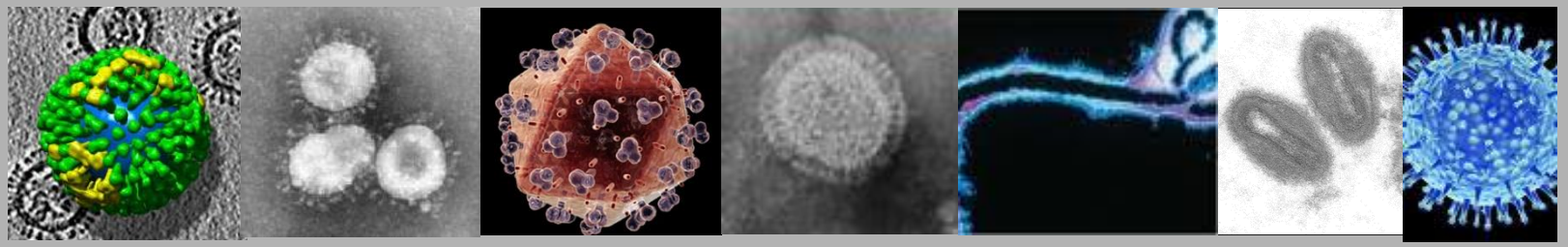


WHAT WILL CAUSE THE NEXT PANDEMIC?

Mark Woolhouse

Epidemiology Research Group

Centre for Immunity, Infection & Evolution

University of Edinburgh, UK

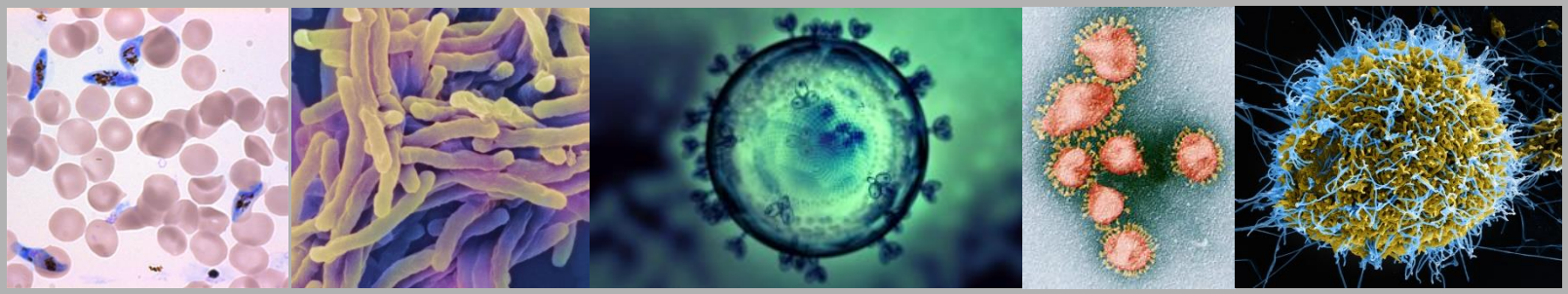
THE INFECTIOUS DISEASE PROBLEM

- A quarter of all deaths; a quarter of all illness
  - "Big Three" = malaria, TB, AIDS
  - HIV/AIDS (since 1980s): 35M deaths, 37M currently infected

- ~1500 different kinds of pathogen

+ Several new ones discovered per year

- Epidemic shocks (mortality, cost)
  - SARS (2003): >900 deaths, more than US$50 billion
  - Ebola (2014-15): >11,000 deaths, several billion US$



AFTER EBOLA, WHAT NEXT?

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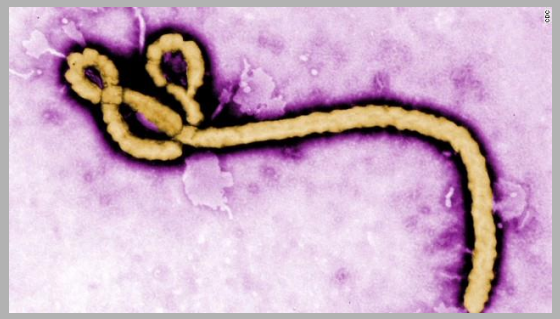
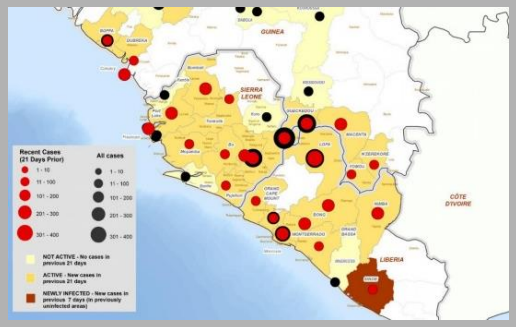




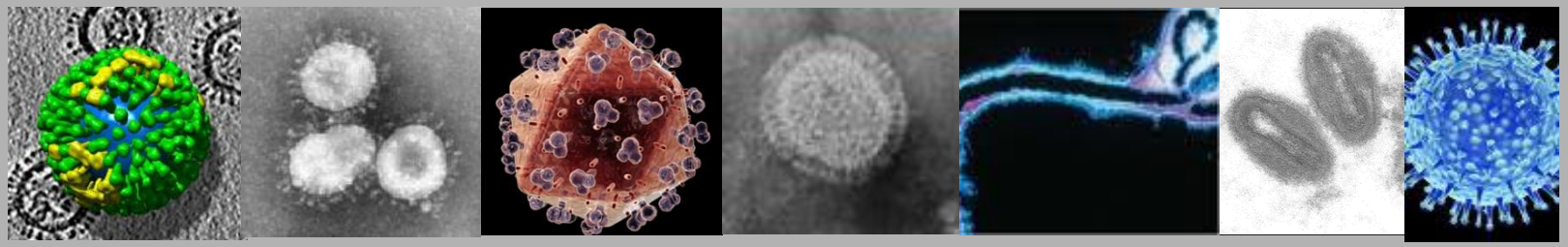
AFTER ZIKA, WHAT NEXT?

Mark Woolhouse

Epidemiology Research Group

Centre for Immunity, Infection & Evolution

University of Edinburgh, UK

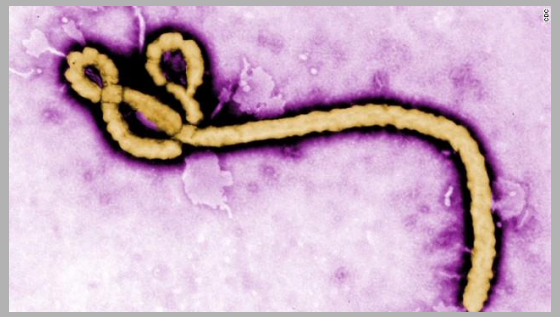


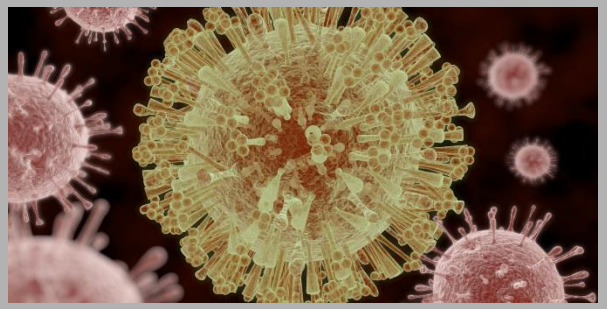
WHAT WILL CAUSE THE NEXT PANDEMIC?

- Discovery
- Human virus – Mammal virus
- Prioritisation
- Response

EMERGING INFECTIOUS DISEASES

- **Mostly viruses**
  - **more than 70% of recently discovered pathogens**



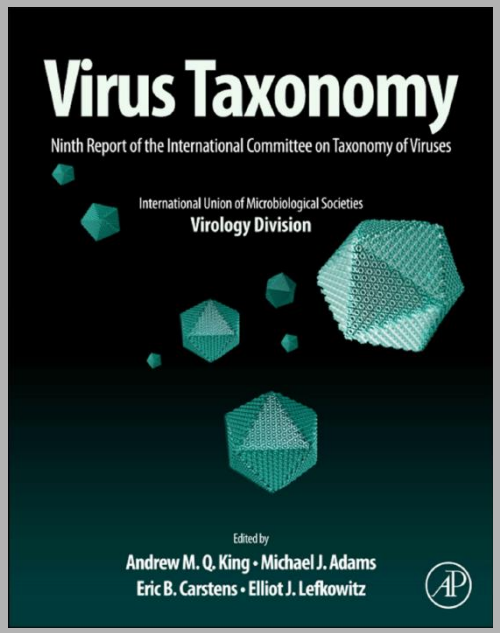


- **Mostly zoonotic, i.e. shared with other animals**
  - **more than 70% of recently discovered pathogens**

HUMAN RNA VIRUS SURVEY

- Systematic review of primary literature
- Formal methodology

→ Taylor *et al.* (2001) *Phil. Trans. B*

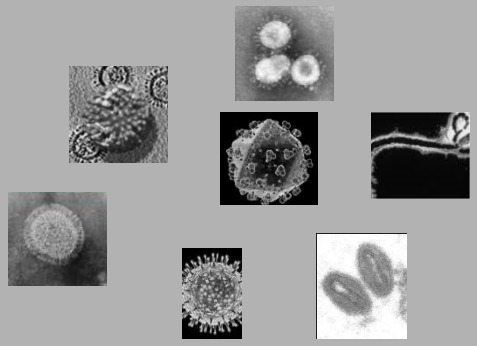


- ICTV (2016) classification of "species"
- Catalogue of RNA virus species reported to infect humans

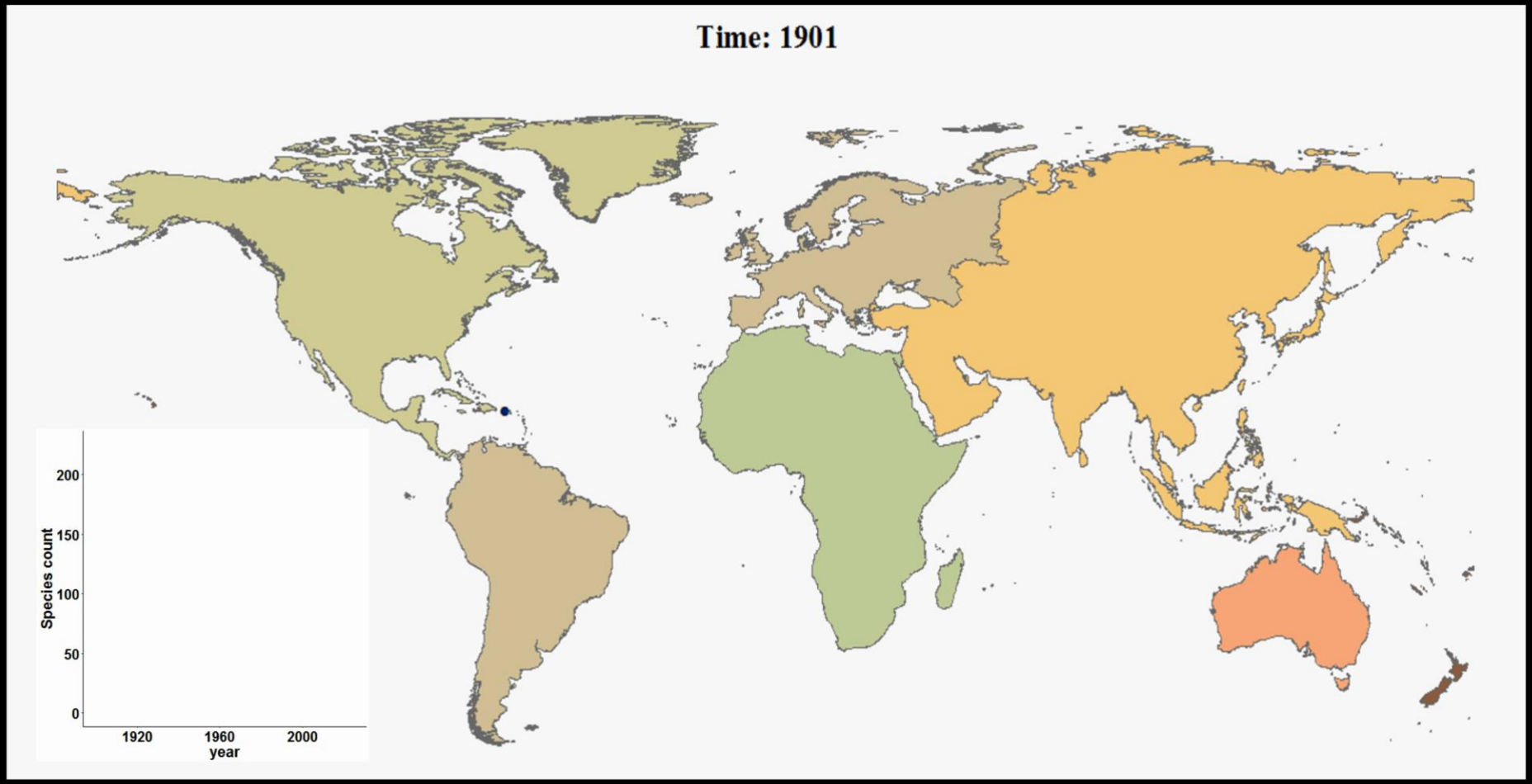
➢ **214** recognised species (to 2015)

➢ **55** genera

➢ **21** families (+1 unassigned genus)



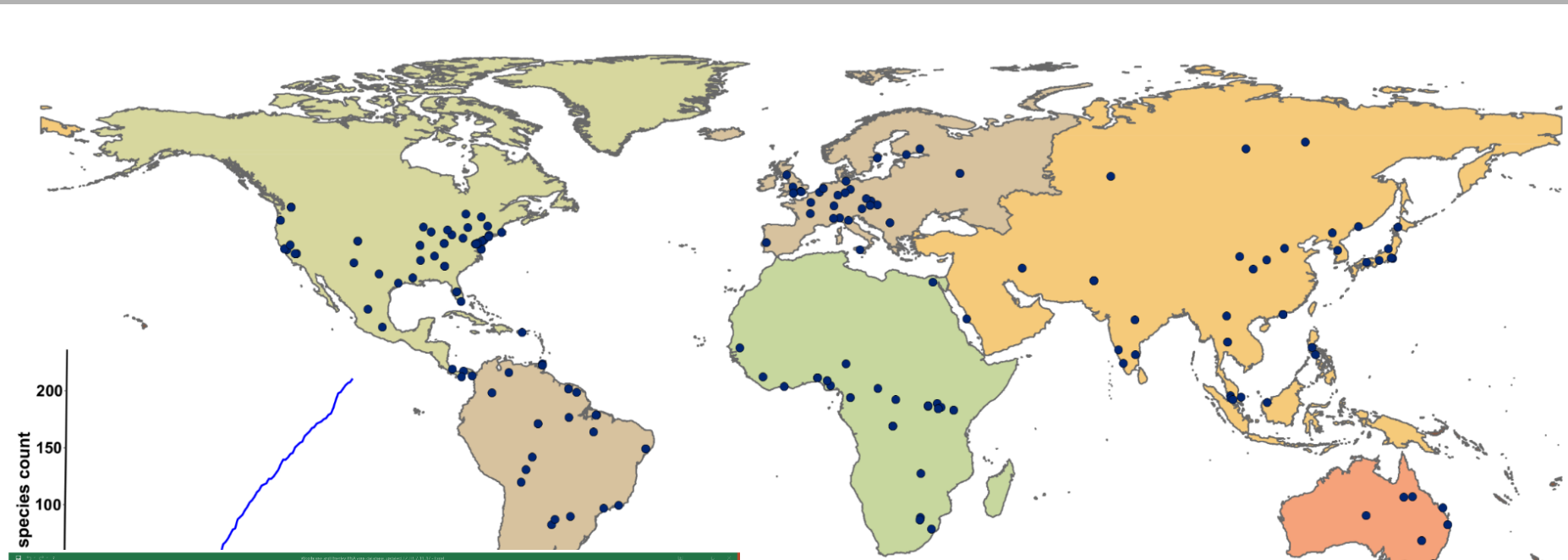
RNA VIRUS DISCOVERY

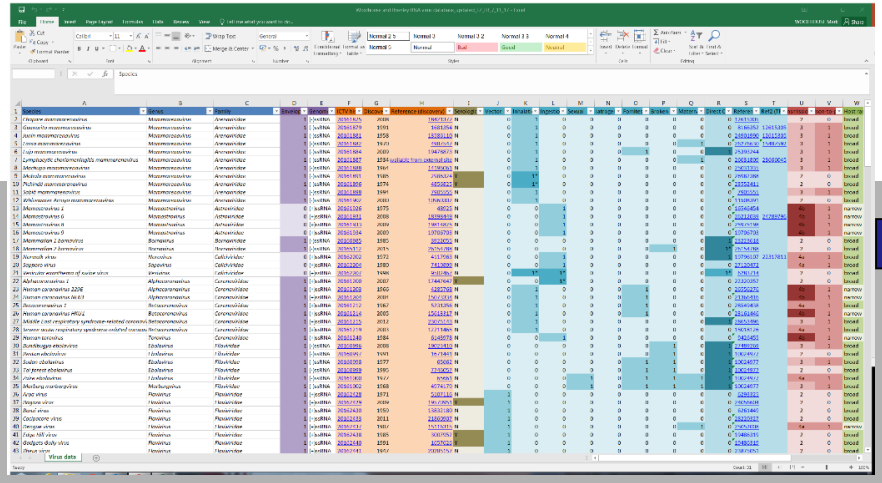


Mark Woolhouse, University of Edinburgh, January 2018

Thanks to Liam Brierley, Feifei Zhang

RNA VIRUS DISCOVERY





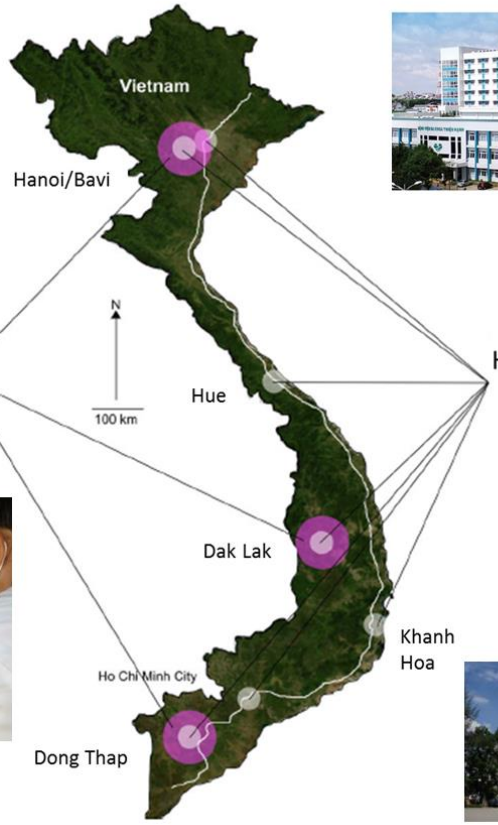
SCIENTIFIC DATA

Mark Woolhouse, University of Edinburgh, January 2018

Thanks to Liam Brierley, Feifei Zhang

WT-VIZIONS

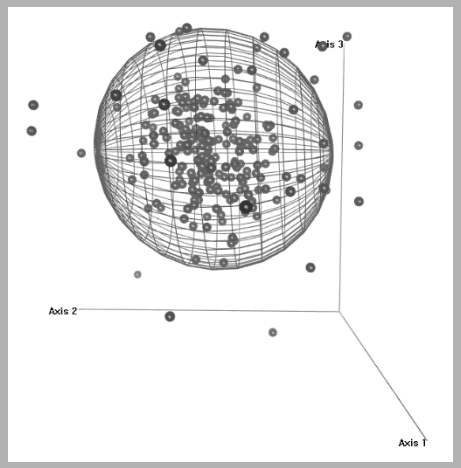
Wellcome Trust-Viet Nam Initiative on Zoonotic Infections

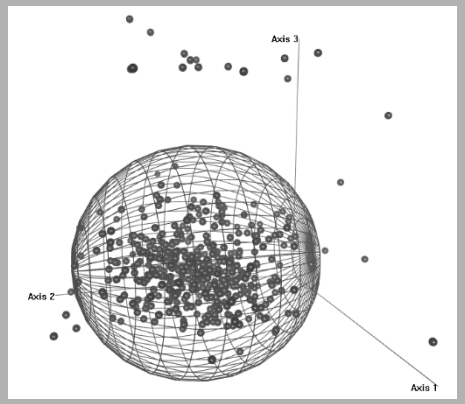


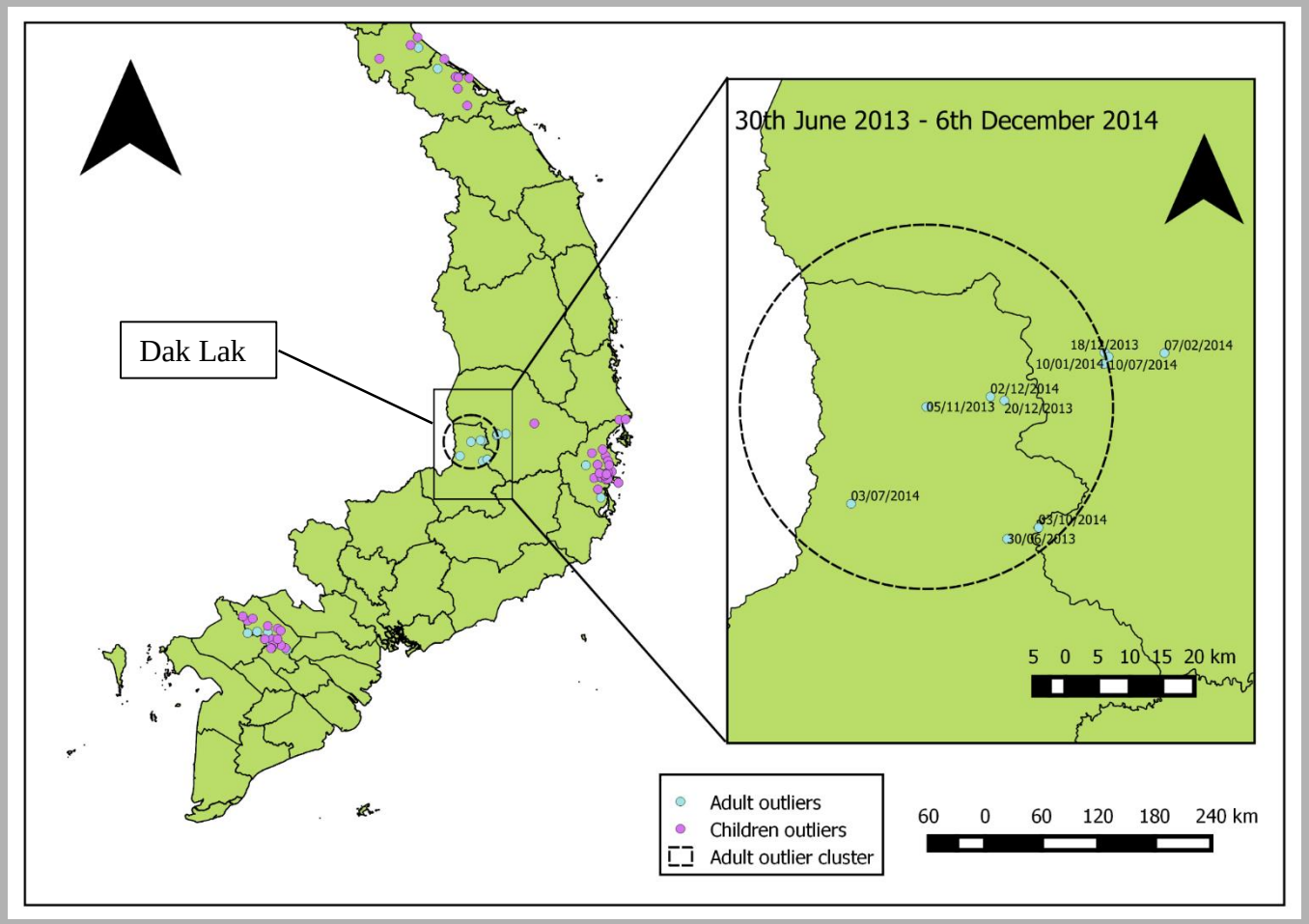
Several thousand enteric, respiratory and CNS samples from hospitalised patients, high risk cohorts and animals

Mark Woolhouse, University of Edinburgh, January 2018

DISEASES OF UNKNOWN ORIGIN



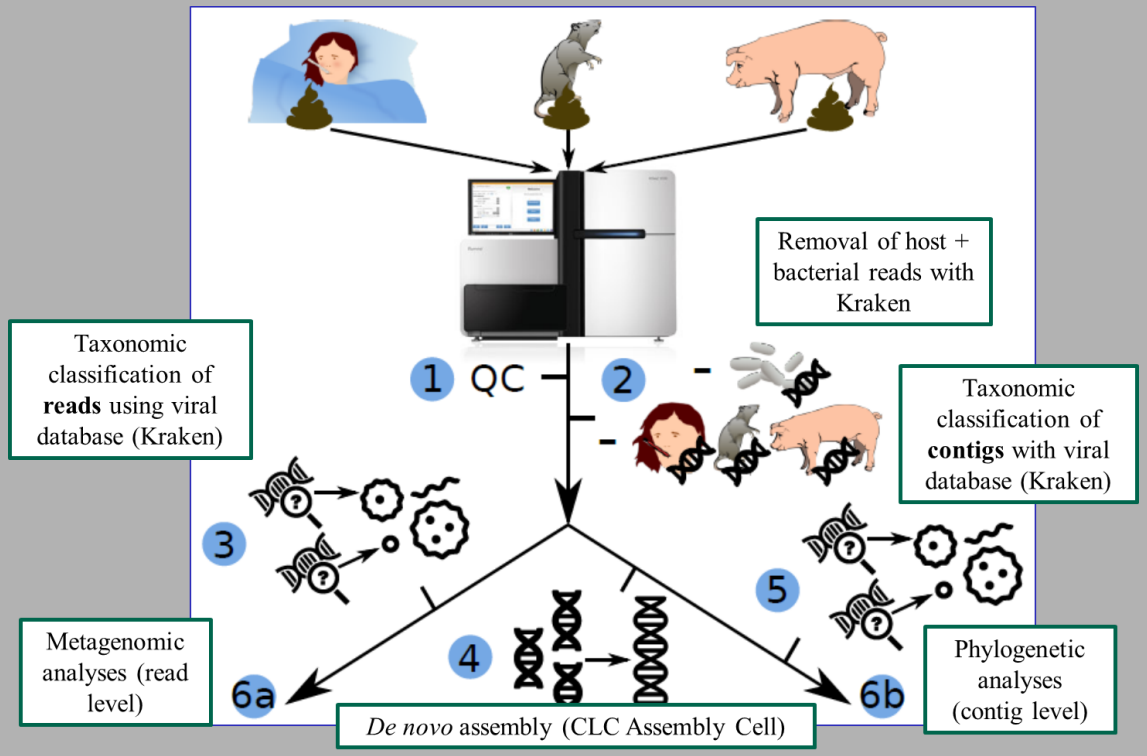


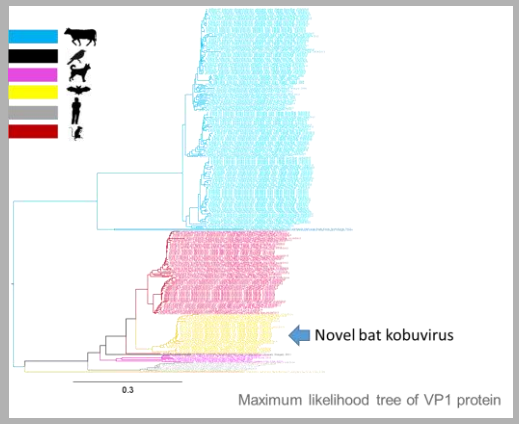


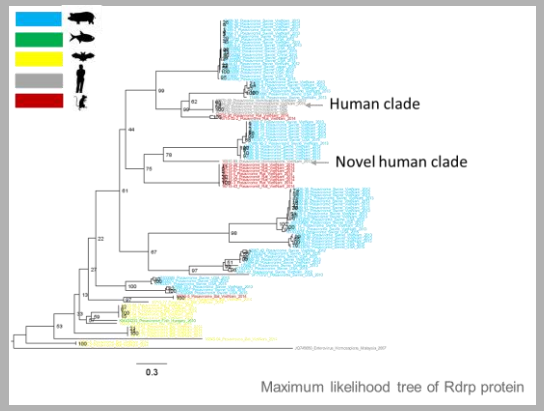
Mark Woolhouse, University of Edinburgh, January 2018

Thanks to Gail Robertson, Margo Chase-Topping

METAGENOMICS + PHYLOGENETICS







Metagenomics detection

- 2102 faecal samples
  - 1260 human, 842 animal
- >7 billion short reads in total
- Signal detection threshold criteria
- Viruses from 61 genera across 22 families

SCIENTIFIC DATA

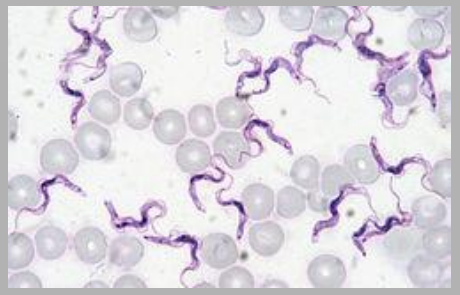
Mark Woolhouse, University of Edinburgh, January 2018

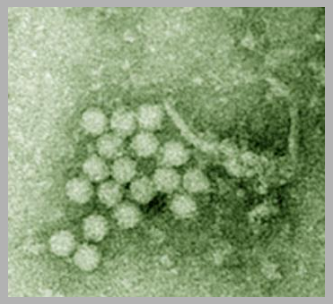
Thanks to Carlijn Bogaardt, Al Ivens, Jordan Ashworth, Lu Lu

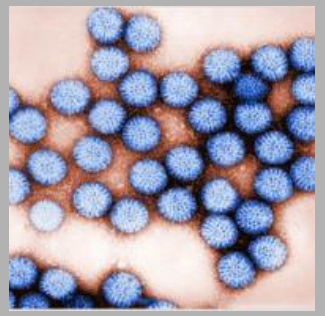
UNUSUAL PATHOGENS

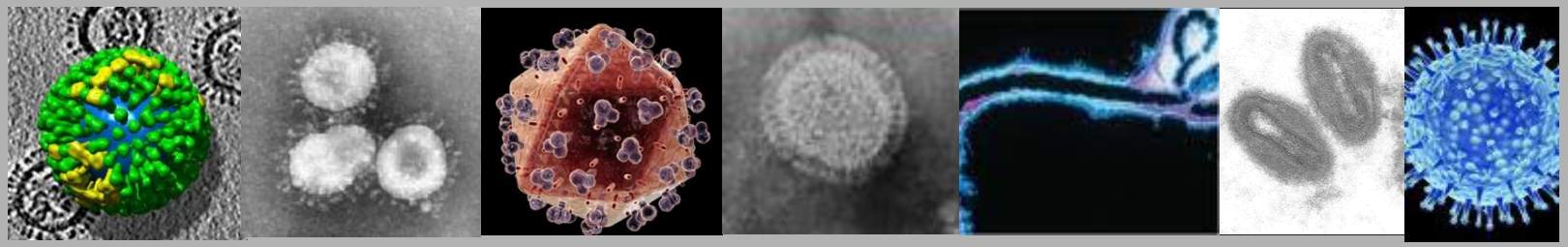
- Novel **cyclovirus** (CyCV-VN) in CSF from hospital patients
- 2 novel enteric **CRESS-DNA viruses** in hospital patients
- Novel porcine-like **rotavirus** (G26P[19]) in paediatric diarrhoea cases
- First human **husavirus** infections outside Europe
- First human cases of ***Trypanosoma evansi*** infection in SE Asia

- Novel **kobuviruses** in bats
- Novel **hunniviruses** in rodents
- Novel ***Bartonella*** spp in bats
- Novel **bunyavirus** in bats
- Novel **cardiovirus** in rats
- Novel bat and rat **stool-associated viruses**
- Proposed new **hepacivirus** in bamboo rats









WHAT WILL CAUSE THE NEXT PANDEMIC?

- Discovery
- **Human virus – Mammal virus**
- Prioritisation
- Response

HUMAN VIRUS, MAMMAL VIRUS

- 188/214 (88%) known human RNA viruses naturally infect other mammals
- Most of the 26 human-specific RNA viruses have close relatives that infect other mammals [except: rubella, hepatitis delta]
- Only 38/214 (18%) infect non-mammals [= birds (37) ± reptiles (7) ± fish (1?)]

- 55/74 (74%) mammal RNA virus genera include human viruses
- 21/23 mammal RNA virus families include human viruses [except: Arteriviridae, Nodaviridae]

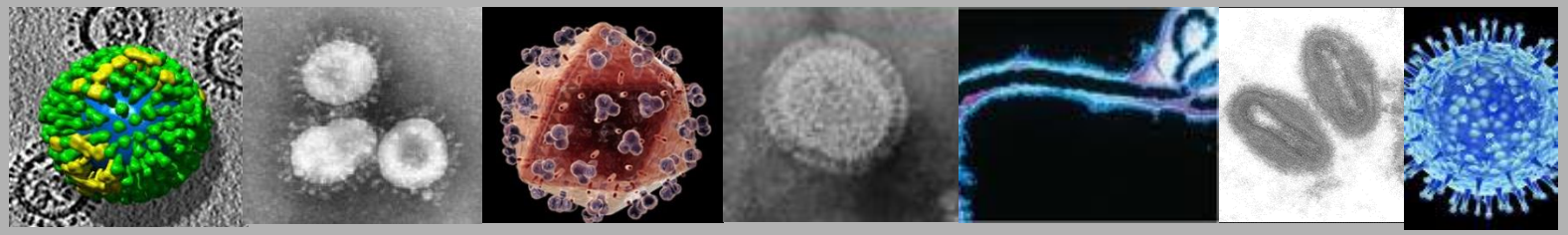
- "Majority of human viruses… are the product of host jumping" Kitchen *et al*. (2011) *PNAS*
- Easier to switch host species than alter tissue tropism or transmission route

➢ Human infectivity evolves very easily within the mammal RNA viruses,
less easily from birds and never(?) from anything else

DYNAMICS OF DIVERSITY

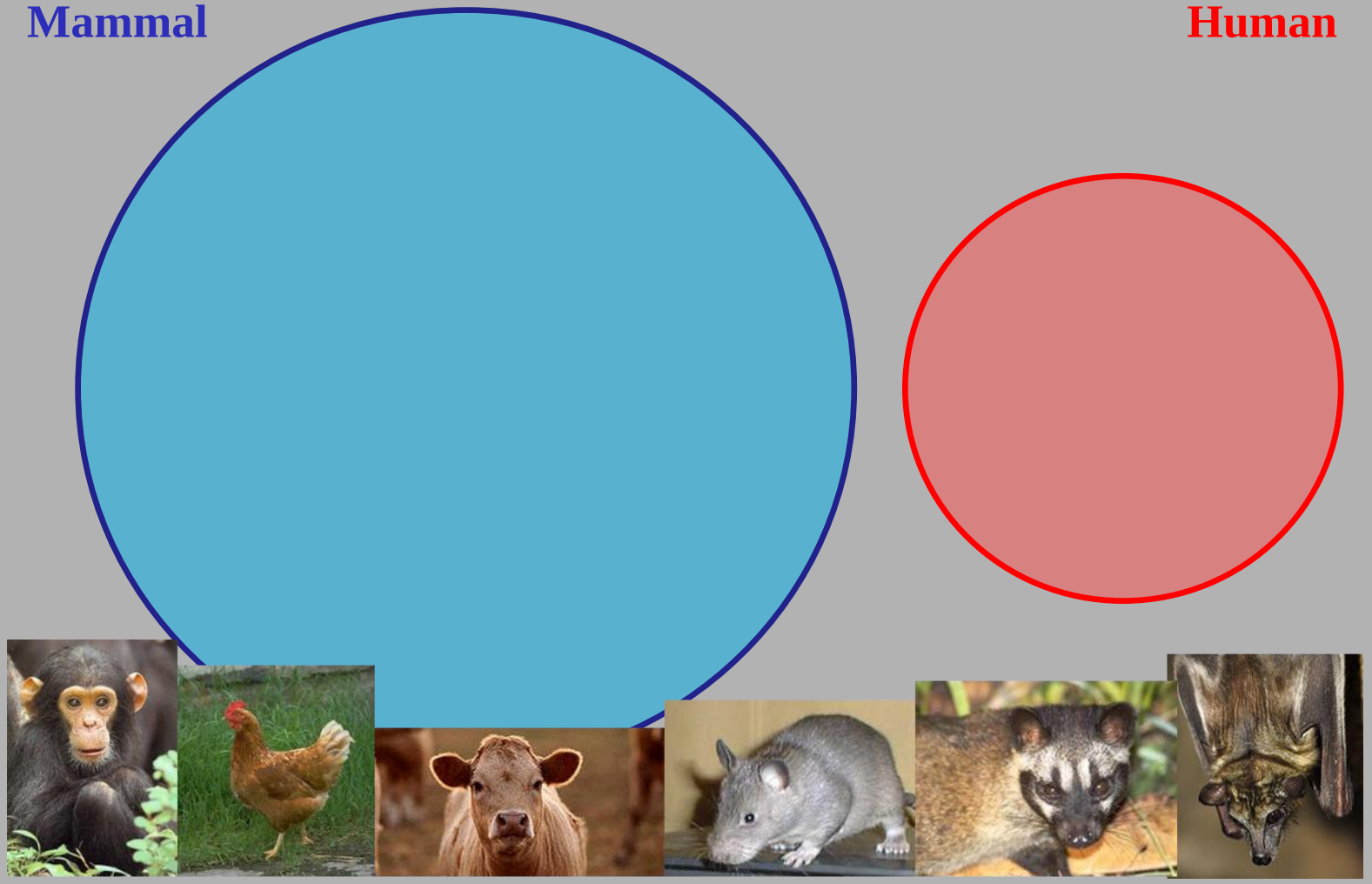
- Apparent loss of human RNA virus diversity:
- 73 species (>⅓ total) not seen (in humans) since 2005

Madrid orthobunyavirus	1961	Orungo virus	1982	Bagaza virus	1996
Ndumu virus	1961	Tacaiuma orthobunyavirus	1983	Equine rhinitis A virus	1996
Rio Bravo virus	1962	New Jersey vesiculovirus	1983	Erbovirus A	1996
Whataroa virus	1964	Maraba vesiculovirus	1984	Alphacoronavirus 1	1997
Wyeomyia orthobunyavirus	1965	Mobala mammarenavirus	1985	Dugbe nairovirus	1998
Banzi virus	1965	Louping ill virus	1985	Vesicular exanthema of swine virus	1998
Patois orthobunyavirus	1967	Changuinola virus	1985	Aroa virus	1998
Guama orthobunyavirus	1967	European bat 1 lyssavirus	1986	Kokobera virus	1998
Foot-and mouth-disease virus	1967	Dhori virus	1987	Omsk hemorrhagic fever virus	1998
Cocal vesiculovirus	1967	Alagoas vesiculovirus	1987	Uganda S virus	1999
Thogoto virus	1969	Semliki Forest virus	1987	Tonate virus	1999
Getah virus	1969	Indiana vesiculovirus	1988	Whitewater Arroyo mammarenavirus	2000
Mokola lyssavirus	1972	Shuni orthobunyavirus	1990	Thiafora nairovirus	2000
Pichindé mammarenavirus	1973	Edge Hill virus	1990	Highlands J virus	2000
Nyando orthobunyavirus	1973	Simian virus 41	1990	Langat virus	2001
Catu orthobunyavirus	1974	Corriparta virus	1990	Una virus	2001
Punta Toro phlebovirus	1974	Gadgets Gully virus	1991	Rotavirus H	2002
Lebombo virus	1975	Mucambo virus	1991	Parainfluenza virus 5	2003
Oriboca orthobunyavirus	1976	Piry vesiculovirus	1993	Chapare mammarenavirus	2004
Uukuniemi phlebovirus	1977	Sabiá mammarenavirus	1994	Candiru phlebovirus	2004
Ntaya virus	1977	Bwamba orthobunyavirus	1994	SARS coronavirus	2004
Everglades virus	1977	Black creek canal hantavirus	1995	Thottapalayam hantavirus	2005
Great Island virus	1978	New York hantavirus	1995	Ilheus virus	2005
Isfahan vesiculovirus	1978	Tai forest ebolavirus	1995	Mammalian orthoreovirus	2005
Eyach virus	1980				



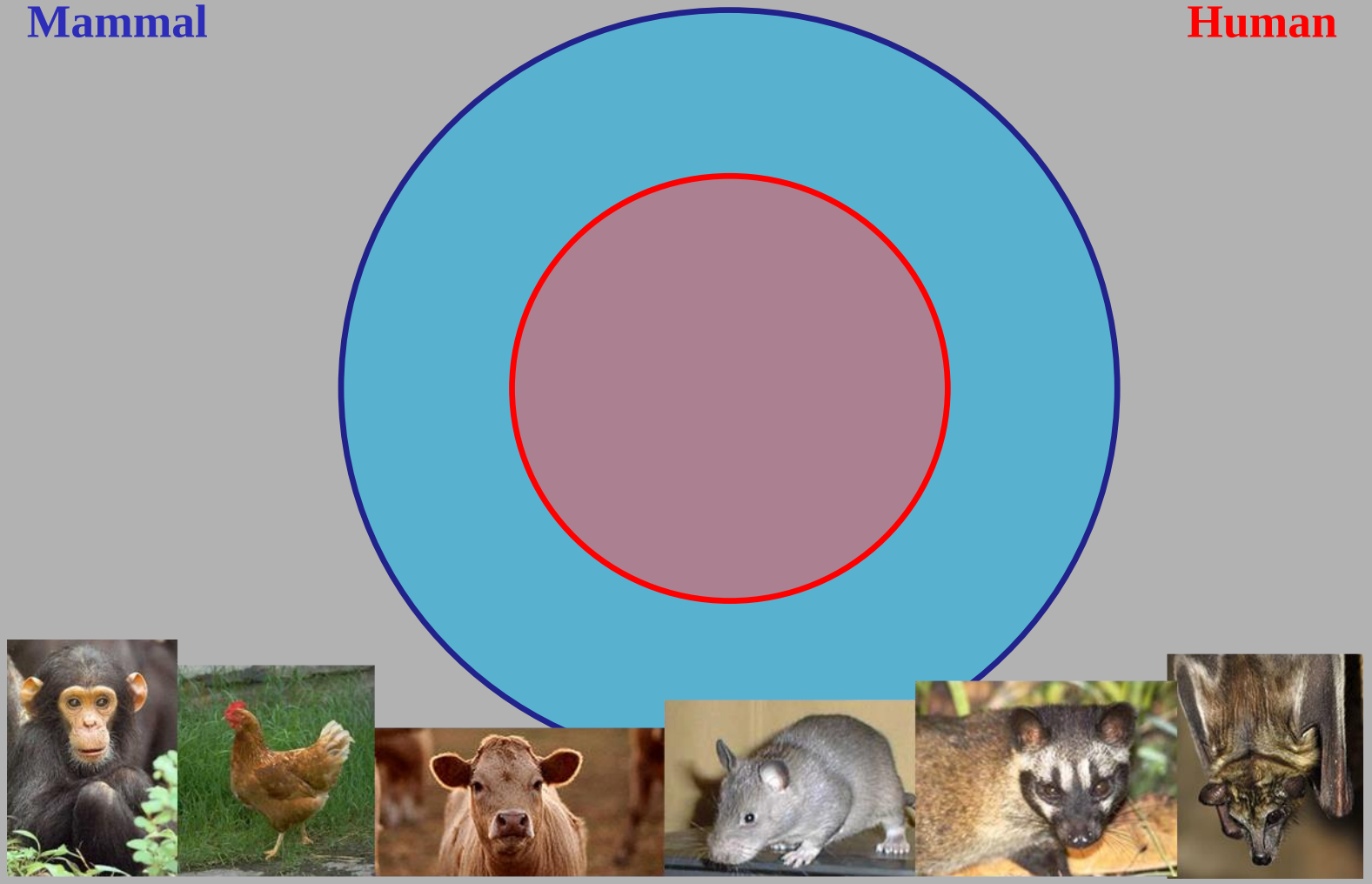
updated from Woolhouse *et al*. (2013) *Future Virol.*

HUMAN VIRUS, MAMMAL VIRUS



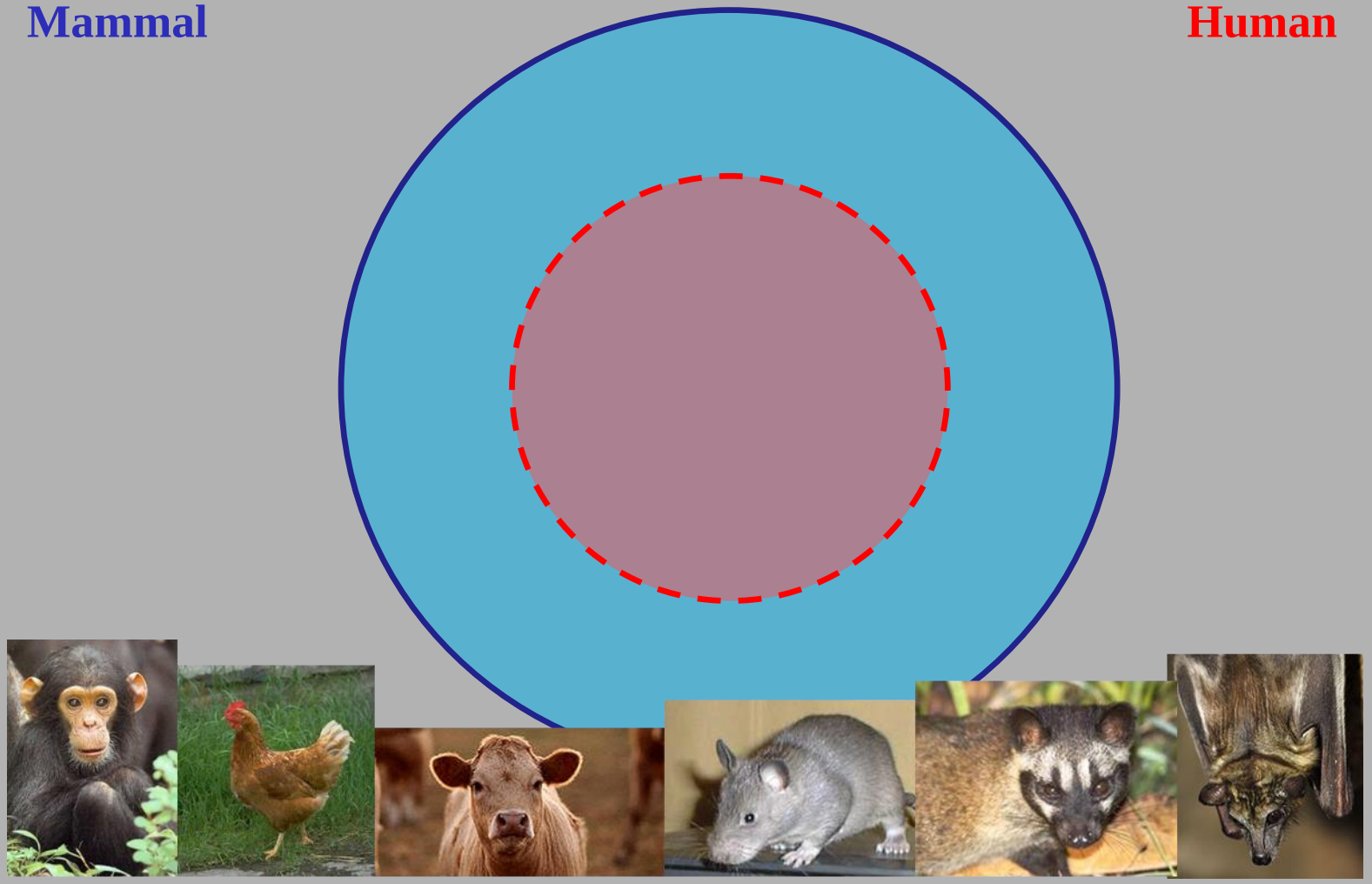
Woolhouse *et al*. (2014) in *One Health*

HUMAN VIRUS, MAMMAL VIRUS



Woolhouse *et al*. (2014) in *One Health*

HUMAN VIRUS, MAMMAL VIRUS

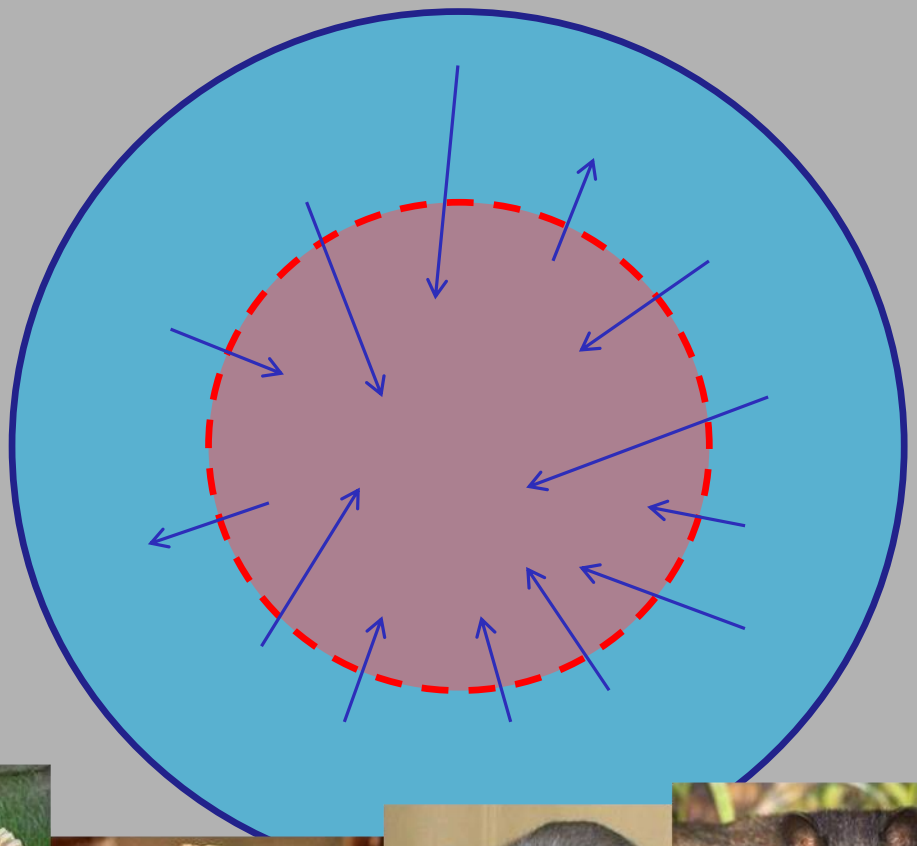


Woolhouse *et al*. (2014) in *One Health*

HUMAN VIRUS, MAMMAL VIRUS

Mammal

Human

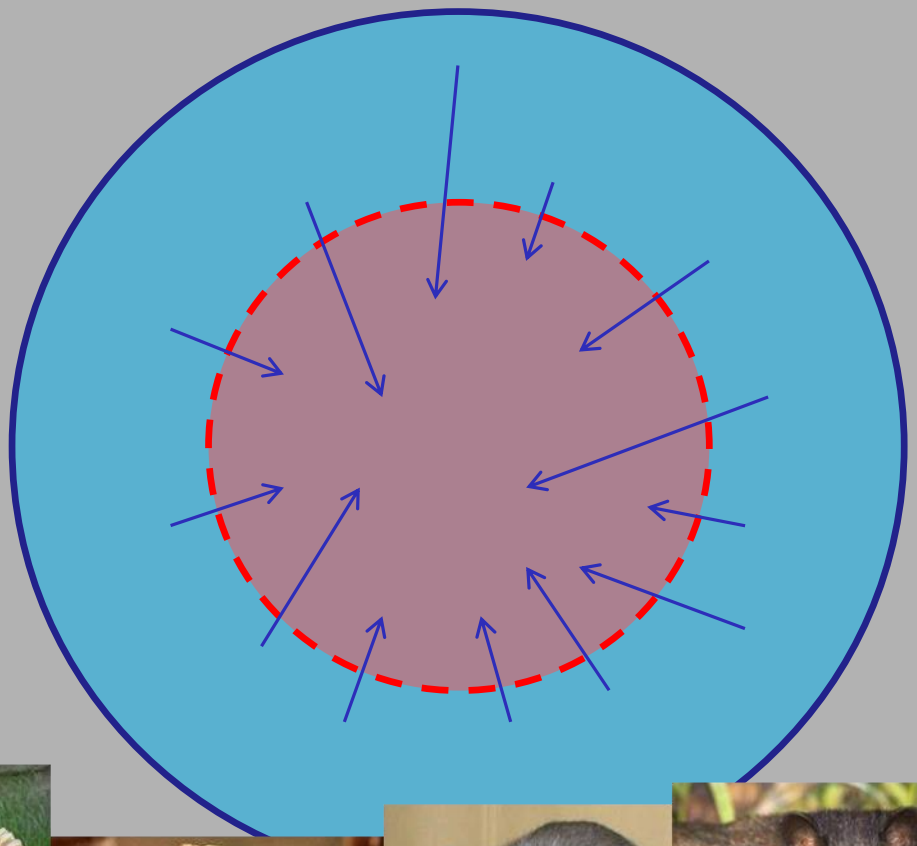


Woolhouse *et al*. (2014) in *One Health*

HUMAN VIRUS, MAMMAL VIRUS

Mammal

Human

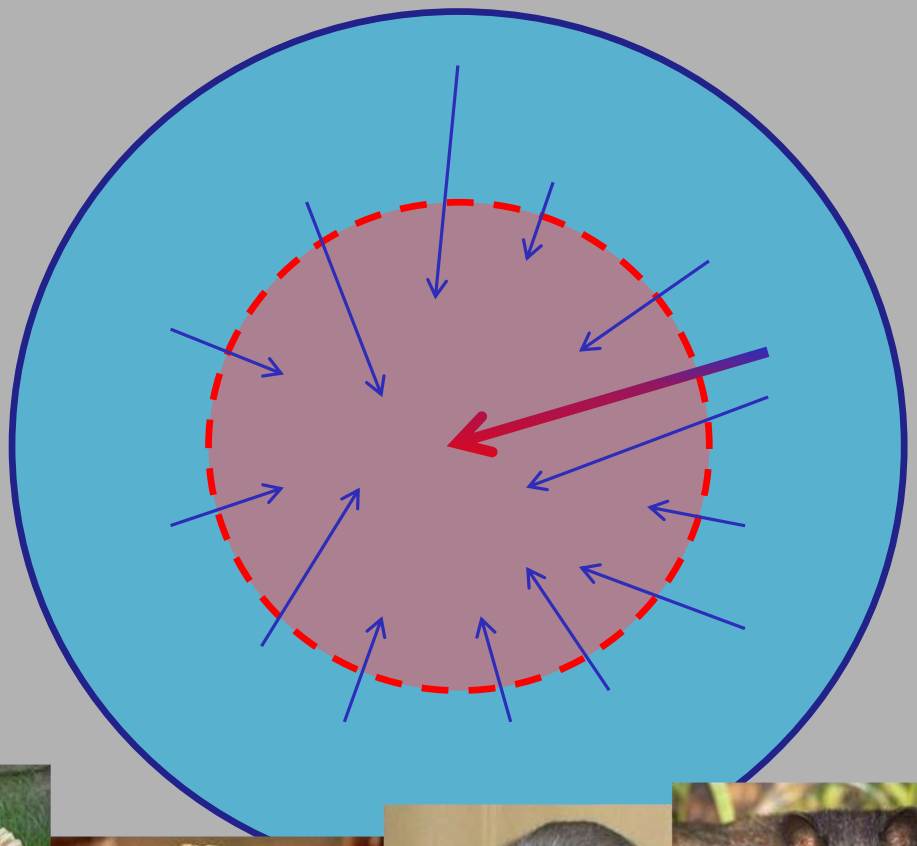


Woolhouse *et al*. (2014) in *One Health*

HUMAN VIRUS, MAMMAL VIRUS

Mammal

Human



Woolhouse *et al*. (2014) in *One Health*

DIVERSITY OF MAMMAL VIRUSES

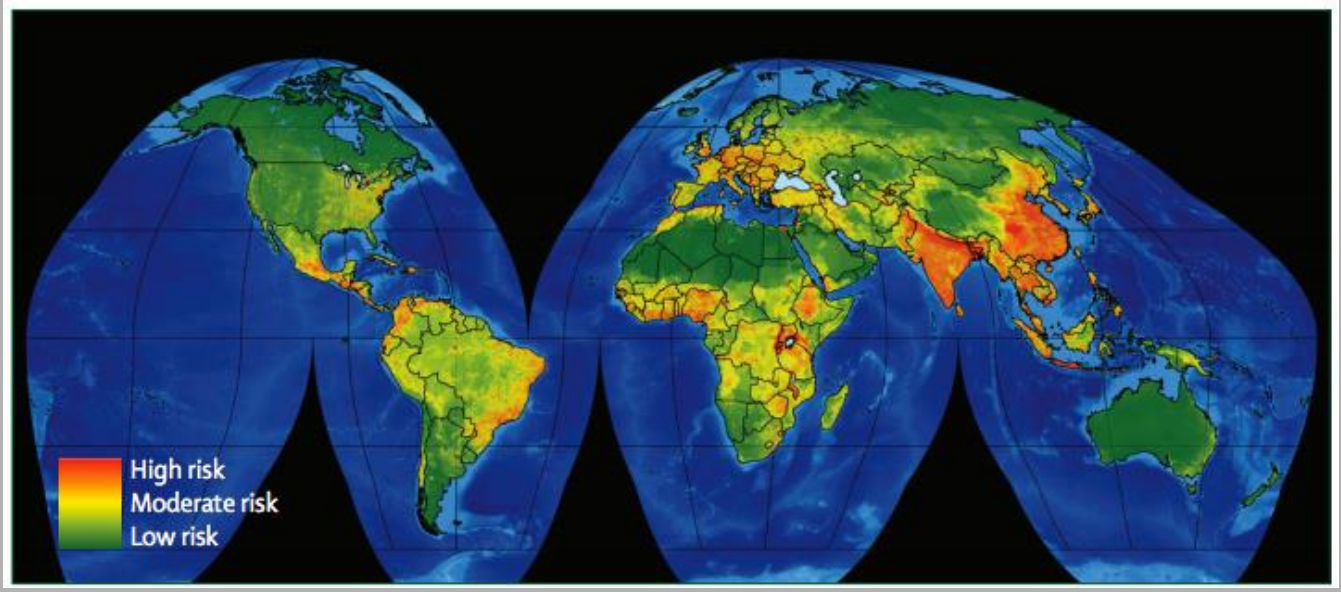
- 87M eukaryote virus species Geoghegan & Holmes (2017) *R. Soc. Open Biol.*
- >5000 species of mammal, 10 each Morse (1993) *Emerging Viruses* → 50,000
- Estimated 23 RNA viruses in one population of *Pteropus giganteus* Anthony *et al*. (2013) *mBio* → 100,000

? Do most mammals have any unique viruses at all? Critical community size

- Humans = 30% global land zoomass
- Livestock = 67%
- Wildlife = 3%

Smil (2012) *Harvesting the Biosphere*

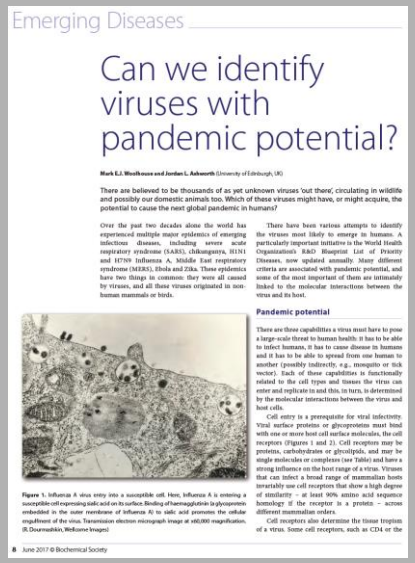
GLOBAL VIROME



GLOBAL VIROME PROJECT

(PREDICTIVE) GENOMIC SURVEILLANCE

- Sequence data easy to obtain
- Inferring phenotype from genotype not at all easy to do
- Key trait is cell receptor usage
- Know 78 different receptors from 94 human-infective viruses (across 19 families)
- Receptor →
  - Infectivity
  - Tissue tropism →
    - Pathogenicity
    - Transmissibility


Emerging Diseases

Can we identify viruses with pandemic potential?

Mark E.J. Woolhouse and Jordan L. Ashworth (University of Edinburgh, UK)

There are believed to be thousands of as yet unknown viruses 'out there', circulating in wildlife and possibly our domestic animals too. Which of these viruses might have, or might acquire, the potential to cause the next global pandemic in humans?

Over the past two decades alone the world has experienced multiple major epidemics of emerging infectious diseases, including severe acute respiratory syndrome (SARS), chikungunya, H1N1 and H7N9 influenza A, Middle East respiratory syndrome (MERS), Ebola and Zika. These epidemics have two things in common: they were all caused by viruses, and all those viruses originated in non-human mammals or birds.

There have been various attempts to identify the viruses most likely to emerge in humans. A particularly important initiative is the World Health Organization's R&D Blueprint List of Priority Diseases, now updated annually. Many different criteria are associated with pandemic potential, and some of the most important of them are intimately linked to the molecular interactions between the virus and its host.

Pandemic potential

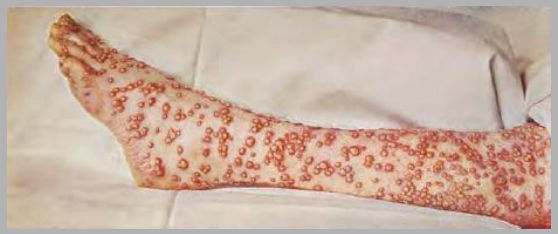
There are three capabilities a virus must have to pose a large-scale threat to human health: it has to be able to infect humans, it has to cause disease in humans and it has to be able to spread from one human to another (possibly indirectly, e.g., mosquito or tick vector). Each of these capabilities is functionally related to the cell types and tissues the virus can enter and replicate in and this, in turn, is determined by the molecular interactions between the virus and host cells.

Cell entry is a prerequisite for viral infectivity. Viral surface proteins or glycoproteins must bind with one or more host cell surface molecules, the cell receptors (Figures 1 and 2). Cell receptors may be proteins, carbohydrates or glycolipids, and may be single molecules or complexes (see Table) and have a strong influence on the host range of a virus. Viruses that can infect a broad range of mammalian hosts invariably use cell receptors that show a high degree of similarity – at least 90% amino acid sequence homology if the receptor is a protein – across different mammalian orders.

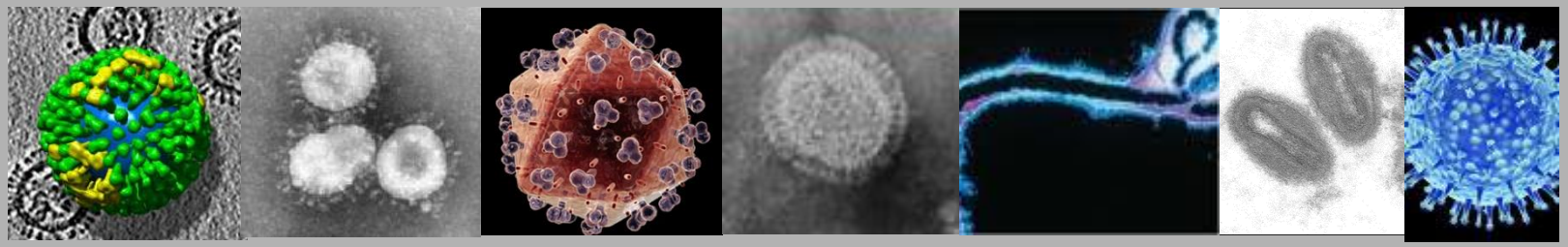
Cell receptors also determine the tissue tropism of a virus. Some cell receptors, such as CD4 or the

Figure 1. Influenza A virus entry into a susceptible cell. Here, influenza A is entering a susceptible cell expressing sialic acid on its surface. Binding of haemagglutinin (a glycoprotein embedded in the outer membrane of influenza A) to sialic acid promotes the cellular engulfment of the virus. Transmission electron micrograph image at x60,000 magnification. (R. Dourmashkin, Wellcome Images)

8 June 2017 © Biochemical Society



Woolhouse & Ashworth (2017) *Biochemist*



WHAT WILL CAUSE THE NEXT PANDEMIC?

- Discovery
- Human virus – Mammal virus
- **Prioritisation**
- Response

THE VIRUS PYRAMID

LEVEL 4

EPIDEMIC SPREAD

***N* = 60**

$R_0 > 1$

LEVEL 3

TRANSMISSION

***N* = 31**

LEVEL 2

INFECTION

***N* = 123**

LEVEL 1

EXPOSURE

***N* = ???**

updated from Woolhouse *et al*. (2014) in *One Health*

CHANGING LEVELS

SYNOPSIS

Assessing the Epidemic Potential of RNA and DNA Viruses

Mark E.J. Woolhouse, Liam Brierley, Chris McCaffery, Sam Lycett

Many new and emerging RNA and DNA viruses are zoonotic or have zoonotic origins in an animal reservoir that is usually mammalian and sometimes avian. Not all zoonotic viruses are transmissible (directly or by an arthropod vector) between human hosts. Virus genome sequence data provide the best evidence of transmission. Of human transmissible virus, 37 species have so far been restricted to self-limiting outbreaks. These viruses are priorities for surveillance because relatively minor changes in their epidemiologies can potentially lead to major changes in the threat they pose to public health. On the basis of comparisons across all recognized human viruses, we consider the characteristics of these priority viruses and assess the likelihood that they will further emerge in human populations. We also assess the likelihood that a virus that can infect humans but is not capable of transmission (directly or by a vector) between human hosts can acquire that capability.

A series of recent emerging infectious disease outbreaks, including the 2014 Ebola virus disease (EVD) epidemic in West Africa and the continuing Zika virus disease epidemic in the Americas, have underlined the need for better understanding of which kinds of pathogens are most likely to emerge and cause disease in human populations. Many, although not all, emerging infectious diseases are caused by viruses, and these frequently emerge from nonhuman host reservoirs (*1*–*3*). The enormous diversity (*4*) and high rates of evolution (*5*) of viral pathogens discourage attempts to predict with any precision which ones are most likely to emerge in humans. However, there is some consensus, at least in general terms, regarding the kinds of traits that are most essential in determining the capacity of a virus to infect, cause disease, and spread within human populations (Table 1). We focus on one of these traits, the capacity of a virus to spread from one human to another (by any transmission route other than deliberate laboratory exposure), a key determinant of the epidemic potential of a virus.

A theoretical framework for studying the dynamics of infectious disease outbreaks is well established (*6*). The capacity of an infectious disease to spread in a host population can be quantified in terms of its basic reproduction number, R_0. R_0 is defined as the average number of secondary cases generated by a single primary case in a large, previously unexposed host population, and its value tells us a great deal about the epidemiology of a pathogen. $R_0 = 0$ indicates no spread in that population; this value would apply to zoonotic infections that do not spread between humans. R_0 in the range $0<R_0\leq1$ indicates that chains of transmission are possible but that outbreaks will ultimately be self-limiting. $R_0>1$ indicates that major epidemics can occur or that the disease may become endemic in that host population. A higher value of R_0 also indicates that a greater reduction in transmission rates must be achieved to control an epidemic (*6*). R_0 values have been estimated for >60 common human pathogens (*7*), including human influenza A virus ($R_0\leq2$), measles virus ($R_0\leq18$), and dengue virus ($R_0\leq22$).

R_0 is determined by a combination of pathogen traits, such as its transmission biology, which is itself a complex interplay between the within-host dynamics of the pathogen and the host response to infection, and host traits, such as demography, behavior, genetics, and adaptive immunity. Consequently, for any given infectious disease, R_0 can vary between host species and between host populations. Infectious diseases with R_0 close to 1 are a particular concern because small changes in their epidemiologies can lead to major changes in the threat they pose to public health (*8*).

R_0 is closely related to another conceptual approach to disease emergence, the pathogen pyramid. There are different versions of this scheme (*3*,*9*). We consider a pyramid of 4 levels (Figure 1). Level 1 represents the background chatter of pathogens to which humans are continually or sporadically exposed but most of which are not capable of causing infection. Other levels can be considered in terms of the R_0 of the pathogen in humans: level 2 corresponds to $R_0 = 0$, level 3 to $0<R_0\leq1$, and level 4 to $R_0>1$.

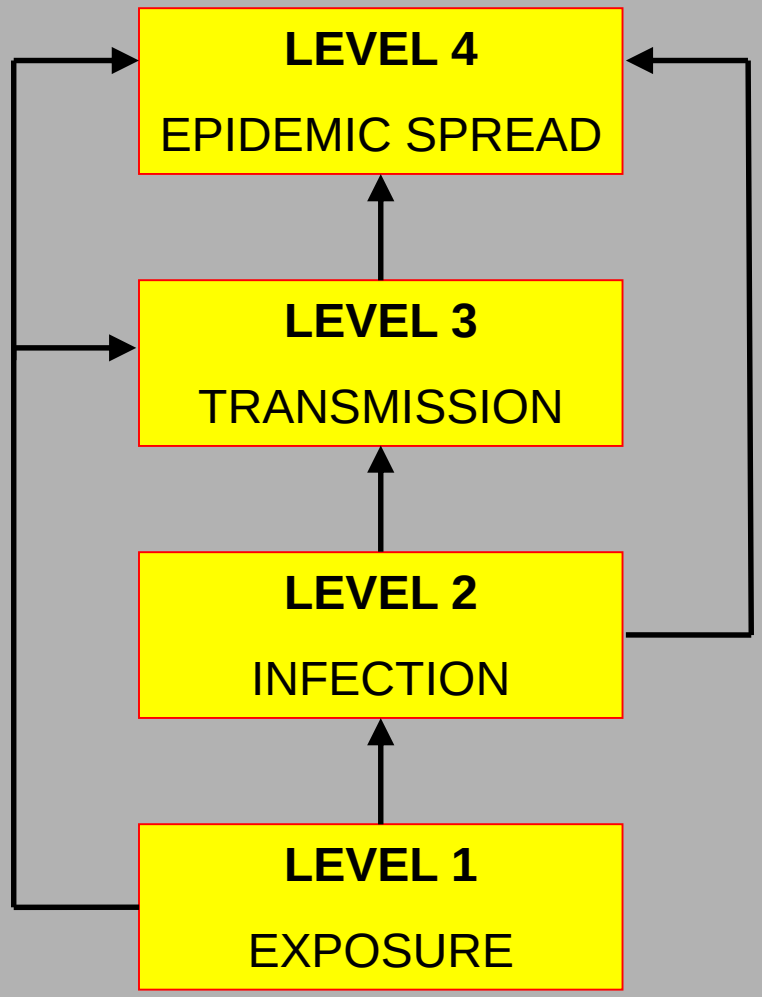
Data and Analysis

Identifying and Characterizing Level 3 and 4 Viruses

We updated our previous systematic literature review (*10*) of the capacity of virus species to transmit between humans (i.e., level 3 and level 4 viruses; online Technical Appendix, http://wwwnc.cdc.gov/EID/article/22/12/16-0123- Techapp1.pdf). Such viruses are

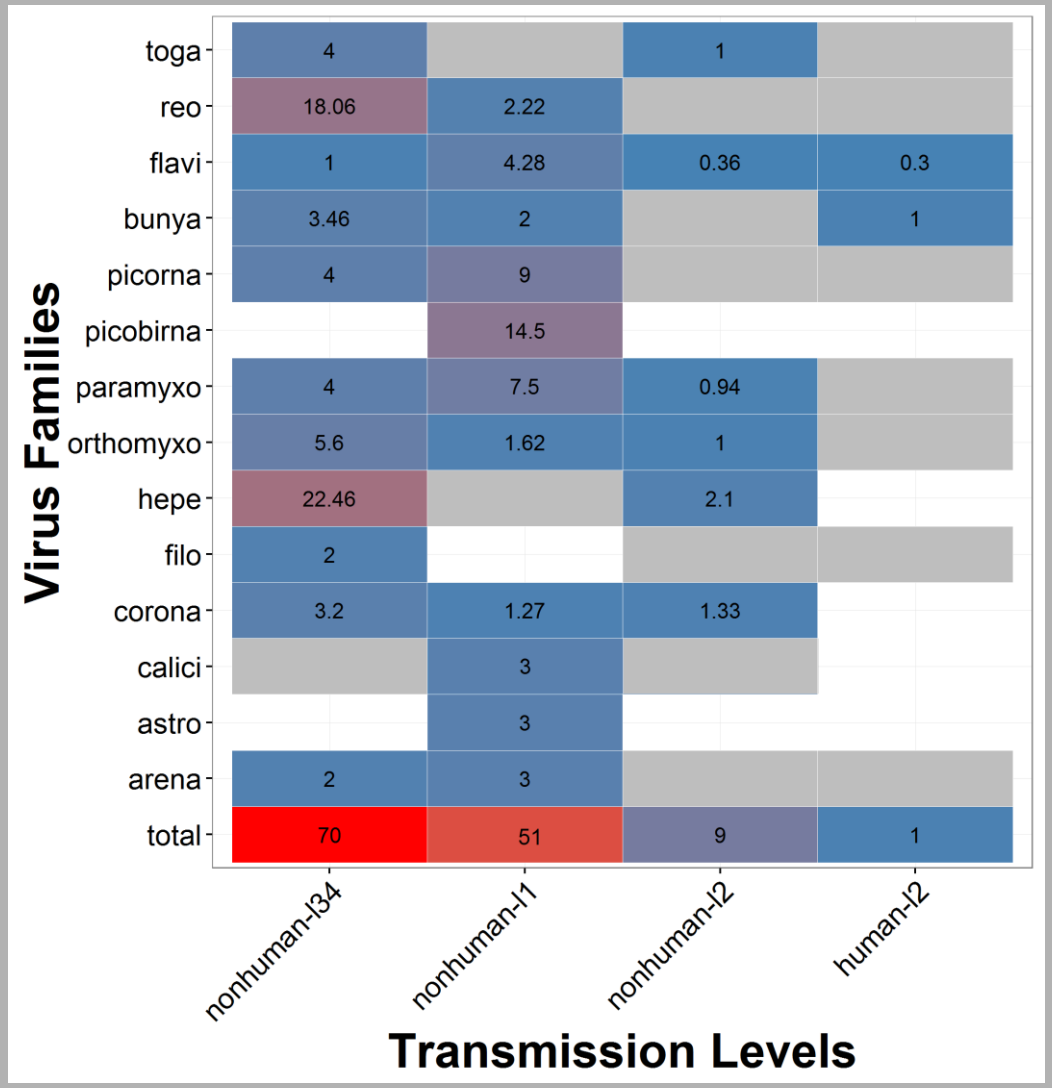
Author affiliation: University of Edinburgh, Edinburgh, UK

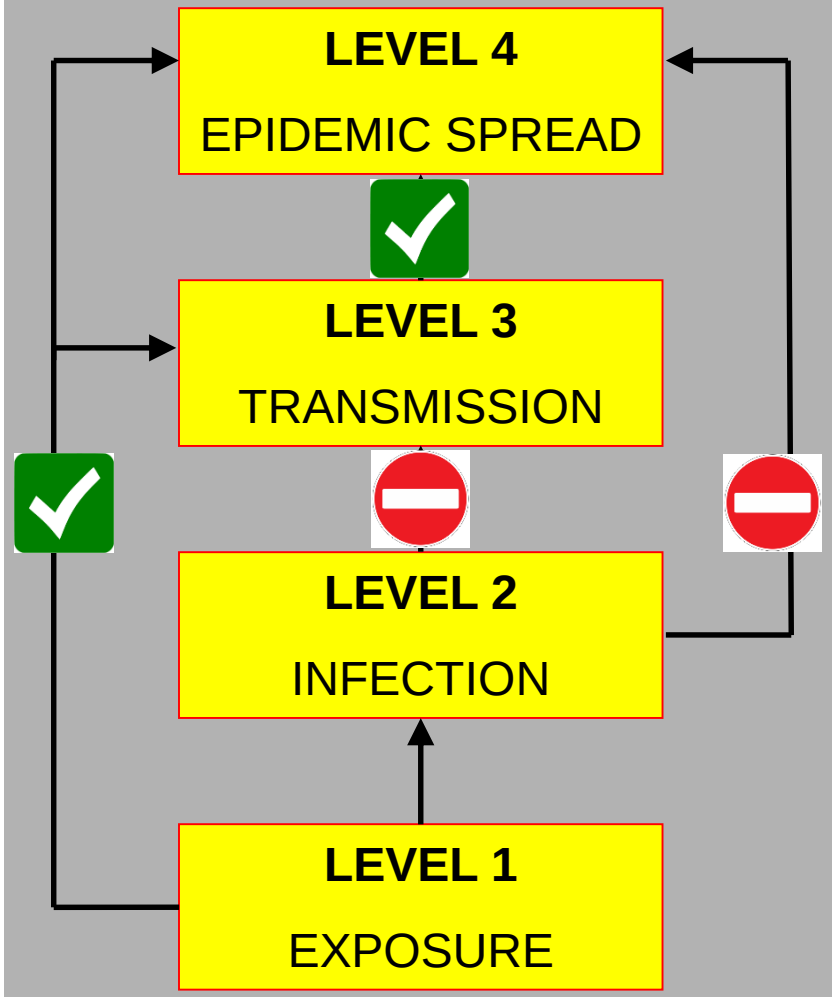
DOI: http://dx.doi.org/10.3201/eid2212.160123



Woolhouse *et al*. (2016) *Emerg. Infect. Dis.*

CHANGING LEVELS



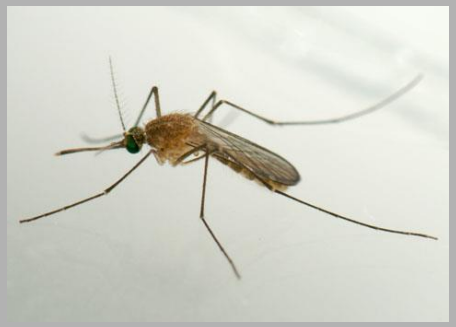


Mark Woolhouse, University of Edinburgh, January 2018

Thanks to Lu Lu, Alex Bhattacharya

AN EXAMPLE: ARBOVIRUSES

- 91 human RNA virus species from 8 families are transmitted by vectors
- All 19 level 3/4 RNA arboviruses are carried by anthropophilic vectors
- Main anthropophilic vector species are from 5 dipteran genera:
  - *Aedes* spp.
  - *Anopheles* spp.
  - *Culex* spp.
  - *Culicoides* spp.
  - *Phlebotomus* spp.
- All four Level 4 arboviruses [YFV, DENV, CHIK + ZIKA] are carried by *Aedes* spp.
- There are no anthropophilic ticks: so no Level 4 tick-borne viruses



Woolhouse *et al*. (2016) *Emerg. Infect. Dis.*

‘LEVEL 3’ RNA VIRUS SPECIES

Arenaviruses
(Dandenong*)
Guanarito
Junin
Lassa
Lujo
(Lymphocytic choriomeningitis)
Machupo
Sabia

Bunyaviruses
Andes
Bwamba
Crimean-Congo haemorrhagic fever
Oropouche
Rift Valley
Severe fever with thrombocytopenia syndrome

Coronaviruses
Middle East respiratory syndrome

Filoviruses
Bundibugyo ebola
Lake Victoria marburg
Sudan ebola
Zaire ebola

Flaviviruses
(Japanese encephalitis)
(Usutu)
(West Nile)
Zika

Paramyxoviruses
Nipah

Reoviruses
(Colorado tick fever)
Nelson Bay
Rotavirus H

Rhabdoviruses
Bas-congo*
(Rabies)

Togaviruses
Barmah forest
Chikungunya
O’nyong-nyong
Ross river
Semliki forest
Venezuelan equine encephalitis

N = 28
- includes 7 viruses known only through iatrogenic and/or vertical routes (parentheses)
- 8 with outbreaks >100 cases (bold)

*not ICTV recognised

Mark Woolhouse, University of Edinburgh, January 2018

adapted from Woolhouse *et al*. (2016) *Emerg. Infect. Dis.*

‘LEVEL 3’ RNA VIRUS SPECIES

Arenaviruses

Guanarito
Junin
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Bwamba
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Oropouche
Rift Valley
Severe fever with thrombocytopenia syndrome

Coronaviruses
Middle East respiratory syndrome

Filoviruses
Bundibugyo ebola
Lake Victoria marburg
Sudan ebola
Zaire ebola

Flaviviruses

Zika

Paramyxoviruses
Nipah

Reoviruses

Nelson Bay
Rotavirus H

Rhabdoviruses
Bas-congo*

Togaviruses
Barmah forest
Chikungunya
O’nyong-nyong
Ross river
Semliki forest
Venezuelan equine encephalitis

N = 25
- includes 7 viruses known only through iatrogenic and/or vertical routes (parentheses)

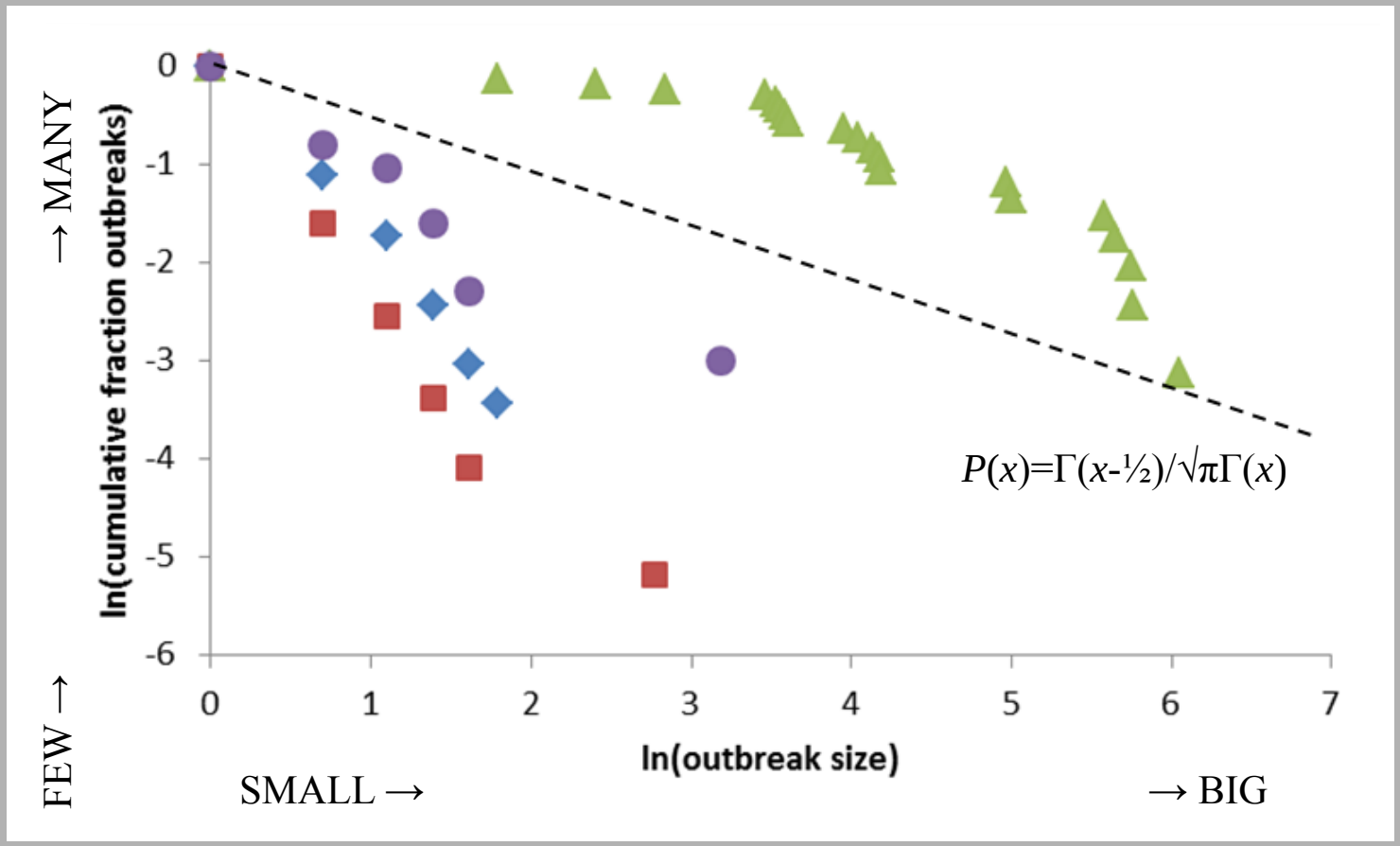
5 with outbreaks >100 cases (bold)

*not ICTV recognised

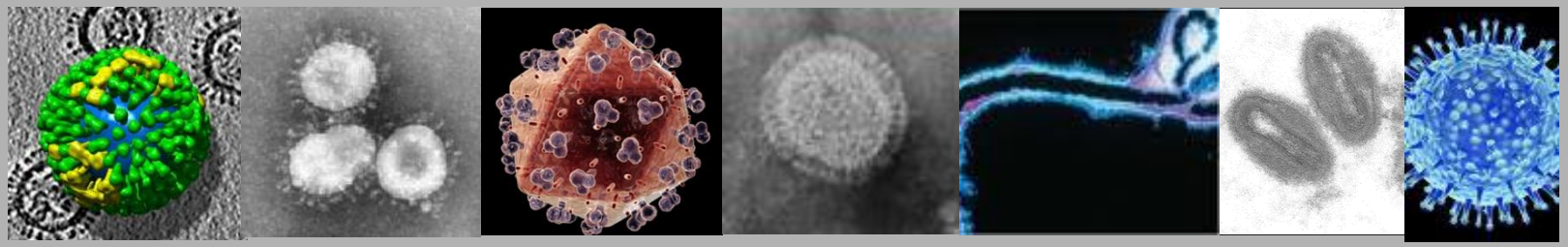
Mark Woolhouse, University of Edinburgh, January 2018

adapted from Woolhouse *et al*. (2016) *Emerg. Infect. Dis.*

OUTBREAK DYNAMICS



Woolhouse *et al*. (2016) *Emerg. Inf. Dis.*



WHAT WILL CAUSE THE NEXT PANDEMIC?

- Discovery
- Human virus – Mammal virus
- Prioritisation
- **Response**

RESPONSE PRIORITIES

Coalition for Epidemic Preparedness Innovations

CEPI
New vaccines
for a safer world

Mission Approach Governance Partners News Calls Resources

Priority diseases Regulatory affairs Preliminary Business Plan 2017-2021 Meetings

CEPI will initially target the MERS-CoV, Lassa and Nipah viruses, which have known potential to cause serious epidemics. It aims to develop two promising vaccine candidates against each of these diseases, so these are available without delay if and when an outbreak begins.

CEPI took the WHO's R&D Blueprint for Action to Prevent Epidemics as its starting point. This contains a list of priority pathogens against which medical countermeasures are urgently needed. CEPI's Scientific Advisory Committee chose these three diseases based on a set of criteria including the public health impact, the risk of an outbreak occurring and the feasibility of vaccine development, based on current knowledge, tools and pipeline candidates.

More information about the priority diseases can be found here:

- MERS-CoV
- Lassa virus
- Nipah virus

You can find information about the pipeline dataset for MERS, Lassa and Nipa vaccine candidates here:

RESPONSE PRIORITIES

World Health Organization

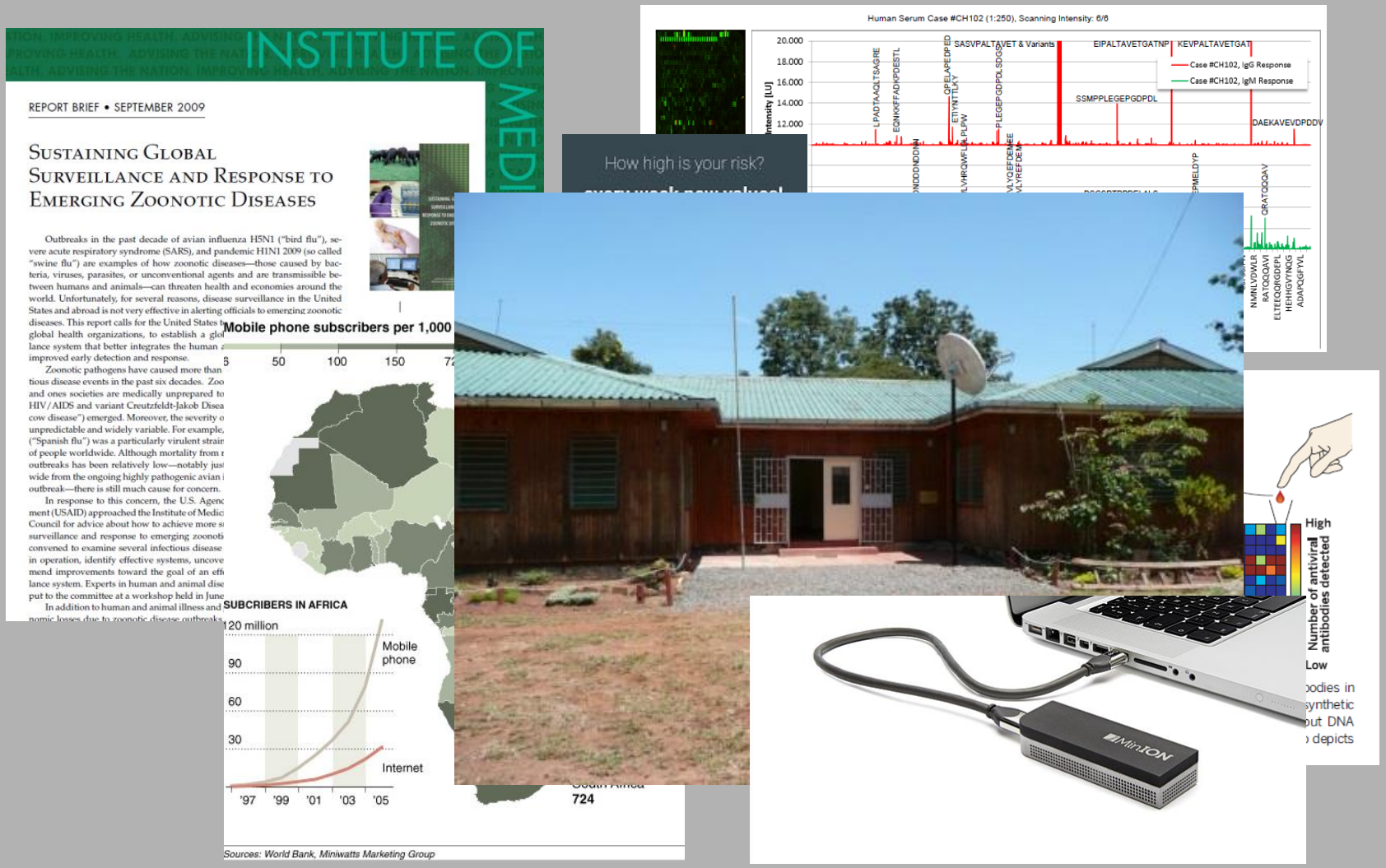
LIST OF TOP EMERGING DISEASES LIKELY TO CAUSE MAJOR EPIDEMICS

The 2017 list of disease priorities needing urgent R&D attention comprises:

Arenaviral hemorrhagic fevers (including Lassa Fever)
Crimean Congo Haemorrhagic Fever (CCHF)
Filoviral diseases (including Ebola and Marburg)
Middle East Respiratory Syndrome Coronavirus (MERS-CoV)
Other coronaviral diseases (such as Severe Acute Respiratory Syndrome, (SARS))
Nipah and related henipaviral diseases
Rift Valley Fever (RVF)
Severe Fever with Thrombocytopenia Syndrome (SFTS)
Zika
Disease X

The list will be updated annually or when new diseases emerge

DIAGNOSTICS AND SURVEILLANCE



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ADDING VALUE TO DATA

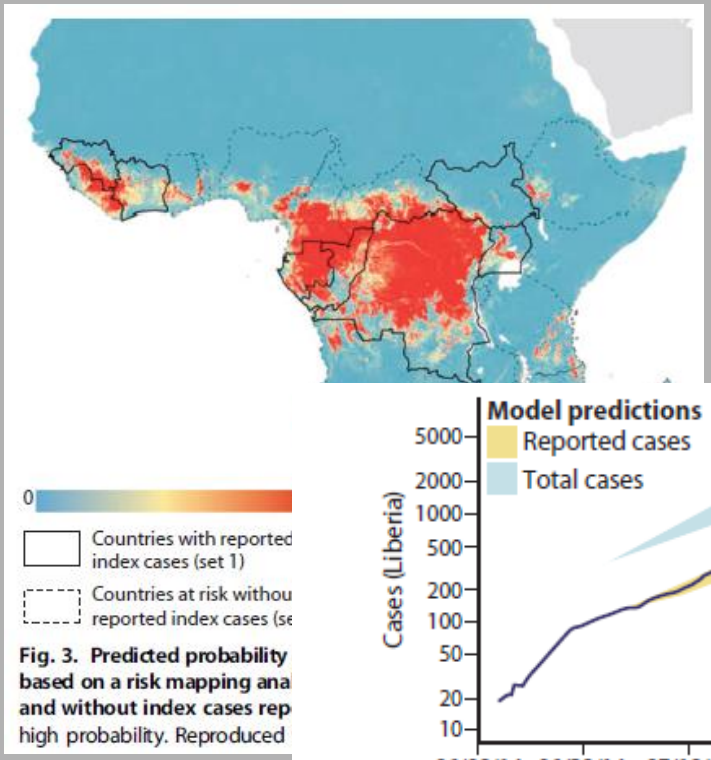
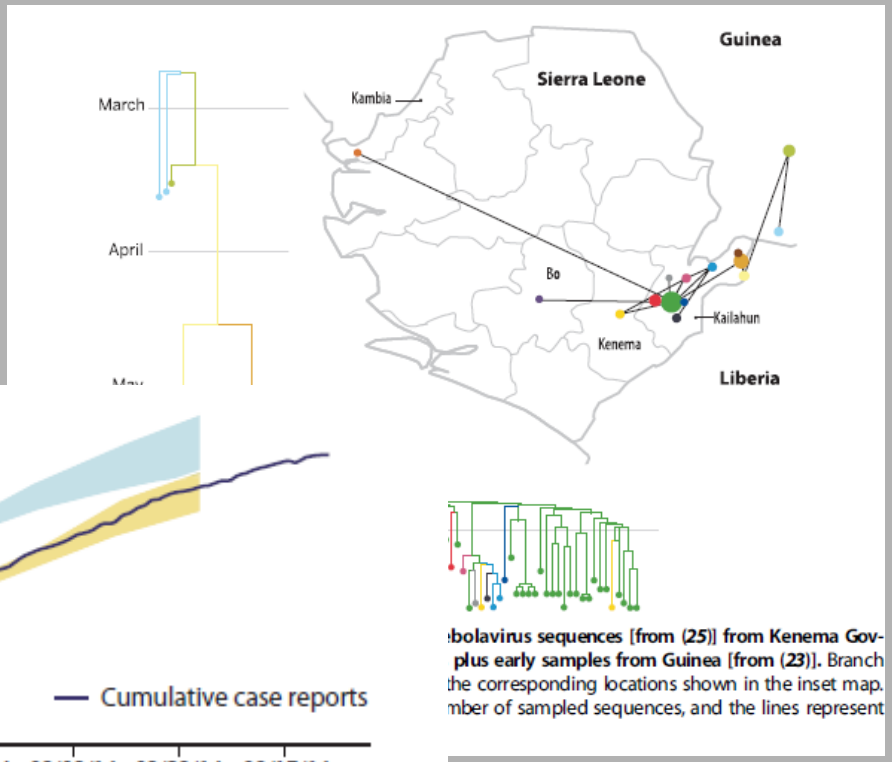


Fig. 3. Predicted probability based on a risk mapping ana and without index cases rep high probability. Reproduced



bolavirus sequences [from (25)] from Kenema Gov- plus early samples from Guinea [from (23)]. Branch the corresponding locations shown in the inset map. mber of sampled sequences, and the lines represent

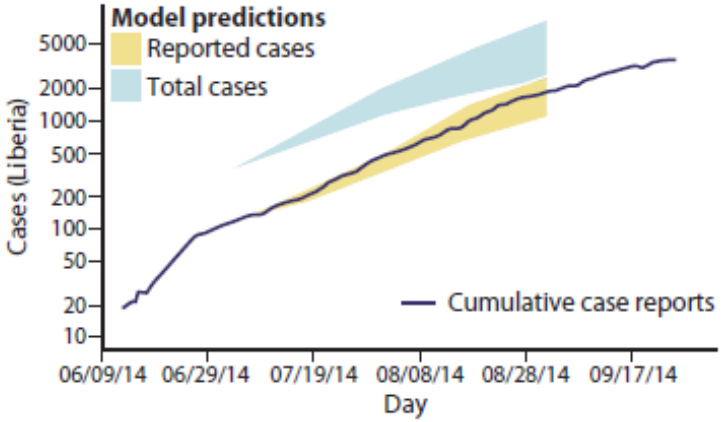


Fig. 2. Projected numbers of cases of EVD in Liberia in 2014 obtained using a branching process model with an ensemble of plausible parameter values. The 95% prediction intervals from 4 July 2014 (yellow shading) are compared with the observed cumulative case numbers (logarithmic scale) over the following 2 months (blue line). The 95% prediction intervals for a model that incorporates estimated levels of underreporting are also shown (blue shading). Reproduced with authors' permission from (*32*).

Woolhouse *et al*. (2015) *Sci. Trans. Med.*

OPENNESS AND DATA SHARING

CalID	VisitID	calID_visitID	VisitDate	CADOB	CADOD	Age at VO	Age class	RT	LN	ECF	Status	RLB.T.pan	RLB.T.taur	RLB.T.mut	RLB.T.welf	RLB.T.sp.s
CA010110012	VPC010012	CA010110012_VPC010012	31/08/2008	08/06/2008	31/08/2008	84	2	NA	NA	NA	1	NA	NA	NA	NA	NA
CA010110025	VRC160025	CA010110025_VRC160025	09/09/2009	05/06/2009	NA	96	2	39.7	0	0	0	1	0	0	0	0
CA010210035	VRC160035	CA010210035_VRC160035	02/07/2008	13/03/2008	NA	111	3	39.2	0	0	0	0	1	1	0	0
CA010210036	VRC160036	CA010210036_VRC160036	02/07/2008	12/03/2008	14/07/2008	112	3	40.4	0	1	1	1	1	0	0	0
CA010210038	VCC010038	CA010210038_VCC010038	18/06/2008	18/04/2008	NA	61	2	39.8	1	0	0	1	0	0	0	0
CA010210046	VCC010046	CA010210046_VCC010046	09/12/2008	12/11/2008	NA	27	1	40.8	1	1	0.5	1	0	0	0	0
CA010210058	VRC160058	CA010210058_VRC160058	13/01/2010	17/09/2009	NA	118	3	40.7	0	0	0	1	0	1	0	0
CA010310071	VRC060071	CA010310071_VRC060071	11/10/2008	27/08/2008	NA	45	1	40.8	1	1	0.5	1	0	0	0	0
CA010310074	VRC060074	CA010310074_VRC060074	17/12/2008	02/11/2008	NA	45	1	39.1	1	1	0.5	1	1	0	0	0
								38.8	0	0	0	1	1	0	0	0
								NA	NA	NA	1	NA	NA	NA	NA	NA
								39.2	0	0	0	1	0	1	1	1
								40.8	1	1	1	1	0	1	1	1
								40.5	1	1	0.5	1	1	1	0	0
								38.9	0	0	0	1	0	1	1	0
								38.9	0	0	0	1	0	1	0	0
								39.4	1	0	0	1	0	1	0	0
								38	0	0	0	1	0	1	0	0
								38.4	1	0	0	1	0	0	0	0
								39.7	0	0	0	1	1	0	0	0
								41.2	1	1	1	1	0	0	0	0
								40.2	0	0	0	1	0	0	1	0
								41	1	2	0.5	1	0	0	0	0
								39.9	1	0	0	1	0	0	0	0
								39.1	0	0	0	1	0	1	0	0
								38.6	0	0	0	0	1	1	0	0
								IA	0	0	1	1	0	0	0	0
								37.9	0	0	0	0	0	1	1	1
								39.6	1	0	0	1	0	0	0	0
								41.3	1	1	0.5	1	0.5	1	0	0
								41.2	1	1	0.5	1	0	0.5	0	0
								41.4	1	1	1	1	0	0	0	0

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INFLUENZA

Role for migratory wild birds in the global spread of avian influenza H5N8

The Global Consortium for H5N8 and Related Influenza Viruses*†

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+ Foresight and IOM/NAS committees

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TACKLING INFECTIONS TO BENEFIT AFRICA

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