 'ideal' meteorological conditions). They discussed that other actual atmospheric stability conditions could explain misclassified farms.

Schley et al. (2009) used the Numerical Atmospheric-dispersion Modelling Environment (NAME) and did, however, go further by applying an exponential dose–response model. As one of the few, they assessed the quality of their predicted infection risks by calculating a specificity and sensitivity rate, being 82% and 94% respectively regarding the farms' infectivity status. Sanson et al. (2011) showed that the occurrence of airborne spread was very significant ($p \approx 0.00$), although other transmission routes could not be excluded. Topographic effects highly influenced the bioaerosol spread and were thus very important to include in such risk assessment studies.

Gloster et al. (2010) performed a comparison study among six ADMs, namely the Californian Puff Model (CALPUFF), the Hybrid Single-Particle Lagrangian Integrated Trajectory model (HYSPLIT), the Lagrangian Operational Dispersion Integrator (LODI), the Modèle Lagrangien Courte Distance (MLCD), NAME, and the Risø Mesoscale PUFF model (RIMPUFF). Unfortunately, the results were not analysed statistically: they simply concluded that all six models could be used for dispersion assessment during outbreaks, although (small) differences in predicted areas at risk were observed.

4.1.2. France and the United Kingdom, 1981–1982

In 1981–1982 an epizootic occurred in Bretagne (France) with successive outbreaks in the UK at the Island of Jersey (75 km north) and the Isle of Wight (250 km north). Donaldson et al. (1982) applied a Gaussian model and concluded that criteria for a successful long-range transmission – favourable wind speed, wind direction, stable atmosphere, and high emission rates – were fulfilled. However, if there was indeed an airborne link between the farms in France and the UK, then more outbreaks in France might have been expected, yet were not reported. Moutou and Durand (1994) used the ICAIR model and explained 10 out of 13 secondary outbreaks, but both their method description and analyses were described very limitedly. Sørenson et al. (2000, 2001) used RIMPUFF, developed time-dependent species-specific emission rates, and applied a virus inactivation rate and an infection probability function. They concluded that transmission to the UK was unlikely, as the predicted virus concentrations were about 500 times lower than the assumed threshold concentration to infect cattle. In a sensitivity analysis they showed that 1000 infected pigs were required to infect susceptible cattle up to 300 km downwind.

4.1.3. United Kingdom, 2001

In 2001 outbreaks occurred at 1849 farms across the UK (Gibbens et al., 2001). Several studies emphasised the important effect of topography, atmospheric stability, and low wind speeds on pathogen dispersion. Gloster et al. (2003) and Mikkelsen et al. (2003) used four models – the Gaussian dispersion equation, NAME, DERMA (Danish Emergency Response Model of the Atmosphere), and RIMPUFF. They determined specific emission periods and the areas at risk, and simulated dispersion, taking into account local topography effects. They explained seven out of 12 infected farms. Gloster et al. (2005a) also used multiple models – NAME, ADMS and DERMA – for comparison during very stable atmospheric conditions. They could explain airborne infections in two out of three epizootic clusters. However, in none of these studies relevant quantitative analyses were performed; a dose–response model was not used nor was the infection risk characterised.

Exposure from another potential source of FMDV, burning carcases, was investigated in a series of studies using NAME (Champion et al., 2002; Gloster et al., 2001; Jones et al., 2004). In one of them (Jones et al., 2004) an exponential dose–response model was used to estimate the probability of infections in cows and sheep downwind, being less than 0.3% and 0.04%, respectively. They concluded that infection from burning carcases was therefore unlikely. However, taking into account the presence of hundreds or thousands of livestock animals at several kilometres downwind of a plume, a few infections might have been possible (indeed, one farm was infected).

4.1.4. Other epizootics

Sørenson et al. (2000 , 2001) simulated dispersion from Germany to Denmark in 1982 using RIMPUFF and assumed emission from 1000

pigs. These emission rates combined with stable atmospheric conditions and favourable wind conditions gave high infection risks in Denmark. However, when fewer pigs were assumed or cattle or sheep were considered as a source, then the probability of infection decreased tremendously. Since the number of infected animals was chosen arbitrary, it is, however, arguable whether the conclusion will hold if the actual number of infected animals would have been lower, or cattle or sheep were shedding instead of pigs.

Traulsen et al. (2010) also used RIMPUFF and simulated another (unspecified) epizootic in Germany comprising 729 farms. They performed a risk factor analysis using Monte-Carlo simulations including data on emissions, farm locations, and livestock susceptibility. Significant correlations were found between modelled concentration, several time parameters, farm type, farm density, and control strategies. Although these results are plausible, technical details were lacking unfortunately.

In a subsequent study they investigated whether simple *fuzzy logic* could replace complex ADMs (Traulsen and Krieter, 2012). That is, they replaced numerical data (spatial coordinates, wind speed, atmospheric stability, and modelled concentrations) by factors (low/medium/high). Compared to a Gaussian dispersion model, the sensitivity and specificity rates were on average 81% and 97%, respectively. However, the authors assumed true predictions from the Gaussian model, technical details were lacking, meteorological data were still required for their analysis, and the converted modelled concentrations were simulated by an ADM.

Maragon et al. (1994) and Moutou and Durand (1994) used the ICAIR puff model to simulate FMDV spread in northern Italy in 1993 in a rather qualitative way. The areas at risk were small due to low wind speeds, a low humidity, and limited farm contact, but airborne transmission to nearby farms could potentially have occurred according to the model. Additional hypothetical emission from two pig farms resulted in "many more farms at risk". The model was proposed as a tool for decision-makers for future outbreaks, although the analyses were highly qualitative and no infection risks were calculated.

Schley et al. 2009 simulated an epizootic in the UK in 2007 using NAME. They marked four farms as primary sources and predicted the infection risks for all other farms. The sensitivity rate was about 67% (4 out of 6 farms); the specificity rate was approximately 92%.

Daggupaty and Sellers (1990) explained airborne infection at all twelve infected farms in Canada (1951–1952) using a Gaussian plume model, although additional infection routes (such as movement of persons and livestock) could not be excluded at six of the farms.

Finally, two epizootics in South Korea (2010–2011) were simulated with CALPUFF, of which one was explained by airborne transmission (Kritana et al., 2014). Transport of faeces and contaminated persons potentially increased infection probabilities. The authors used threshold values for inactivation by humidity to determine the most likely period of transmission. In addition, a smartphone application was highlighted for FMDV dispersion simulations, requiring data on serotypes, inactivation rates, and farms (location, livestock type, and livestock numbers) to simulate dispersion during field measurements.

4.1.5. Hypothetical simulations

Two series of studies were published discussing the possible consequences of FMDV outbreak in Australia and Austria. In Australia, the number of infectious units and virus deposited per hectare as a function of distance from a random initial source was simulated using the Gaussian plume equation (Cannon and Garner, 1999; Garner and Cannon, 1995). It was, simply, concluded that Australian weather conditions would not be a limiting factor for virus spread.

A more advanced risk assessment module was subsequently developed containing HYSPLIT, an intra-virus production model including five infectivity statuses (susceptible, latent, infectious, immune and death), and a binomial dose–response model (Garner et al., 2006; Hess et al., 2008). The number of farms at risk was calculated from a dose–response model. An additional sensitivity analysis showed that virus strain, pathogen inactivation and temperature would largely affect concentration; relative humidity was of minor importance (Hess et al., 2008).

In Austria, a Gaussian plume decision-support system for potential outbreaks was developed (Rubel and Fuchs, 2005), with a special focus on atmospheric stability, emission rates and wind speeds, where atmospheric stability was of great importance (concentrations at two kilometres from a potential source varied by a factor of 1,000 between unstable and highly stable conditions). The effect of wind speed was much smaller. Mayer et al. (2008) subsequently proposed an improved (Lagrangian) model for complex dispersion in the mountainous Austrian landscape. Case studies depicted a significant influence of local wind systems on the airborne spread. The authors concluded that the model was an appropriate tool for risk assessment of airborne virus spread. Although their ADM was more advanced than most other models described in this section, they only made a limited assessment of the risk by applying threshold doses.

4.1.6. Summary

- Quantitative analyses were only performed in a minority of studies. Most studies did not go further than geographical visualisation or determination of the number of farms possibly affected (using a threshold concentration or dose for infection). Only a few studies used a dose–response function and determined the sensitivity and specificity rate regarding the farms' infectivity statuses.
- Atmospheric stability and landscape topography were depicted as important factors influencing the surface concentrations and thus the exposure levels.
- Most studies focused on short-range transmission up to several kilometres. Long-range transmission could not be proven, although ADMs are suited for it.

4.2. *B. anthracis*

B. anthracis is a spore-forming bacterium causing anthrax in humans and animals through exposure to infected livestock or contaminated animal products (Pohanka and Skládal, 2009). Its spores are very resistant to extreme physical conditions, such as desiccation, heat, and disinfection (Shafazand et al., 1999). The incubation period is only two to six days (Pohanka and Skládal, 2009).

B. anthracis is a highly pathogenic bioterrorism-related agent (Anderson and Bokor, 2012). Outbreaks or releases have rarely been described, but some intentional releases caused much concern (Shafazand et al., 1999). In 1993, spores were aerosolised by a Japanese cult, although no one was infected (Keim et al., 2001). In 2001, a total of 22 cases were identified in the United States who were exposed to contaminated mail (Jernigan et al., 2002).

The number of publications regarding atmospheric dispersion modelling of *B. anthracis* is limited. Meselson et al. (1994) simulated the release from a military facility in the former Soviet Union in 1979 that had resulted in 77 human infections (of whom 66 were lethal), and death of livestock up to 50 km downwind. They estimated that approximately four billion spores had been released.

Due to its high pathogenicity and biosafety classification, multiple emergency preparedness models have been developed. Stuart and Wilkening (2005) simulated the dispersion of 10^{15} spores in an urban environment with the Gaussian dispersion equation and a dose–response model. The maximum distance of lethal infections varied from 25 km up to more than 200 km, dependent on the chosen decay function. The authors highlighted the need for predictive models to efficiently provide information during a crisis. A very similar analysis was performed in two other studies (Craft et al., 2005; Wein et al., 2003). Their emergency response tool also contained sub-models on

(age-dependent) dose–response, disease progression, antibiotic distribution and hospital care. They calculated an average probability of infection of about 65% at 200 km downwind. Buckeridge et al. (2006) developed a tool including a Gaussian plume model, a dose–response model, disease states, clinical visits, and pharmaceutical prescriptions. They estimated that the number of infected persons would range from 15,000 (0.01 kg anthrax) to 49,000 (1 kg). Nicogossian et al. (2011) used the Operational Multiscale Environment Model with Grid Adaptivity (OMEGA) model to simulate a hypothetical release of one million spores in the subway of Washington D.C. (USA), with subsequent dispersion in the outdoor environment. They concluded that a significant number of commuters and resident would have been exposed and being overload the existing health care infrastructure. Isukapalli et al. (2008) used CALPUFF for a hypothetical release of 10^{12} spores in an urban environment in New Jersey (USA) and accounted for activity patterns and physiological variability. Uncertainty analyses with respect to atmospheric conditions, population demographics, emission rate, and other characteristics were expressly recommended for future analyses. They also advised to include a source characterisation option in a comprehensive planning scheme for detecting bioterrorism-related releases. Finally, Tang et al. (2009) performed a meteorological flow field analysis to locate a source of a hypothetical anthrax release in an unsteady three-dimensional atmospheric wind field in an urban street canyon "with high accuracy".

Except for the latter publications, all these publications with emergency preparedness models have not only included a realistic quantified emission rate, but they also have performed a full quantitative microbial risk assessment, including the steps of (1) hazard identification, (2) dose–response relationships, (3) exposure assessment, and (4) risk characterisation.

4.2.1. Summary

- Only one retrospective simulation modelling study regarding the dispersion of *B. anthracis* was published.
- In several other studies a full emergency response model was developed taking all QMRA steps into account, including a quantified emission rate, an ADM and dose–response model, and clinical consequences.

4.3. Avian influenza virus

Influenza viruses are widespread and due to their high mutation rate many subtypes exist. Poultry farms are an important reservoir for the avian influenza virus (AIV) (Peiris et al., 2007), which play a critical role in the genesis of pandemic influenza viruses (Shortridge, 1992). AIV transmission to humans is largely facilitated by contact with animals and excretion of contaminated droplets or aerosols (Killingley and Nguyen-Van-Tam, 2013), and to a lesser extent through transport of (dead) birds or contaminated objects (vehicles, humans, or fomites), water, food, and contact with infected wildfowl or insects (Dent et al., 2008). Major outbreaks of avian influenza have occurred in China, Italy, the Netherlands, and Thailand (Areechokchai et al., 2006; Capua et al., 1999; Chan, 2002; Ellis et al., 2004; Koopmans et al., 2004; Stegeman et al., 2004; Tang and Chen, 2013).

The number of studies with AIV dispersion simulations is limited. Ssematimba et al. (2012) developed a Gaussian plume model and simulated the epizootic in the Netherlands in 2003. The authors highlighted the need for quantification of dispersion patterns to understand pathogen transmission between farms. By means of an exponential dose–response model they estimated that the airborne route accounted for 24% of new infections within 25 km of a source farm. That is, airborne AIV dispersion could have played a significant role in short-range transmission, but it could not completely explain long-range transmission. If, however, the epizootic had been reconstructed in time, then potentially the transmission from sources to susceptible farms, that subsequently would act as new source farms, might have been identified, thus potentially increasing the percentage. The results discussed in the sensitivity analyses were in accordance with the theory: wind speed (negative), deposition velocity (positive), emission height (negative) and inactivation (negative) influenced the probability of infection. These effects were largest at distances up to 2 km.

Seo and Lee (2013) and Seo et al. (2014) applied CFD modelling (Fluent) to the South Korean outbreak of 2008. The possibility of spread from each of the 39 farms to another was calculated as a function of wind direction and three different wind speeds. However, a thorough analysis on the results was not presented, although two other transmission networks were added in an additional study, namely a medicine and feed network, revealing that all contributed (Lee et al., 2014).

4.3.1. Summary

- The number of AIV studies in which an ADM was applied is limited.
- In one study the airborne route accounted for 24% of total transmission for distances up to 25 km.

4.4. L. pneumophila

Legionnaire's disease (or legionellosis) in humans is caused by a respiratory infection with the bacterium *L. pneumophila* (Leclerc et al., 2002). Inhalation of bacteria originating from natural fresh-water, potable water, cooling-towers or soil is the most likely cause of infection (Bovallius and Roffey, 1987). Large outbreaks have been associated to cooling-towers such as reported from Spain (6×), Australia, UK (3×), Italy, France, Sweden, US, New Zealand, Norway, Canada, the Netherlands, and Germany (Walser et al., 2014).

Legionella dispersion from cooling-towers was simulated in several studies. Nguyen et al. (2006) simulated an outbreak in France (2003–2004) with 86 human cases including 18 lethal infections using the ADMS model. The analysis was rather visual and qualitative: it was concluded that the model showed "good coverage of the municipalities where cases lived". We re-analysed the predicted concentrations and attack rates per municipality with a linear regression function in R (version 3.1.2) showing that the correlation was indeed positive, but not significant ($p \approx 0.12$).

A Norwegian outbreak (2005) with 56 human cases including ten lethal infections was modelled in a series of four studies. Nygård et al. (2008) performed a similar analysis to that of Nguyen et al. (2006) and used the Integrated PUFF model (INPUFF). An air scrubber at a biological treatment plant as source gave the best fit and resulted in the highest number of cases exposed. In three subsequent studies CFD modelling was applied to investigate whether Legionellae were indeed generated from that specific source (Blatny et al., 2011, 2008; Fossum et al., 2012). Modelling results were used to select optimal sampling sites in the area, thereby detecting bacteria up to 200 metres downwind (Blatny et al., 2008). Additional measurements on particle size distribution revealed that the majority of the bacteria were captured in either small (<4 μm) or large (>16 μm) size fractions (Blatny et al., 2011).

4.4.1. Summary

- A limited number of studies described dispersion modelling of *L. pneumophila*, all regarding industrial water units as source.
- ADMs were used in epidemiological studies to attribute the source of local epidemics.

4.5. C. burnetii

Q fever is a zoonotic disease caused by the bacterium *C. burnetii* (Parker et al., 2006). It is present worldwide with the exception of New Zealand. Ruminants are its main host (Angelakis and Raoult, 2010), which excrete the pathogen via their birth products, milk, faeces, and/or urine (Arricau Bouvery et al., 2003; Berri et al., 2002; Guatteo et al., 2007, 2006; Kim et al., 2005; Rodolakis et al., 2007; Van den Brom et al., 2012). Human infections occur from inhalation, leading to asymptomatic infections, (mild) clinical signs (e.g., fever and headache), more severe disease (pneumonia or hepatitis), or even mortality (Angelakis and Raoult, 2010; Dijkstra et al., 2012; Parker et al., 2006). Large outbreaks have occurred in countries including Canada, France, Germany, Italy, Slovakia, Switzerland, the Netherlands, the United Kingdom, and the United States (Dupuis et al., 1987; Georgiev et al., 2013; Hawker et al., 1998; Kováčová et al., 1998).

Despite its global abundance, the number of ADM studies regarding *C. burnetii* transmission is limited. An outbreak in the United Kingdom (2007) with 30 human cases was simulated to a limited extent with NAME to identify its source (Wallensten et al., 2010). Results showed that airborne transmission from all selected potential sources could have occurred. Extra difficulties arose due to a lack of emission data and timing of infection.

The outbreak in the Netherlands (2007–2010) was the largest Q fever epidemic ever described with over 4000 notified human cases. Infection was mainly associated with large dairy goat farms (Roest et al., 2011). Three regional epidemics were simulated with the Operational Priority Substance Short Term (OPS-ST) model (Sauter et al., 2011; Van Leuken et al., 2015). Due to the absence of quantified emission rate data, three simple emission profiles were defined and the best linear fit of the incidence–concentration function compared to a model with no predictors and one with distance as a single predictor were determined. In all three areas the ADM-concentrations correlated significantly better to the observed incidence than those of two simple models. Better emission rate parameterisations would improve the simulation modelling, thus allowing for a quantified risk assessment.

4.5.1. Summary

- The number of ADM studies simulating *C. burnetii* dispersion is very limited, despite the large number of epidemiological studies on Q fever.
- (Better) emission rate data would improve risk assessment.

4.6. Pseudorabies virus

Aujeszky's disease is caused by the Pseudorabies virus (PRV) (Mettenleiter, 2000) with domestic pigs and wild boar as principal hosts (Christensen et al., 1993; Ruiz-Fons et al., 2008). Outbreaks have occurred in countries including Denmark, Germany, Ireland, Poland, the United Kingdom, and the United States (Christensen et al., 1993; Gloster et al., 1984; Henderson et al., 1995; Müller et al., 2003; Obaldía, 2005; Salwa, 2004; Scheidt et al., 1991).

No recent studies on PRV dispersion modelling were found in the literature. Gloster et al. (1984) simulated PRV dispersion among eleven pig herds in the UK (1981–1982) using the Gaussian dispersion equation. They suggested that airborne transmission was possible in seven out of 11 herds. Transmission via other routes was classified as unlikely at the majority of the farms. As with the FDMV dispersion studies, a quantitative analysis (including dose–response models) was not applied.

Casal et al. (1997) re-analysed this epizootic and another in the United States (1988) among 10 pig herds using ALOHA and assumed constant wind speeds. The predicted number of infected animals was strongly dependent on the chosen wind speed. It was noted that in reality virus concentrations would not be homogeneous and the amount of virus inhaled is Poisson-distributed. Thus, despite a low mean dose, individual animals might have been infected. Furthermore, Grant et al. (1994) also analysed the U.S. outbreak with the Gaussian dispersion equation. During the outbreak, stable atmospheric conditions, strong winds and low temperatures occurred, which are favourable conditions for spread of airborne pathogens.

4.6.1. Summary

As with the FDMV dispersion studies, quantitative analyses were lacking. Although airborne spread was explained at most farms, the level of geographical visualisation or determination of the number of farms possibly affected was not exceeded.

4.7. Other pathogenic bioaerosols

4.7.1. Livestock and urban environments

Gloster (1983) analysed two outbreaks of Newcastle disease among poultry farms in the UK in 1959–1960 and 1969 with a Gaussian plume model (although, comparable to FMDV and PRV, rather limitedly). They classified the estimated daily virus doses as "very low". Infections up to 8 km from the initial source were explained.

Models based on Computational fluid dynamics (CFD) are suited to simulate dispersion in urban environments, which differ from rural environments due to large differences in turbulent conditions (Gao et al., 2008). The Severe Acute Respiratory Syndrome (SARS) virus and the human influenza virus are typical urban viruses with human–human transmission. Yu et al. (2004) used Fluent to simulate a SARS virus outbreak in Hong Kong in 2003 with 187 cases. Virus was excreted from a bathroom and transported outdoor by an exhaust fan. Modelled exposure correlated significantly to the expected exposure (homes of cases) in six nearby buildings. Liu and You (2012) simulated human influenza virus transmission among five apartment buildings. Virus particles were dispersed tens of metres downwind, thereby leading to a "high risk of secondary infection in large areas". However, technical details and extended analyses were lacking.

4.7.2. Wastewater

In several studies measured concentrations close to wastewater treatment plants (WWTP) were compared to predicted ADM concentrations. In some studies ADMs were even used to quantify the contamination of the surrounding environment. For instance, Stellacci et al. (2010) performed a QMRA and simulated the dispersion of *Cryptosporidium, Campylobacter*, and rotavirus in the surrounding of an Italian WWTP. Concentrations at 100 m downwind were lower than the limits for drinking water. Dungan (2014) performed a QMRA on exposure to *Campylobacter jejuni, Escherichia coli, Listeria monocytogenes*, and *Salmonella* spp. from irrigation of diluted dairy wastewater. They used the AMS/EPA Regulatory Model (AERMOD) and found maximum relative exposure risks of −5.8 and −0.1 (^{10}log) at 1000 m downwind (both for *C. jejuni*), assuming inactivation rates of 0.07 s^{-1} (day) and 0.002 s^{-1} (night), respectively. It was recommended scheduling irrigation events to day-time, given the higher wind speeds causing more dilution, and higher inactivation rates due to desiccation and ultraviolet light exposure.

Furthermore, Sorber et al. (1976) detected coliform bacteria, including *Klebsiellae* and faecal *Streptococci* in air samples near a WWTP in Arizona (USA) and used a Gaussian plume model for concentration predictions. By means of a linear regression model (R) we re-analysed their data showing that a high correlation ($r = 0.72$) existed between the measured and modelled data, although borderline statistically significant ($p = 0.07$).

Teltsch et al. (1980) detected *E. coli* in air samples near an Israelian WWTP and used the Gaussian dispersion equation for prediction modelling. A highly significant correlation was found ($r = 0.93$, $p \approx 0.00$). Holden and Babcock (1985) measured total viable particle concentrations near a WWTP to retrieve an emission rate using

the Gaussian dispersion equation for back-calculation. However, they found a positive inactivation rate, indicating the contribution of other sources. Li et al. (2013) measured mesophilic bacteria concentrations at several points downwind of a WWTP rotating-brush aerator and determined emission rates. They used a Gaussian plume model (Dowd et al., 2000) and created source depletion curves, resulting in "acceptable low risk values at various downwind distances". Peterson and Lighthart (1977) analysed exposure to municipal wastewater from cooling towers. They discussed the effects of particle size, source height, wind speed, inactivation, and atmospheric stability on airborne concentrations.

4.7.3. Biosolids

Aerosolisation of pathogens from biosolid material, such as domestic sewage sludge or compost, may also occur. Rotavirus, coronavirus, *Salmonella* spp., and *E. coli* were detected at various distances from a field at which sewage sludge was applied as fertiliser (Dowd et al., 2000). Concentrations were predicted with the Gaussian dispersion equation and converted to infection risks as a function of exposure duration and various wind speeds, yielding maximum risks of 37.3% (bacteria) and 100% (viruses) (given 24-h exposure at 100 m distance) (unfortunately, confidence intervals were not given).

Furthermore, emission rates of mesophilic actinomycetes and the fungus *Aspergillus fumigatus* from static compost windrows were determined in a series of studies (Taha et al., 2005, 2006, 2007). A wind tunnel was used to determine emission rates; the models SCREEN3 and AMDS were used to generate concentration profiles as a function of distance. The concentrations reduced to background values of 1000 CFU/m^3 within 100–250 m of the source site, corresponding to legal requirements. Low et al. (2007) compared Clostridia, Chloroflexi, and Euryarchaeota concentrations downwind of a land application site in Arizona (USA) with concentrations predicted with the Gaussian dispersion equation. The Gaussian model was classified as adequate for predicting concentrations downwind from the site.

4.7.4 . Other

Finally, several ADM studies had no specific sources included. Lighthart and Mohr (1987) investigated reovirus (Respiratory Enteric Orphan virus) concentrations with a Gaussian plume model and concluded that wind speeds largely influenced the source depletion curves, thereby being 'potentially important' as a dilutor. Lin et al. (2014) measured and predicted (using a Gaussian plume model) *Fusarium* concentrations. Concentrations were generally lowest in winter. Sprigg et al. (2014) simulated dispersion of the fungi *Coccidioides immites* and *Coccidioides posadasii*, the causative agents of human valley fever. They used the Dust Regional Atmospheric Model (DREAM) and could associate the occurrence of a large dust storm and specific vegetation and land cover data to a Californian epidemic in 2011 with 3600 cases.

The abundance and diversity of airborne pathogenic and non-pathogenic bacteria and fungi at 2700 m altitude were measured in the United States (Smith et al., 2012, 2013). They created back-trajectories using HYSPLIT and concluded that the microorganisms originated from China or Japan. Thus, after 10 days travelling across the Pacific Ocean, viable microorganisms were still detected.

4.7.5. Summary

- Dispersion in urban environments was usually modelled with CFD techniques. Dispersion at field sites was usually modelled with Gaussian models.
- In addition to sources related to livestock and urban environments, exposure from WWTPs and biosolids was modelled. A few studies focused on exposure from WWTPs also performed a QMRA.
- In several studies actual infection risks were calculated with dose–response models.

5. Model parameterisations

5.1. Emission

Three pragmatic approaches were identified from the studies discussed in Section 4, namely the use of:

- Arbitrary emission data, thus leading to relative concentration maps (Fossum et al., 2012; Gloster et al., 1984; Lighthart and Frisch, 1976; Sorber et al., 1976; Van Leuken et al., 2015; Wallensten et al., 2010).
- Realistic assumptions, such as a release of 10^{15} *B. anthracis* spores (Craft et al., 2005; Isukapalli et al., 2008; Nicogossian et al., 2011; Stuart and Wilkening, 2005), or the use of morbidity, severity and duration of the disease as a proxy for emission (Gloster, 1983) – although the reliability may be arguable.
- Varying emission rates through sensitivity analyses (e.g., Buckeridge et al., 2006)

In all other studies measurements (Section 5.1.1) or emission models (Section 5.1.2) were applied to determine emission rates.

5.1.1. Measurements

The simplest method to retrieve emission rates from measurements is by determining a concentration and flow rate:

$$Q = C \cdot U \cdot A \tag{7}$$

where Q is the emission rate [pathogen amount per second], C is concentration [pathogen amount per m^3], U is wind speed [m/s], and A is the cross-sectional area from which the pathogen is released [m^2]. An alternative method to determine the flow rate (U•A) is by using the eddy covariance technique, that is used to measure vertical turbulent fluxes (e.g., Kormann et al., 2001)

Holden and Babcock (1985), who focused on pathogen release from a WWTP, found a rate of approximately 20,000 aerosols per second using Eq. (3). Similar assessments were performed by Dowd et al. (2000) and Taha et al. (2006).

Taha et al. (2005) determined a compost windrow emission rate of *A. fumigatus* through a wind tunnel experiment using:

$$Q = \phi \cdot \frac{C}{A} \tag{8}$$

where ϕ is the flow rate [m^3 s^{-1}]. Note that in fact, Eqs. (3) and (4) are similar, except for that Eq. (4) describes the emission per surface unit instead from a point source.

Paez-Rubio et al. (2007) incorporated multiple measurements of total bacteria, total coliforms, *Clostridia*, and endotoxins in a vertical column (Holmén et al., 2001):

$$Q = \int_{z_0}^{h} \frac{U(z) \cdot C(z) \cdot t \cdot \cos(\theta)}{W} dz \tag{9}$$

which is in fact an extended version of Eq. (4). Eq. (5) is an integration in height from roughness length z_0 [m] to the top of the plume h [m], taking into account height-dependent wind speed and concentration data. The emission rate was scaled by time t [s], the angle between wind direction and field edge (θ), and the width of the column W [m].

Finally, emission rates were derived from measurements at one or several distances from a source that were incorporated into a dispersion model. Li et al. (2013) measured concentrations of mesophilic bacteria at two, five and ten metres downwind of a WWTP and thus determined Q. Low et al. (2007) calibrated the Gaussian dispersion equation by comparing modelled and measured concentrations at five metres from a bio-solid source.

There was no livestock-related ADM study in which measurements were incorporated.

5.1.2. Pathogen production models

Pathogen production models are useful in case of livestock-borne pathogens. For FDMV an intra-farm virus production model was developed for cattle, sheep and pigs, giving the virus amount emitted per infected premise per day (Sørenson et al., 2000, 2001). It was used in several dispersion studies (Gloster et al., 2005b; Mikkelsen et al., 2003; Traulsen et al., 2010). A major disadvantage, however, is the lack of dynamics within a population, which herd dynamics models include. Such models are based on multiple infectivity states, such as 'susceptible', 'latent' (infected but not yet infectious), 'infectious', 'recovered', or 'dead'. Herd dynamics models calculate the number of individuals in and the transfer rate between each compartment per time unit, where the reproduction ratio R_0 is defined as the average number of secondary infections caused by one infectious individual introduced in a naive population. The infection will fade out if $R_0 < 1$ (either naturally or enforced, e.g., by vaccination) (Bouma, 2005; Hogerwerf et al., 2011); if $R_0 = 1$, the infection will be sustained and if $R_0 > 1$ an epidemic will occur.

Garner et al. (2006) and Hess et al. (2008) used a Rinderpest population model (James and Rossiter, 1989), which applies Monte-Carlo simulations and gives the number of susceptible, (partially) immune, affected, and mildly affected cattle and wildlife, and allows for vaccination intervention. Specific FMDV data regarding the length of the latent, infectious, symptomatic, and excretion period, mortality rate, and maximum daily virus production were retrieved from literature (Alexandersen et al., 2003; Sørenson et al., 2000).

Examples of other population dynamic models (although not used for ADM simulations) are those for *C. burnetii*, the SARS virus, and the Porcine Reproductive and Respiratory Syndrome virus (Courcoul et al., 2011; Hogerwerf et al., 2013; Naheed et al., 2014; Nodelijk et al., 2000).

5.2. Inactivation

In the ADM studies, either threshold values related to environmental conditions (Section 5.2.1) were used, or inactivation rates as a function of time were applied (Section 5.2.2). Therefore, to improve risk assessment, much more effect should be invested to determine pathogen specific inactivation parameterisations as a function of environmental conditions.

5.2.1. Threshold values

Many studies, primarily those that focused on FMDV, applied threshold inactivation values. Historical measurements showed that FMDV survival was highest for a relative humidity > 60%, reduced for 20–60%, and very small for < 20% (Donaldson, 1972). There is also a (qualitative) description on the effect of temperature: FMDV would survive for "long periods" at "low" temperatures and for "considerable periods" at temperatures in the range of 20–27 °C (Donaldson, 1972; Donaldson and Ferris, 1975; Gloster et al., 2005a).

These values were used in several studies. Kritana et al. (2014) used the threshold value for relative humidity to determine the most likely period of transmission in a sequence of days. Gloster et al. (2005a) assessed the FMDV viability in a qualitative way given the observed temperature; and Cannon and Garner (1999) assessed the probability of a major FMDV outbreak given the climatic conditions in Australia. That is, they concluded that weather conditions would not be a limiting factor for airborne FMDV spread (unfortunately, technical details are lacking).

In several other FMDV studies modelled concentrations were set to zero in case of a relative humidity < 60% (Casal et al., 1995; Gloster et al., 1981). A similar approach was applied to PRV concentrations (Grant et al., 1994): 100% survival in case of a relative humidity >85%, no survival for <25% and a linear survival curve for 25–85%.

5.2.2. Rates

In many other studies inactivation was expressed as a rate, i.e. as a decrease in time (cf. Eq. (2)). Rates for rotavirus (2.86×10^{-2} s^{-1}), coronavirus (2.66×10^{-2} s^{-1}), *Salmonella* spp. (2.35×10^{-4} s^{-1}), and *E. coli* (1.92×10^{-4} s^{-1}) were used in a wastewater simulation study (Dowd et al., 2000). Sørenson et al. (2000) proposed a rate for FMDV inactivation (3.2×10^{-4} h^{-1}, or 8.9×10^{-8} s^{-1}), which, however, might be very small compared to historical measurements (Donaldson, 1972). Nevertheless, it was adopted by several other studies (Garner et al., 2006; Hess et al., 2008; Traulsen et al., 2010).

Ssematimba et al. 2012 assumed an AIV inactivation rate of 2.89×10^{-6} s^{-1}. Additional sensitivity analyses (with rates from 4.0×10^{-7} to 2.0×10^{-6} s^{-1}) showed that the effect on infection risk was about 10–20%. Lighthart and Frisch, (1976) and Peterson and Lighthart (1977) incorporated higher decay rates of 0, 0.001, 0.01, and 0.1 [s^{-1}] in their Gaussian model description. They showed that, for the two highest decay rates, the majority of the viable cells were inactivated within 1–10 km from the source. Li et al. (2013) assumed rates of 0.0, 4.0×10^{-3}, 6.0×10^{-3}, 0.02, and 0.12 s^{-1} and created source depletion curves in their WWTP investigation. The highest inactivation rate resulted in a four times lower concentration compared to the lowest rates.

For *B. anthracis* four exponential decay models were proposed (Stuart and Wilkening, 2005), despite *B. anthracis* being very persistent. These models were defined as a function of time with an inactivation rate of 1.67×10^{-4} s^{-1} (although the actual inactivation rate is very much dependent on the amount of UV radiation and ozone concentration (Spotts Whitney et al., 2003)).

6. Probability of infection

The simplest technique to assess a risk (comparing doses to threshold values) was applied in many FDMV studies, e.g., 0.06 (cattle), 1.11 (pigs), and 7.70 (sheep) $TCID_{50}/m^3$ (Gloster et al., 2005a, 2005b, 2010, 2003, , 2001; Mikkelsen et al., 2003; Rubel and Fuchs, 2005; Sørenson et al., 2000; Traulsen and Krieter, 2012). $TCID_{50}$ is the median dose to infect a tissue culture; however, the actual meaning of this measure can be disputed, since it represents a probability of infection and not a concentration or dose. Furthermore, threshold values were also used in several other studies (Blatny et al., 2011; Cannon and Garner, 1999; Casal et al., 1997; Champion et al., 2002; Daggupaty and Sellers, 1990; Donaldson et al., 1987; Gloster et al., 1981; Lee et al., 2014; Seo et al., 2014; Traulsen et al., 2010).

Several other papers describe the application of dose–response models (see Section 3 and Appendix A). With respect to FMDV, the binomial model was used in two Australian studies (Garner et al., 2006; Hess et al., 2008), with the single-hit probability being equal to 0.031 (cattle), 0.045 (sheep), and 0.003 (pigs). The exponential model was used for risk assessment of burning carcases (Jones et al., 2004) and for analyses of the 1966–1968 and 2007 epizootics in the UK (Sanson et al., 2011; Schley et al., 2009).

For *B. anthracis* an empirical age-dependent model was incorporated in an emergency response model (Wein et al., 2003) (although empirical models lack biological physical basis, so they cannot be extrapolated to domains outside the domain of interest):

$$P_{inf}(D, a) = \Phi\left(\gamma + \delta \log(D) + \epsilon a + \zeta a^2\right) \tag{10}$$

where Φ is the standard normal cumulative distribution function, and a is the age [years]. An alternative age-dependent model was used in a follow-up paper (Craft et al., 2005):

$$P_{inf}(D, a) = \min\left(1, \frac{D}{c_1 - c_2 a}\right) \tag{11}$$

Stuart and Wilkening (2005) and Isukapalli et al. (2008) used comparable functions in their anthrax emergency preparedness models.

Ssematimba et al. (2012) applied an exponential logit model to estimate the risk of AIV infections:

$$P_{inf}(D) = [1 + \exp(\eta + \kappa \cdot D]^{-1} \quad (12)$$

where η and κ are shape parameters.

Finally, Dowd et al. (2000) used the exponential (rotavirus and coronavirus) and beta-Poisson model (*Salmonella* sp. and *E. coli*) in their biosolids risk assessment. Dungan (2014) applied the beta-Poisson model to dispersion of *C. jejuni, E. coli, L. monocytogenes*, and *Salmonella* sp. Stellacci et al. (2010) applied it to their risk assessment of *Cryptosporidium, Campylobacter* and rotavirus dispersion from a WWTP.

7. Conclusions

In this review we discussed studies modelling the dispersion of bioaerosols that are pathogenic to humans and animals, with a special focus on risk assessment. The choice for a specific type of atmospheric dispersion model (ADM) – Gaussian, Eulerian, or Lagrangian – depends on the spatial scale of interest, the complexity of the analysis, and one's preference for forward or backward analysis. For instance, Gaussian plume models neglect the heterogeneity of a complex wind field, while models based on computational fluid dynamics (CFD) simulate dispersion at a high three-dimensional resolution.

Transmission routes included human-human, livestock-livestock, livestock-human, and industrial-human. Short-range (several kilometres) transmission was indicated in many studies, however, solid evidence for long-range (tens to hundreds of kilometres) transmission was not found. In order to predict exposure levels as accurately as possible, high-resolution data on wind speed, wind direction, atmospheric stability, and topography are essential for dispersion modelling, and humidity, temperature, and ultraviolet radiation are crucial for modelling inactivation.

Parameterisations for re-aerosolisation were not included in the studies reviewed, although re-aerosolisation could result in additional exposure. That is, it may occur particularly in case of a high degree of contamination of the environment. As a result, additional (environmental) sources may contribute to the total exposure.

In addition, we have not found studies with quantified and substantiated choices for a specific particle size distribution profile (although several ADMs have included options for a particle size distribution, the choice for a specific profile is crucial. For instance, virus particles are much smaller and lighter than spores and are thus they are transmitted much further (when neglecting inactivation).

A major drawback of a majority of the studies was the lack of quantitative analyses and application of a full quantitative microbial risk assessment (QMRA) (including dose–response functions). In particular, (qualitative) conclusions solely based on dispersion maps, threshold doses and expert judgement were frequently encountered.

Examples of full emergency preparedness models were only found in *B. anthracis* dispersion studies. To improve risk assessment for other outbreaks and releases, it is highly recommendable developing such models for other pathogens as well. They would not only include an ADM, but also (1) well-quantified emission and inactivation rates, (2) estimated doses based on exposure duration, breathing rate, lung volume, and particle size distribution, and (3) dose–response models to estimate infection probabilities. Inactivation and emission rates are crucial, and should be quantified whenever possible. Then, such full risk assessment models will not only estimate the areas at risk qualitatively, but also quantify the expected health outcome in the human population or livestock farms.

Declaration of interest statement

None.

Acknowledgements

None.

Appendix A

Table A.1.

Appendix B

Scopus search query (d.d. October 10, 2014).

TITLE-ABS-KEY((((atmospheric* OR airborne OR aerial) W/3 (model* OR dispersion OR dispersal OR simulat*)) OR (predict* W/3 spread*) OR (wind* W/3 (model* OR simulat*)) OR "gaussian puff" OR "gaussian plume" OR (plume W/3 model*) OR (puff W/3 model*) OR lagrangian OR euler* OR cfd OR "computational fluid dynamic*" OR computational-fluid-dynamic*) **AND** (pathogen* OR microorganism* OR "micro-organism*" OR microbial* OR bioaerosol* OR "bio-aerosol*" OR "viable aerosol*" OR *virus* OR *bacter* OR *fever* OR *virinae* OR *viridae* OR *microbium* OR *microbia* OR fungus OR fungi OR zo$notic* OR zo$nos* OR endotoxin* OR *spore* OR esbl "a(h1)" OR "a(h1n1)" OR "a(h3)" OR "a(h5)" OR "acquired immunodeficiency syndrome" OR "acute respiratory infection" OR "bloodstream infection" OR "bovine spongiform encephalopathy" OR "bubonic plague" OR "carneocephallus brevicaea" OR "creutzfeldt jakob" OR "e. coli" OR "fibricola seoulensis" OR "foot and mouth" OR "genital wart" OR "gongylonema pulchrum" OR "loboa loboi" OR "lymphogranuloma venereum" OR "meticillin resistant staphylococcus aureus" OR "multiple eschars" OR "rift valley" OR "toxic shock syndrome" OR "west nile" OR *bacill* OR *encephalit* OR *herpes* OR *influenza* OR *meningitis* OR *tubercul* OR abiotrophi* OR absettarov OR absidi* OR acanthamoeb* OR acanthocephal* OR acanthopodid* OR achillurban* OR acidaminococc* OR aconoidasis* OR acremon* OR acrophialophora* OR actinomadur* OR actinomyc* OR aedes OR aerococc* OR aeromon* OR aeromonad* OR afipi* OR agarical* OR agaricomyc* OR agrococc* OR agromyc* OR ajellomyc* OR alaria* OR alcaligen* OR alternar* OR amapar* OR amoebid* OR amoebozo* OR amphimer* OR amycolatops* OR anaerococc* OR anaplasm* OR anatrichosom* OR ancylista* OR ancylostom* OR angiostrongyl* OR anisaki* OR anoplocephalid* OR anoplur* OR anseriforme* OR anserina* OR anthrax OR aonchothec* OR aphanoasc* OR apiospor* OR apophall* OR apophysomyc* OR arachnomyc* OR archamoeb* OR archea OR archiacanthocephal* OR arthrin* OR arthrini* OR arthroderm* OR artiodactyl* OR artyfechinostom* OR ascari* OR ascocotyl* OR ascomycot* OR aspergill* OR asthma OR astigmat* OR aureobasidi* OR aureobasidi* OR austrobilharzi* OR babesi* OR balamuthi* OR balantidi* OR bartonell* OR basidiobol* OR basidiomyc* OR basipetospor* OR baylisascar* OR beauveri* OR bergeyell* OR bertiell* OR bilharziell* OR bilophil* OR bipolari* OR blastocyst* OR blastomyc* OR bluetongue OR bolbosom* OR bordetell* OR borreli* OR bosea OR botryomyc* OR botryosphaeri* OR botryt* OR botulism OR brachyspir* OR brevundimon* OR brucell* OR burkholder* OR candida* OR capnocytophag* OR cathaemasi* OR cedece* OR cellulomon* OR centrocest* OR cephaliophor* OR cephalospor* OR cephalotrich* OR cercospor* OR cerinoster* OR chaetomi* OR chaetophom* OR cheilospirur* OR chikungunya OR chiroptera* OR chlamyd* OR chlamydophil* OR chlorococcal* OR chloroflex* OR choanephor* OR choanozo* OR cholera OR chromelospor* OR chroococc* OR chryospor* OR chryseomon* OR chrysonil* OR chrysospor* OR ciliophor* OR citrococc* OR cladophialophor* OR cladorrhin* OR cladospor* OR clavicipit* OR clavispor* OR clinostom* OR clinostomatid* OR clonorch* OR clostrid* OR coccidioid* OR coccodin* OR cochliobol* OR cokeromyc* OR coleophom* OR colletotrich* OR collinsell* OR comamona* OR conidiobol* OR coniochaet* OR coniothyr* OR conoidasid* OR contracaec* OR coprin* OR cordyc* OR corynespor*

Table A.1

Overview of all atmospheric pathogen dispersion studies discussed in this review, including their main characteristics: pathogen, study type (hypothetical outbreak, model analysis, outbreak, simulation, simulation and measurements), country and year of investigation, pathogen source, model type and model name, meteorological data, inclusion of deposition (Dep.) and particle size distribution (PSD), trajectory type (B = backward, F = forward), emission parameterisation, inactivation parameterisation, and type of dose response model used. (#) = unspecified, (–) = not relevant, (@) = assumptions. All abbreviations are explained in Table 1.

Paper	Pathogen	Type	Country of investigation	Year	Source(s)	Model type (model name)	Meteorological data (institute)	Emission data	Dep.	PSD	Traj.	Inactivation	Dose–response model
Blackall and Gloster (1981)	FMDV	Outbreak	UK	1966-1967	Cattle, pigs, sheep	Gaussian dispersion equation	Local station(s) (Met Office)	Yes (#)	–	––	–	Inactivation for RH < 60%	Threshold (#)
Blatny et al. (2008)	*L. pneumophila*	Simulation and measurements	Norway	2006	Wastewater	CFD (ANSYS-Fluent)	An unspecified NWP (NMI)	(#)	#	#	–	–	–
Blatny et al. (2011)	*L. pneumophila*	Simulation and measurements	Norway	2007	Wastewater	CFD (ANSYS-Fluent)	In situ	Yes (#)	Yes	Yes	F	–	–
Buckeridge et al. (2006)	*B. anthracis*	Hypothetical outbreak	USA	2001–2003	Urban	Puff (HPAC)	Local station(s) (#)	1 kg, 0.1 kg, 0.01 kg	#	#	–	–	Probit model (Glassman, 1966)
Cannon and Garner (1999)	FMDV	Hypothetical outbreak	Australia	1940–1995	Cattle, pigs, sheep	Gaussian dispersion equation	Stations across Australia (ABM)	1.8e5 (cattle), 1.5e5 (sheep), 2.8e8 (pigs) [IU/day]	Yes	Yes	–	Inactivation for RH < 60% or > 27 °C	Binomial model: $r = 0.03$ (cattle) and 0.06 (sheep)
Casal et al. (1995)	FMDV	Outbreak	UK	1967	Pigs	Plume (ALOHA)	@	4e3 (pig), 85 (cattle), 66 (sheep) [ID_{50}/min]. Farm-level: 16e3 ID_{50}/min	–	–	–	Inactivation for RH < 60%	#
Casal et al. (1997)	FMDV, PRV	Outbreak	UK, USA	1967, 1981–1982, 1988	Cattle, pigs	Plume (ALOHA)	#	FMDV: 5.1 (cattle) and 6.8 (pigs) [$\log_{10}$ $TCID_{50}$/animal/day]. PRV: 5.3 $\log_{10}$ $TCID_{50}$/animal/day	–	–	–	Inactivation for RH < 55%	Threshold: FMDV: 10 (cattle) and 400 (pigs) $TCID_{50}/m^3$. PRV: 1 $TCID_{50}/m^3$
Champion et al. (2002)	FMDV	Outbreak	UK	2001	Burning of animal carcasses on open pyres	Puff (NAME)	Unified model (Met Office)	6.5 $\log_{10}$ $TCID_{50}$ per pyre during 3 hours	Yes	Yes	–	–	Threshold: 0.06 $TCID_{50}/m^3$
Craft et al. (2005)	*B. anthracis*	Model analysis	USA	–	Urban	Plume (Wein et al., 2003)	@	1e15 spores (1 kg)	–	–	–	–	Age-dependent model: $P(D, a) = \min(1, \frac{D}{c_1 - c_2 a})$, a = age [years] [$c_1 = 38{,}000$ and $c_2 = 450$]
Daggupaty and Sellers (1990)	FMDV	Outbreak	Canada	1951–1952	Cattle, pigs, sheep	Plume (#)	# (Canadian Climate Center)	3.23e3 (pigs), 1.98 (cattle, sheep) [IU/s]	–	–	–	Inactivation for RH < 60%. Temperature was always < 2.8 °C	Threshold: 1 IU (cattle)
Donaldson et al. (1982)	FMDV	Outbreak	France, UK	1981	Cattle, pigs	Plume (Gloster et al., 1981)	Local station(s) (Met Office)	#	–	–	–	–	Threshold: 1 and 0.01 IU (cattle)
Dowd et al. (2000)	Rotavirus, Coronavirus, *Salmonella* sp., *E. coli*	Simulation with measurements from previous work (Dowd et al., 1997)	USA	1995	Biosolids from wastewater	Gaussian dispersion equation	Sensitivity analysis	Point source: 1.974e6 (*Salmonella*), 27 (virus). Area source: 5.11e6 (*Salmonella*), 750 (virus)	–	–	–	Rates: 2.86e−2 (rotavirus), 2.66e−2 (coronavirus), 2.35e−4 (*Salmonella* sp.), 1.92e−4 (*Escherichia coli*)	Viruses: exponential model [$r = 39.5$]. *Salmonella* sp.: beta-Poisson model [$\alpha = 23{,}000$, $\beta = 0.3126$]
Dungan (2014)	*C. jejuni*, *E. coli* (O157:H7 and non-O157), *L. monocytogenes*, *Salmonella* spp.	Simulation	USA	2000–2004	Wastewater	Plume (AERMOD)	Local stations(s) (NOAA), MM5	27–3.2e6 cells/s	Yes	Yes	–	Rate: 0.002 and 0.07 s^{-1}	Beta-Poisson model for *C. jejuni* [α = #; β = #], *E. coli* O157:H7 [$\alpha = 0.0571$; $\beta = 2.2183$], *E. coli* non-O157 [#], *L. monocytogenes* [$\alpha = 0.49$; $N_{50} = 5.96e5$], and *Salmonella* sp. [#]
Fossum et al. (2012)	*L. pneumophila*	Simulation and measurements	Norway	#	Wastewater	CFD (ANSYS-Fluent)	In situ	#	Yes	Yes	F	–	–
Gao et al. (2008)	SARS virus	Simulation and measurements	China (Hong Kong)	2005	Urban	CFD (ANSYS-Fluent)	@	#	#	#	–	Yes (#)	Exponential model
Garner et al. (2006)	FMDV	Hypothetical outbreak	Australia	2003–2004	Cattle, pigs, sheep	Particle mode (HYSPLIT)	LAPS (ABM)	Modified version of an intra-farm virus model (James and Rossiter, 1989)	Yes	Yes	F	Rate: 6.4e−4 * 0.5 h^{-1}	Binomial model: $r = 0.031$ (cattle), 0.045 (sheep), 0.003 (pigs)
Gloster et al. (1981)	FMDV	Outbreak	UK	1966, 1967	Cattle, pigs, sheep	Gaussian dispersion equation	Local station(s) (Met Office)	8 (pigs), 5 (cattle, sheep) [$\log_{10}$ IU/animal/day]	–	–	–	Inactivation for RH < 60%	Threshold: 1 IU

(*continued on next page*)

Table A.1 (*continued*)

Paper	Pathogen	Type	Country of investigation	Year	Source(s)	Model type (model name)	Meteorological data (institute)	Emission data	Dep.	PSD	Traj.	Inactivation	Dose–response model
Gloster (1983)	Newcastle disease virus	Outbreak	UK	1969	Poultry	Plume (Blackall and Gloster, 1981)	Local station(s) (Met Office)	Proportional to the morbidity, severity and duration of the disease in a flock	–	–	–	–	–
Gloster et al. (1984)	PRV	Outbreak	UK	1981–1982	Pigs	Gaussian dispersion equation	Local station(s) (#)	#	–	–	–	–	–
Gloster et al. (2001)	FMDV	Outbreak	UK	2001	Burning of animal carcasses on open pyres	Puff (NAME)	Unified model (Met Office)	6.5 $\log_{10}$ $TCID_{50}$ per pyre	Yes	Yes	–	–	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Gloster et al. (2003)	FMDV	Outbreak	UK	2001	#	Plume (Gloster et al., 1981), Puff (DERMA, NAME, RIMPUFF)	Local station(s) (Met Office), Unified Model, HiRLAM	(Alexandersen et al., 2003)	Yes	Yes	–	Inactivation for RH < 60% or > 21 °C	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Gloster et al. (2005a)	FMDV	Outbreak	UK	2001	Cattle, pigs, sheep	Puff (NAME)	Local station(s), Unified Model (both Met Office)	(Alexandersen et al., 2003)	Yes	Yes	–	Inactivation for RH < 60% or > 27°C, moderate survival for 20–27 °C.	Threshold: 0.06 $TCID_{50}/m^3$ (cattle)
Gloster et al (2005b)	FMDV	Outbreak	UK	1967–1968	Pigs	Plume (ADMS)	Local station(s) (Met Office)	Virus model (Sørenson et al., 2000)	Yes	Yes	–	Inactivation for RH < 60%	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Gloster et al. (2010)	FMDV	Outbreak	UK	1967	Cattle, pigs	Puff and Particle mode (CALPUFF, HYSPLIT, MLCD, LODI, NAME, RIMPUFF)	Local station(s) (Met Office), unspecified NWP's	Varying: max. 8 $\log_{10}$ $TCID_{50}$/day	Yes	Yes	–	Yes (#)	Threshold: 0.06 $TCID_{50}/m^3$ (cattle)
Grant et al. (1994)	PRV	Outbreak	USA	1988	Pigs	Gaussian dispersion equation	Local station(s) (#)	5.0–6.3 $\log_{10}$ TCID per herd per day	–	–	–	Inactivation for RH < 25% RH, linear inactivation function for RH 25–85%	–
Hess et al. (2008)	FMDV	Hypothetical outbreak	Australia	2004	Pigs	Particle mode (HYSPLIT)	LAPS (ABM)	Modified version of an intra-farm virus model (James and Rossiter, 1989)	Yes	Yes	–	Rate: 6.4e−4 * 0.5 h^{-1}	Binomial model: $r = 0.031$ (cattle), 0.045 (sheep), 0.003 (pigs)
Holden and Babcock (1985)	#	Simulation and measurements	USA	1977	Wastewater	Gaussian dispersion equation	In situ	22,234 [<2 m/s wind speed]; 22,127 [2.1–5.9 m/s]; 19,556 [>6 m/s] [particles/s]	–	–	–	–	–
Isukapalli et al. (2008)	*B. anthracis*	Hypothetical outbreak	USA	2001	Urban	Puff (CALPUFF)	# (NOAA)	(a) 100 g/1 h (b) 100 g/10 h (100 g ~ 1e12 spores)	Yes	Yes	–	Yes (#)	Other DR-models (Craft et al., 2005, Wein et al., 2003) and: (I) $P(D,a) = \Phi(\alpha + \beta \log(D))$ [$\alpha = -2.6361$, $\beta = 0.291$] (variation 1), [$\alpha = 5.6263$, $\beta = 0.621$] (variation 2, ~ ID_{50} = 8600 spores) (II) $P(D,a) = \exp(-\frac{\lambda D}{\lambda+\theta})$ [θ=0.109/day, $\lambda = 8.8e{-}8\ s^{-1}$] (III) $P(D,a) = \frac{\beta\cdot(\exp(\frac{D}{\alpha})-1)}{1+\beta\cdot\exp(\frac{D}{\alpha})-1}$
Kritana et al. (2014)	FMDV	Outbreak	South Korea	2010–2011	Cattle, pigs	Puff (CALPUFF)	Weather Research and Forecasting model	4.3 (cattle) and 6.1 (pigs) $\log_{10}$ $TCID_{50}$/animal/day	Yes	Yes	–	Inactivation for RH < 60% or > 30°C	–
Lee et al. (2014)	AIV	Outbreak	South Korea	2008	Poultry	CFD (ANSYS Fluent)	Local station(s) (KMAA)	PM_{10}-conc. as proxy: 3.6e3 (broiler house) and 116.4 (road) [μg/m3] corrected for bird numbers and stable volumes	Yes	Yes	–	–	Threshold: 20 μg/m^3
Li et al. (2013)	# (mesophilic bacteria)	Simulation and measurements	China	2011–2012	Wastewater	Plume (Dowd et al., 2000)	In situ	3.2722e7 CFU/s	–	–	–	Rates: 0.0, 4.0e−3, 6.0e−3, 0.02, and 0.12 s^{-1}	Risk = dose /reference dose. Dose is based on breathing patterns. Reference dose: 1000 CFU/m^3
Lighthart and Frisch (1976)	–	Model analysis	–	–	–	Gaussian plume (equations)	–	–	–	–	–	Rates: 0, 0.1, 0.01, 0.001 s^{-1}	–

(*continued on next page*)

Table A.1 (*continued*)

Paper	Pathogen	Type	Country of investigation	Year	Source(s)	Model type (model name)	Meteorological data (institute)	Emission data	Dep.	PSD	Traj.	Inactivation	Dose–response model
Lighthart and Mohr (1987)	Reovirus, Venezuelan Equine Encephalomyelitus virus	Simulation	–	#	Field	Plume (Lighthart and Frisch, 1976)	Local station(s) (Oregon State University)	100 particles/m^2/s	Yes	–	–	Function of RH, *T*, radiation and time	–
Lin et al. (2014)	Fusarium	Simulation and measurements	USA	2009–2012	Field	Gaussian dispersion equation	#	#	–	–	–	–	–
Liu and You (2012)	Human influenza virus	Simulation	–	–	Urban	CFD (#)	#	2.1e5 particles/room	–	–	–	–	–
Low et al. (2007)	*Clostridium, Chloroflexi* sp., Euryarchaeota	Simulation and measurements	USA	#	Biosolids	Gaussian dispersion equation	In situ	Back-calculated from concentration measurements	Yes	–	–	–	–
Maragon et al. (1994)	FMDV	Outbreak, hypothetical outbreak	Italy	1993	Cattle, pigs	Puff (ICAIR 3 V)	Local station(s) (#)	7 $\log_{10}$ ID_{50}/day	–	–	–	–	–
Mayer et al. (2008)	FMDV	Hypothetical outbreak	Austria	2006	Cattle, pigs, sheep	Trajectories (equations)	Lokal-Modell-Kürzestfrist (DWD)	Yes: 5.06 (cattle), 7.16 (pigs) 4.94 (sheep) [$\log_{10}$ $TCID_{50}$/animal/day]	–	–	–	–	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Meselson et al. (1994)	*B. anthracis*	Outbreak	Russia (former Soviet-Union)	1979	Military	Plume (#)	Local station(s) (NCAR)	#	Yes	–	–	Rate: 0.001 min^{-1} (or 1.67e−5 s^{-1})	–
Mikkelsen et al. (2003)	FMDV	Outbreak	UK	2001	Cattle, pigs	Plume (Gloster et al., 1981), Puff (DERMA, NAME, RIMPUFF)	Local station(s) (Met Office), Unified Model, HiRLAM	Virus model (Sørenson et al., 2000)	Yes	Yes	F	–	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Moutou and Durand (1994)	FMDV	Outbreak	France, Italy, UK	1981–1982, 1993	Cattle, pigs, sheep	Puff (ICAIR 3 V)	Local station(s) (#)	#	–	–	–	Inactivation for RH < 60%	–
Nguyen et al. (2006)	*L. pneumophila*	Outbreak	France	2003–2004	Cooling tower	Plume (ADMS)	Yes (#)	#	Yes	Yes	–	–	–
Nicogossian et al. (2011)	*B. anthracis*	Hypothetical outbreak	USA	#	Urban	Particle mode (OMEGA)	Yes (#)	10 kg with 106 spores/mg	Yes	Yes	–	–	#
Nygård et al. (2008)	*L. pneumophila*	Outbreak	Norway	2005	Wastewater	Puff (INPUFF)	Local station(s) (NMI)	100 g/s	Yes	–	–	–	Attack rate analysis
Peterson and Lighthart (1977)	–	Model analysis	–	–	Cooling towers	Gaussian dispersion equation	–	0.062–0.18 m^3/s	Yes	Yes	–	Rates: 0.0001, 0.001, or 0.01 s^{-1}	–
Rubel and Fuchs (2005)	FMDV	Hypothetical outbreak	Austria	2003	Pigs	Gaussian dispersion equation	Unspecified model (DWD)	8.6 $\log_{10}$ $TCID_{50}$/animal/day	–	–	–	Inactivation for RH < 55% or > 27°C.	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Sanson et al. (2011)	FMDV	Outbreak	UK	1967–1968	Pigs	Puff (NAME)	Local station(s) (Met Office)	0–6 $\log_{10}$ $TCID_{50}$/animal/day	Yes	Yes	–	–	Yes (#)
Sauter et al. (2011)	*C. burnetii*	Outbreak	The Netherlands	2008–2009	Goats	Plume (OPS-ST)	Local station(s) (KNMI)	Three time-dependent emission profiles	Yes	Yes	–	–	–
Schley et al. (2009)	FMDV	Outbreak	UK	1968, 2007	Cattle, pigs, sheep	Puff (NAME)	Local station(s) Unified Model (both Met Office)	Yes (#)	Yes	Yes	–	–	Exponential model
Seo and Lee (2013)	AIV	Outbreak	South Korea	2008	Poultry	CFD (ANSYS Fluent)	Yes (#)	#	Yes	Yes	–	–	–
Seo et al. (2014)	AIV	Outbreak	South Korea	2008	Poultry	CFD (ANSYS Fluent)	# (KMAA)	PM_{10}-conc as proxy: 3.6e3 (broiler house) and 116.4 (road) [μg/m3], corrected for bird numbers and stable volume	Yes	Yes	–	–	Threshold: 20 $\mu g/m^3$
Smith et al. (2012)	Various pathogens	Simulation and measurements	USA	2011	#	Trajectories (HYSPLIT)	GDAS	–	–	–	B	–	–
Smith et al. (2013)	Various pathogens	Simulation and measurements	USA	2011	#	Trajectories (HYSPLIT)	GDAS	–	–	–	B	–	–
Sorber et al. (1976)	*Escherichia, Klebsiella, Enterobacter, Streptococcus*	Simulation and measurements	USA	1974	Wastewater	Gaussian dispersion equation	In situ, local station(s)	–	–	Yes	–	–	–
Sørenson et al. (2000)	FMDV	Outbreak	Denmark, France, Germany, UK	1981, 1982	Cattle, pigs, sheep	Puff (RIMPUFF)	Local station(s) (#), HiRLAM	3.5–4.7 (cattle), 4.3–8.6 (pigs), 2.4–5.1 (sheep) [$\log_{10}$ $TCID_{50}$/animal/day]	Yes	Yes	–	Inactivation for RH < 55%. Rate for RH > 55%: 3.2e−4 h^{-1} ≈ 8.9e−8 s^{-1}	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Sørenson et al. (2001)	FMDV	Outbreak	Denmark, France, Germany, UK	1981, 1982	Cattle, pigs, sheep	Puff (RIMPUFF)	HiRLAM	Virus model (Sørenson et al., 2000)	Yes	Yes	–	Inactivation for RH < 55%	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Sprigg et al. (2014)	*C. immites, C. posadasii*	Outbreak	USA	2011	Env.	Eulerian (DREAM)	NCEP/NCAR, ECMWF	Yes (#)	Yes	Yes	–	–	–

(*continued on next page*)

Table A.1 (*continued*)

Paper	Pathogen	Type	Country of investigation	Year	Source(s)	Model type (model name)	Meteorological data (institute)	Emission data	Dep.	PSD	Traj.	Inactivation	Dose–response model
Ssematimba et al. (2012)	AIV (H7N7)	Outbreak	The Netherlands	2003	Poultry	Gaussian dispersion equation	Local station(s) (KNMI)	0.0122 g dust/animal/h	Yes	–	–	Rates: 4.0e−7–2.0e−6 s^{-1}	Exponential model (variation): $P_{inf}(D) = [1 + \exp(\alpha + \gamma \cdot D]^{-1}$ [$\alpha = 4.67$, $\gamma = -1.87$]
Stellacci et al. (2010)	*Cryptosporidium*, *Campylobacter*, rotavirus	Simulation	Italy	#	Wastewater	Plume (GIADA)	Yes (#)	57.87 oocysts/s (*Cryptosporidium*), 578.7 CFU/s (*Campylobacter*), 587.7 MPN/s (rotavirus)	#	#	–	Rate: 0.1 s^{-1}	Beta-Poisson model: *Cryptosporidium* [$\alpha = 0.115$, β=0.176, D_{50}=73], *Campylobacter* [$\alpha = 0.024$; $\beta = 0.011$; $D_{50} = 3.84e10$], rotavirus [$\alpha = 0.2531$; $\beta = 0.4265$; $D_{50} = 6.17$]
Stuart and Wilkening (2005)	*B. anthracis*	Hypothetical outbreak	–	–	Urban	Gaussian dispersion equation	@	10^{15} spores (~1 kg)	–	–	–	Four models (sensitivity analysis)	Probit model: $p_{\text{death}}(x, y, t) = \int_{-\infty}^{\zeta} \frac{1}{\sqrt{2\pi}} \exp[-\frac{\chi^2}{2}]d\chi$
Taha et al. (2005)	*A. fumigatus*, mesophylic actinomycetes	Simulation and measurements	UK	#	Biosolids	Plume (SCREEN3)	In situ	3.6e3–2.17e4 CFU/m^2/s	–	–	–	–	–
Taha et al. (2006)	*A. fumigatus*	Simulation and measurements	UK	2004	Biosolids	Plume (SCREEN3)	In situ	5e5–8.6e8 CFU/s	–	–	–	–	–
Taha et al. (2007)	*A. fumigatus*, mesophylic actinomycetes	Simulation and measurements	UK	2005	Biosolids	Plume (ADMS, SCREEN3)	In situ	4.8e4–1.6e7 CFU/s	–	–	–	–	–
Tang et al. (2009)	*B. anthracis*	Simulation	–	–	Urban	CFD (equations)	@	–	–	–	B	–	–
Teltsch et al. (1980)	*E. coli*	Simulation and measurements	Israel	1978	Wastewater	Gaussian dispersion equation	In situ, local station(s) (Israelian Meteorological Service)	Yes (#)	–	–	–	8.8e−3 s^{-1} (early morning), 6.6e−2 s^{-1} (afternoon)	–
Traulsen et al. (2010)	FMDV	Outbreak	Germany	2003	Cattle, pigs, sheep	Plume (RIMPUFF)	#	Virus model (Sørenson et al., 2000)	Yes	Yes	–	Rate: 6.4e−4 × 0.5 h^{-1}	Threshold: 0.045 $TCID_{50}$
Traulsen and Krieter (2012)	FMDV	Outbreak	#	#	Pigs	Gaussian dispersion equation	Local station(s) (#)	6.7 $\log_{10}$ $TCID_{50}$/s (pigs)	–	–	–	–	Threshold: 0.06 (cattle), 1.11 (sheep), 7.70 (pigs) $TCID_{50}/m^3$
Van Leuken et al. (2015)	*C. burnetii*	Outbreak	The Netherlands	2009	Goats	Plume (OPS-ST)	Local station(s) (KNMI)	Three time-dependent emission profiles	Yes	Yes	–	–	–
Wallensten et al. (2010)	*C. burnetii*	Outbreak	UK	2007	Sheep	Puff (NAME)	Local station(s), Unified Model (Met Office)	–	Yes	Yes	–	–	–
Wein et al. (2003)	*B. anthracis*	Model analysis	USA	–	Urban	Gaussian dispersion equation	@	10^{15} spores (~1 kg)	–	–	–	–	Age-dependent probit model: $P(D, a) = \Phi(\alpha + \beta \log(D) + \gamma a + \delta a^2)$ with a = age [years] [$\alpha = -9.733$, $\beta = 1.025$, $\gamma = -0.016$/year, and $\delta = 6e{-}4/year^2$]
Yu et al. (2004)	SARS virus	Outbreak	China (Hong Kong)	2003	Urban	CFD (ANSYS-Fluent)	Local station(s) (Hong Kong Observatory)	–	–	–	–	–	–

OR corynosom* OR cowpox OR coxiella* OR creutzfeldt-jakob OR cryptococc* OR cryptocotyl* OR cryptosporid* OR culicid* OR culicin* OR cunninghamell* OR curvular* OR cyclophyllid* OR cyclospor* OR cylindrocarpon* OR davaineid* OR davidiell* OR deinococc* OR delfti* OR dendrocygnin* OR dendryphi* OR dengue OR dermatophil* OR dermocystid* OR desulfovibrion* OR diarrhoea OR dichotomophthor* OR dicrocoel* OR dientamoeb* OR dietzi* OR dilepidid* OR dinemaspor* OR dioctophym* OR dipetalonem* OR diphtheria OR diphyllobothr* OR diplococc* OR diplogonopor* OR diplostom* OR dipodasc* OR diptera* OR dipylid* OR dirofilar* OR dissitimur* OR doratomyc* OR dothid* OR dothior* OR dracuncul* OR drechsler* OR drepanidotaen* OR duganell* OR dysentery OR ebola OR echinochasm* OR echinococc* OR echinoparyph* OR echinostom* OR edwardsiell* OR eggerthell* OR ehrlichi* OR eikenell* OR eimeriid* OR emericell* OR emmonsi* OR encephalitozoon* OR engyodonti* OR enoplid* OR entamoeb* OR enterob* OR enterococc* OR enterocytozoon* OR entomophthor* OR epicocc* OR epidermophyt* OR episthmi* OR erwini* OR erysipelothr* OR escherich* OR eucoccidiorid* OR eucole* OR euglenoz* OR eupenicilli* OR eurotial* OR eurotiomycet* OR eurytrem* OR eustrongylid* OR ewingell* OR exobasidiomyc* OR exophial* OR exserohil* OR fasciol* OR filifactor* OR filobasidi* OR finegoldi* OR firmicut* OR flavimon* OR fmd OR fonseca* OR foot-and-mouth OR francisell* OR franki* OR fulvimari* OR fusari* OR ganoderm* OR gardnerell* OR gastrodisc* OR gastroenteritis OR gemell* OR geotrich* OR giardi* OR gibberell* OR gigantobilharz* OR glomerell* OR gnathostom* OR gonococcal OR gonorrhoea OR gordoni* OR granulicatell* OR grimont* OR guillain-barre OR guillain–barré OR gymnoasc* OR gymnophall* OR h10n7 OR h1n2 OR h2n2 OR h3n1 OR h3n2 OR h3n8 OR h4n6 OR h5n1 OR h5n2 OR h5n7 OR h7n1 OR h7n2 OR h7n3 OR h7n7 OR h9n2 OR haematonectr* OR haemolytic-uremic OR haemonch* OR haemophil* OR haemorrhagic OR haemosporid* OR hafni* OR halosphaer* OR hansenul* OR hanta OR haplorch* OR helcococc* OR hepatitis OR herpotrichiell* OR heterobilharz* OR heterolobos* OR heteroph* OR hexamitid* OR hib* OR himasthl* OR histoplasm* OR hiv* OR hortae* OR hu39694 OR hymenolep* OR hypocrea* OR hypoder* OR hyponectr* OR hysteri* OR inermicapsifer* OR isaria OR isoparorch* OR isospor* OR issatchenk* OR jiangell* OR kineococc* OR kinetoplast* OR kingell* OR klebsiell* OR kluyver* OR kocuri* OR kurthi* OR lachnospir* OR lagochilascar* OR lasiodiplodi* OR lasiosphaer* OR lechevalieri* OR lecithodendriid* OR lecythophor* OR legionell* OR legionnaires* OR leifson* OR leishman* OR lentz* OR leptosphaer* OR leptospir* OR leptotrich* OR lewia* OR libertell* OR ligula* OR listeri* OR lithothel* OR litostomat* OR lojkan* OR lophiostom* OR lyngby* OR macracanthorhynch* OR madurell* OR magnaporth* OR malaria OR malassez* OR mammomonogam* OR mannheimi* OR mansonell* OR marshallag* OR massar* OR massili* OR mathevotaen* OR measles OR mecistocirr* OR megamon* OR megasphaer* OR melanommat* OR memnoniell* OR meningococc* OR meningonem* OR mergina* OR mesocestoid* OR mesomycetoz* OR mesorhizobi* OR metagonim* OR metamonad* OR metastrongyl* OR methanosarcinal* OR methanosphaer* OR metorch* OR metschnikow* OR microasc* OR microbotryomyc* OR micrococc* OR microdochi* OR microfilar* OR micromon* OR micronem* OR microspor* OR microstromatal* OR molineid* OR mollicut* OR moniez* OR monili* OR moniliell* OR moniliform* OR monocercomonadid* OR monographell* OR mononegaviral* OR moraxell* OR morganell* OR mortierrell* OR mrsa OR mucor* OR multiceps* OR mumps OR mycel* OR myceliophthor* OR mycocentrospor* OR mycoleptodisc* OR mycoplasm* OR mycosphaerell* OR myositis OR myriang* OR myriodont* OR myroid* OR myxotrich* OR myzoz* OR naegler* OR nannizz* OR nanophyet* OR nattrass* OR necator OR nectria* OR neisser* OR neocosmospor* OR neodiplostom* OR neoricketts* OR neosartory* OR neotestudin* OR neurospor* OR newcastle* OR nidovir* OR nigrospor* OR nitrosomonadal* OR nocard* OR nosem* OR nostoc OR novosphingobi* OR ochrobactr* OR ochrocon* OR oerskov* OR oesophagostom* OR oidiodendr* OR oidium OR oligacanthorhynchid* OR oligell* OR onchocerc* OR onychocol* OR onygen* OR oomyc* OR ophiostomat* OR opisthorch* OR orientia OR ornithobilharz* OR ostertag* OR ovadendr* OR oxyurid* OR oxyurin* OR paecilomyc* OR palaeacanthocephal* OR panagrolaimid* OR pantoea OR papular* OR paracocc* OR paragonim* OR paramphistomid* OR parascar* OR parastrongyl* OR paratyphoid OR pasteurell* OR pasturell* OR pearsonem* OR pedicul* OR pelliodit* OR pelobiontid* OR peloder* OR penicill* OR pentatrichomon* OR peptococc* OR peptoniphil* OR peptostreptococc* OR percoloz* OR pericon* OR pericon* OR pertussis OR phaeoannellomyc* OR phaeoscler* OR phaeosphaer* OR phaeotrichocon* OR phaneropsol* OR phialemon* OR phialophor* OR philophthalm* OR phocanem* OR phoma OR phoma* OR phormidi* OR phthirus OR phyllostict* OR physalopter* OR pichia* OR piedrai* OR piroplasmid* OR plagiorch* OR plague OR planctomyc* OR planococc* OR plasmod* OR platyhelminth* OR plectropterin* OR pleospor* OR plesiomon* OR pleurophom* OR pneumococc* OR pneumocyst* OR pneumoni* OR poikilorch* OR poliomyelitis OR porphyromon* OR prevotell* OR procerov* OR prohemistom* OR promicromonospor* OR prosthodendr* OR proteus OR protothec* OR protozoa OR providenc* OR pseudallescher* OR pseudamphistom* OR pseudocochliobol* OR pseudomicrodoch* OR pseudomon* OR pseudonocard* OR pseudophyllid* OR pseudoterranov* OR psilorch* OR psilostomatid* OR psittacosis OR pygidiops* OR pyramicocephal* OR pyrenochaet* OR pythia* OR pythium OR pythomyc* OR quambalar* OR rabies OR rahnell* OR raillietin* OR ralstoni* OR raoulterr* OR retortamon* OR rhabdit* OR rhinocladiell* OR rhinosporid* OR rhizobial* OR rhizomucor* OR rhizop* OR rhodococc* OR rhodotorul* OR rickettsi* OR rictular* OR rochalim* OR rothia OR rubella OR ruminococc* OR saccharomonospor* OR saccharomyc* OR saccharopolyspor* OR 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thermoactinomyc* OR thermomonospor* OR thermomyc* OR torul* OR toxocar* OR toxoplasm* OR trachipleistophor* OR trebouxiophy* OR trematod* OR tremell* OR trepomonad* OR treponem* OR trichinell* OR trichobilharz* OR trichocephalid* OR trichocom* OR trichoderm* OR trichomar* OR trichomon* OR trichophyt* OR trichosphaerial* OR trichospor* OR trichostrongyl* OR trichuri* OR triotrichal* OR tritirach* OR troglotrematid* OR tropherym* OR trypanosom* OR tsukamurell* OR tubulin* OR tularaemia OR typhus OR uloclad* OR ureaplasm* OR ustilaginomycotin* OR vahlkampfiid* OR varicella OR veillonell* OR verona* OR verruco* OR verticill* OR vestibuliferid* OR vibrio* OR vittaform* OR volutell* OR wallemi* OR watsoni* OR west-nile OR wolinell* OR wucherer* OR xanthamon* OR xanthomon* OR xylarial* OR yarrowia OR yersini* OR zoogl* OR zygomyc* OR zygospor*) **AND NOT** (indoor OR hospital* OR phytoplankt* OR biomass OR membrane* OR genes OR cardia* OR "operating room*" OR biofilm* OR cough* OR fiber* OR hypertension OR hypotension OR chamber* OR crop* OR chlorophyll* OR

candidate* OR window* OR odour OR electric* OR serration* OR "water quality"))

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