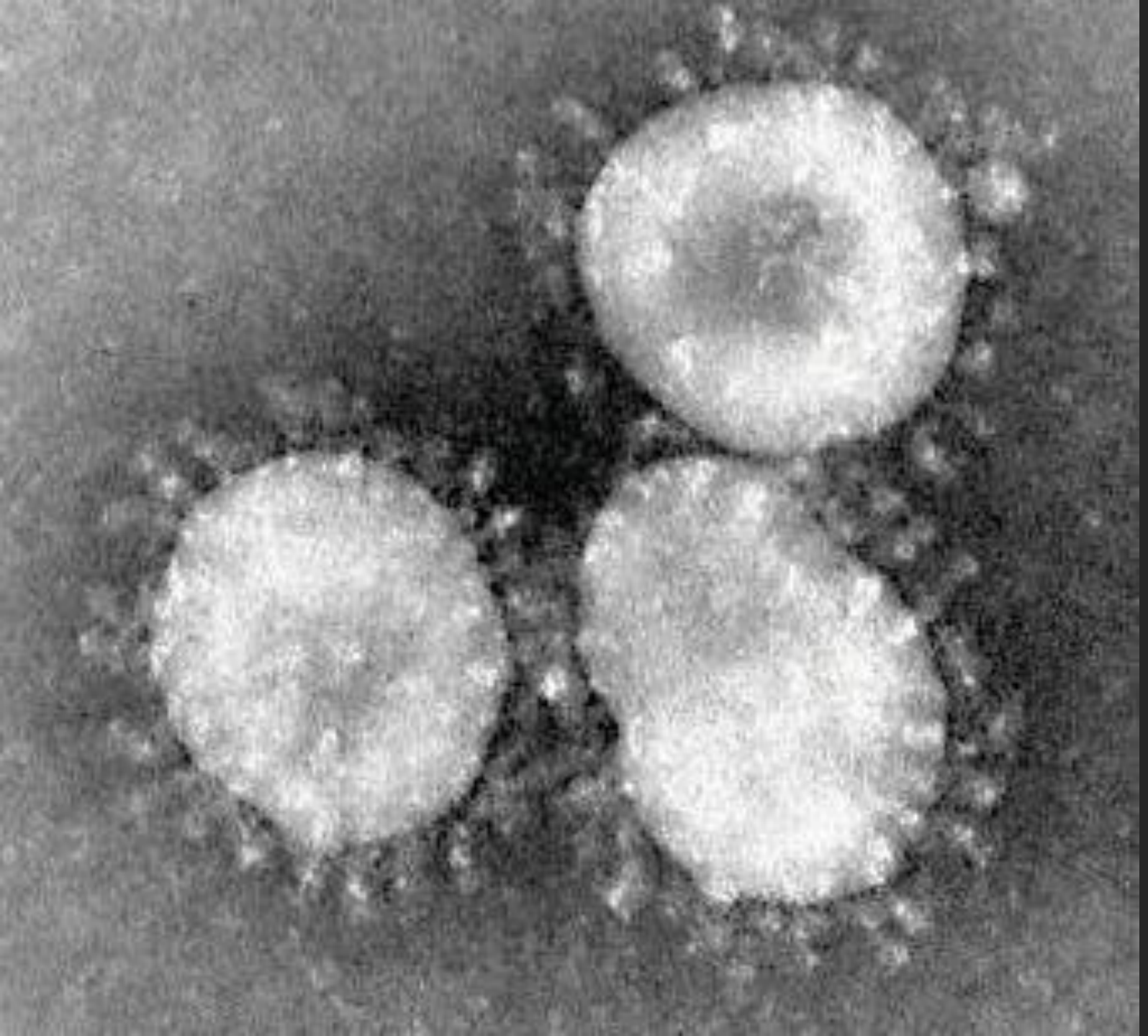

PERSPECTIVES ON COVID-2019

MICHAEL T. LeVASSEUR, PhD, MPH

FEBRUARY 29, 2020

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- Visiting Assistant Professor of Epidemiology & Biostatistics Dornsife School of Public Health
  - PhD in Epidemiology from Drexel University Dornsife School of Public Health (2012-2016)
  - MPH in Epidemiology and Biostatistics from CUNY Hunter School of Public Health (2009-2012)
  - BA in Liberal Arts with concentrations in Molecular Biology and Medical Anthropology from Sarah Lawrence College (2000-2004)
- Research interests include:
  - Infectious disease epidemiology
  - Gender and sexual minority health disparities
  - Data science
  - Programming
  - Big data analytics
  - Bioethics

OUTLINE

Pathophysiology

what is a coronavirus? what is the clinical course? how does this differ from similar viruses?

Origins

where did it come from?

Transmission

how is it spread?

Epidemiology

who is getting infected? who is dying? who is surviving?

Prevention

how can I prevent myself from getting infected? how can I protect my children?

Epidemiologic Forecast

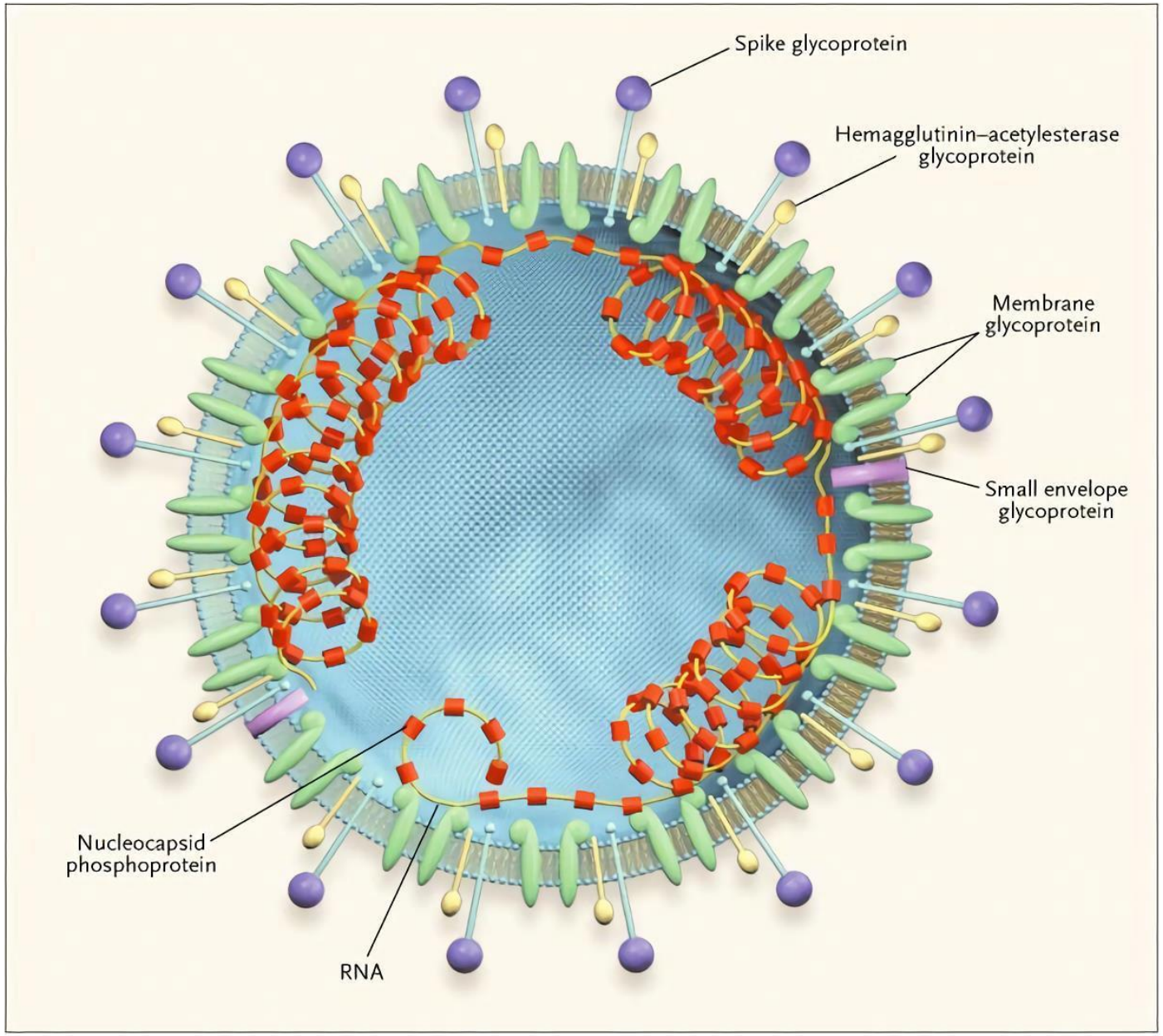
what is a pandemic and is this one? why haven't prevention measures worked? how worried should I be?

Other Questions

is the media reporting this accurately? what is the global impact?

S1: VIROLOGY AND PATHOPHYSIOLOGY

- Basics of coronaviruses
- Symptoms of coronaviruses
- Treatment
- Pathogenicity, virulence, and infectivity of COVID-19



CORONAVIRIDAE

Four subspecies:

Alphacoronaviruses and Betacoronaviruses – infect mammals

Gammecoronaviruses and Deltacoronaviruses – mostly infect birds (some may infect mammals)

Seven known coronaviruses that infect humans

15-29% of all common colds caused by coronaviruses

SARS, MERS, and COVID-19 are the only ones that cause death

CLINICAL SYMPTOMS OF CORONAVIRUSES

Coronavirus	Clinical Symptoms
229E and OC43	General malaise Headache Nasal discharge Sneezing Sore throat Fever and cough
NL63	Cough, Rhinorrhea Tachypnea Fever Hypoxia Obstructive laryngitis (croup)
HKU1	Fever Running nose Cough Dyspnea
SARS	Fever Myalgia Headache Malaise Chills Nonproductive cough Dyspnea Respiratory distress Diarrhea
MERS	Fever Cough Chills Sore throat Myalgia Arthralgia Dyspnea Pneumonia Diarrhea and vomiting Acute renal impairment
COVID-19	Fever Cough Dyspnea Pneumonia

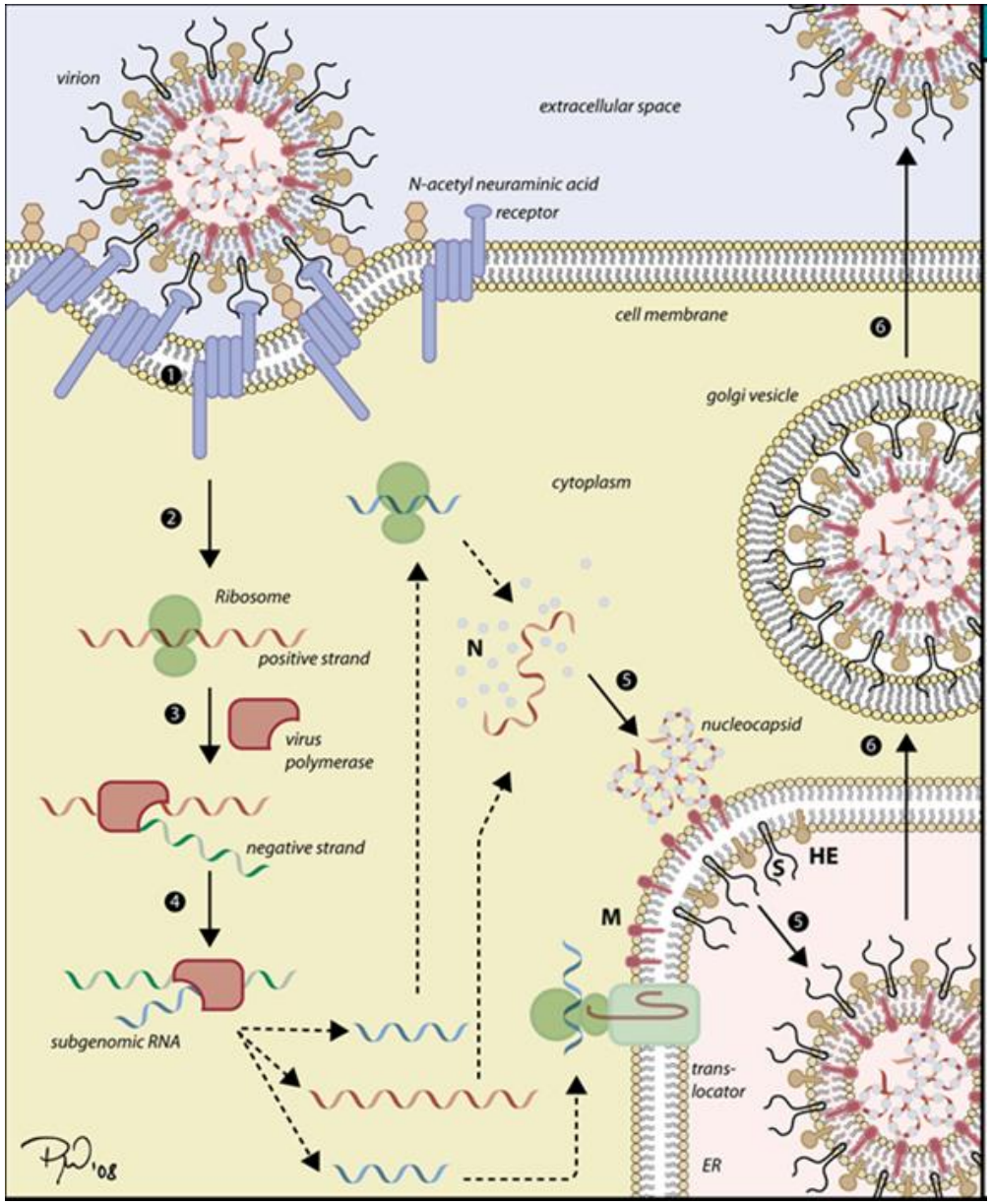
TREATMENT

Mild Cases

- Same things you would do for a cold
  - Bed rest
  - Medications to treat cough and fever
- Should someone receive care at home?
  - Patient is stable enough to receive care at home
  - Appropriate caregivers are available
  - There is a separate bedroom where the patient can recover without sharing immediate space with others.
  - Resources for access to food and other necessities are available
  - The patient and other household members have access to appropriate, recommended personal protective equipment (at a minimum, gloves and facemask) and are capable of adhering to precautions recommended as part of home care or isolation (e.g., respiratory hygiene and cough etiquette, hand hygiene)
  - There are household members who may be at increased risk of complications from 2019-nCoV infection (.e.g., people >65 years old, young children, pregnant women, people who are immunocompromised or who have chronic heart, lung, or kidney conditions)
  - https://www.cdc.gov/coronavirus/2019-ncov/hcp/guidance-home-care.html

Severe Cases

- Acute respiratory distress syndrome (ARDS) developed in 17–29% of hospitalized patients
- Between 23–32% of hospitalized patients with COVID-19 infection and pneumonia have required intensive care for respiratory support
- Among critically ill patients admitted to an intensive care unit
  - 11% received high-flow oxygen therapy
  - 42% received noninvasive ventilation
  - 47% received mechanical ventilation
  - 4-10% required advanced organ support (endotracheal intubation and mechanical ventilation)
  - 2-5% supported with extracorporeal membrane oxygenation (ECMO)
  - Also acute cardiac injury, arrhythmia, shock, and acute kidney injury.
  - https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html



HOW DO CORONAVIRUSES INFECT HUMANS

1) Virus attaches spike protein to cellular receptor (for SARS and COVID-19, this is ACE-2)
2) Virus enters the cell (exact mechanism is unclear)
3) Virus begins transcribing viral RNA directly in the cytoplasm
4) Proteins are synthesized in the endoplasmic reticulum
5) New virions are assembled in the golgi apparatus
6) New virions bud from the host cell and go on to infect other cells

RESEARCH

Medication

- Remdesivir
  - Developed by Gilead Sciences Inc.
  - Investigational broad-spectrum antiviral
  - Previously tested in humans with Ebola virus disease
  - Has shown promise in animal models for treating Middle East respiratory syndrome (MERS) and severe acute respiratory syndrome (SARS)
  - Currently undergoing RCT at University of Nebraska Medical Center in Omaha

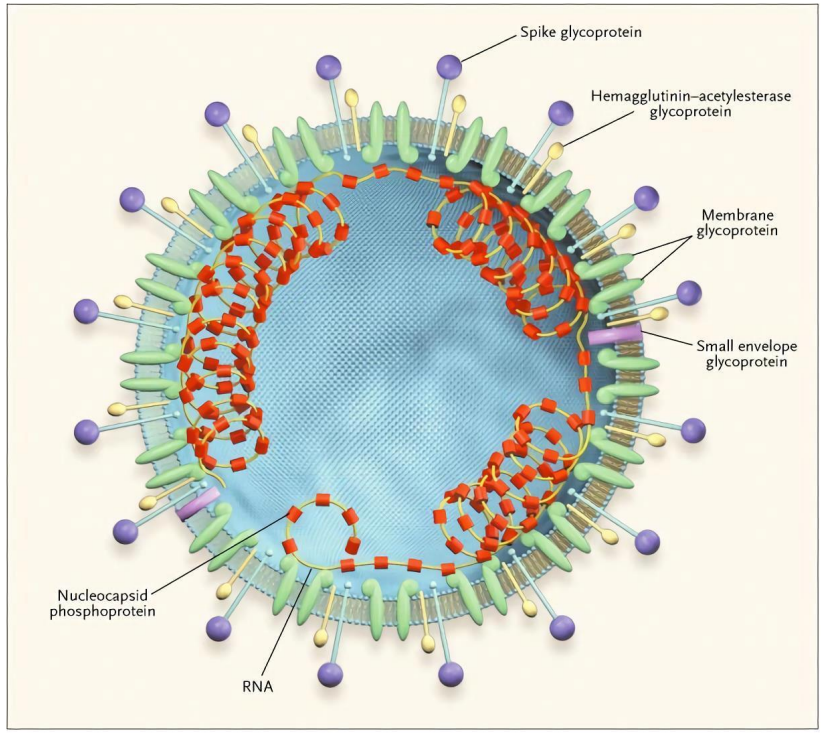
Vaccine

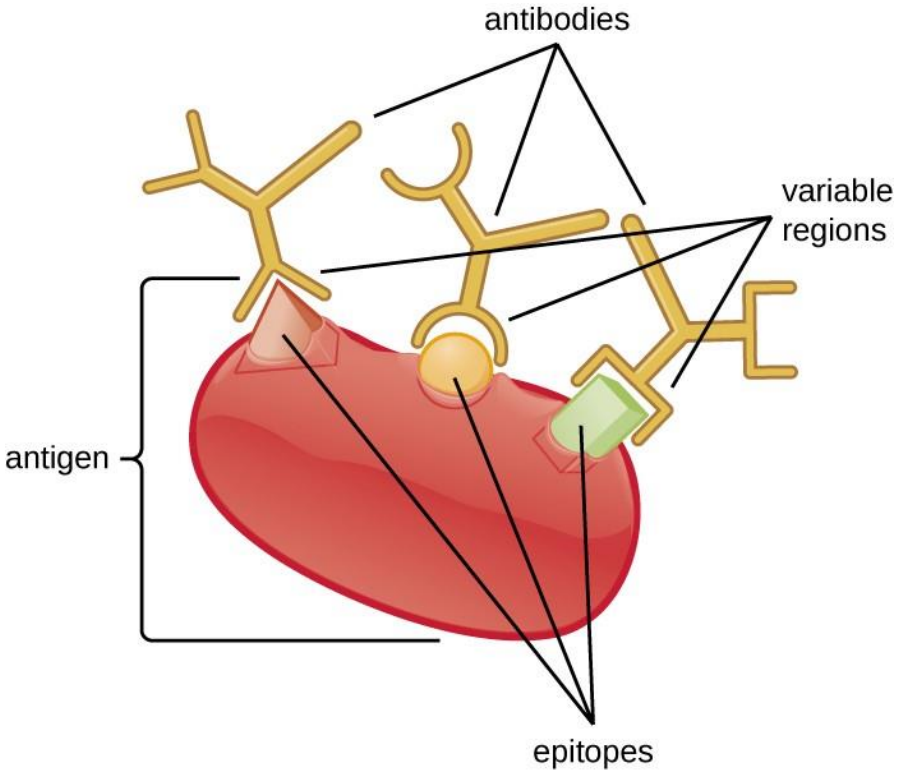
- SARS
  - IgG and neutralizing antibodies peak at 4 months post-infection and wane thereafter
  - 89% of patients still had a response after 24 months
  - 8.7% of patients still had a response after 6 years

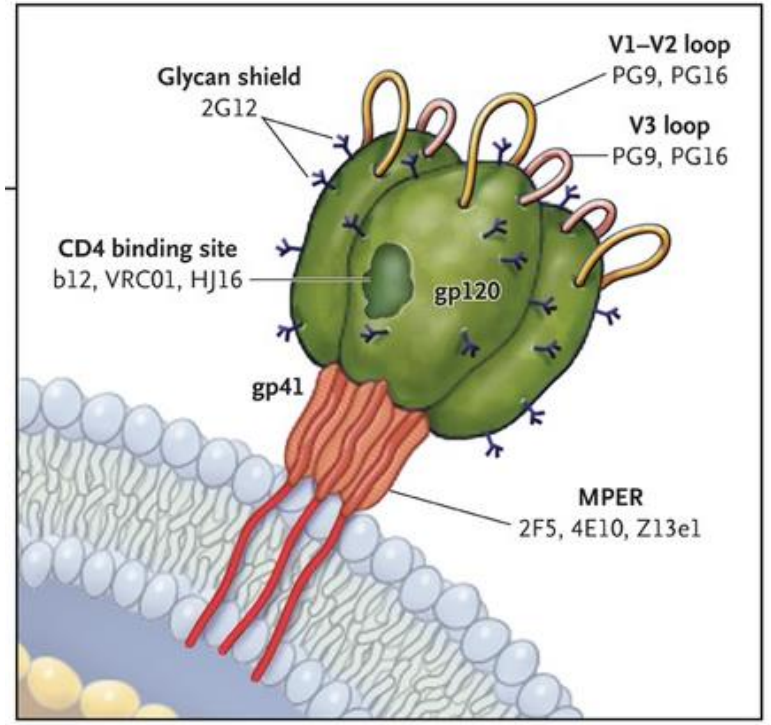
IMMUNE EVASION

- NSP1 inhibits phosphorylation of STAT1
  - When activated, STAT1 and STAT2 are phosphorylated and dimerize resulting in changes that turn the cell into an anti-viral state
- NSP1 inactivates translational capability of ribosomes
  - Ribosomes are cellular proteins that translate mRNA into new protein. If inactivated, a cell cannot do its job
- NSP3 interacts with IRF3 inhibiting phosphorylation, dimerization, and nuclear translocation
  - IRF3 is a transcription factor (think work order) for interferon
- NSP16 renders viral RNA indistinguishable from host T-cell RNA
  - If cellular machinery cannot distinguish human from viral RNA, cellular processes that aim to stop viral infections (e.g. siRNA) will fail
- N protein inhibits NFκB signaling and PKR function
  - NFκB is a master regulator of immune function
  - PKR inhibits the synthesis of viral RNA

GLYCAN SHIELD







ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

- Type of respiratory failure characterized by rapid onset of widespread inflammation of the lungs
- Primary treatment is mechanical ventilation
- Seen in severe pulmonary (pneumonia) or systemic infection (sepsis)
- Prognosis is poor with mortality around 40% and poor quality of life among survivors
- Pneumonia is present in 60% of ARDS patients

MECHANISM OF ARDS DEVELOPMENT IN COV INFECTION POORLY UNDERSTOOD

01

Rapid virus replication.

Both MERS and SARS replicate very rapidly early on after infection, which causes the epithelial cells to respond by producing massive quantities of proinflammatory cytokines and chemokines. This then leads to massive infiltration of inflammatory cells into the lungs.

02

hCoV infection of airway and/or alveolar epithelial cells.

Milder disease is seen among animals infected by viruses that only infect airway epithelial cells rather than both airway (type 1 pneumocytes) and alveolar epithelial cells (type 2 pneumocytes).

03

Delayed IFN responses.

Coronaviruses inhibit the production and activity of interferon (IFN). The delayed signaling further orchestrates the inflammatory monocyte-macrophage response.

04

Monocyte-macrophages and neutrophil accumulation: These cells are the predominant source of cytokines and chemokines associated with hCoV lethal disease observed both in humans and animal models.

Channappanavar R, Perlman S. Pathogenic human coronavirus infections: causes and consequences of cytokine storm and immunopathology. *Semin Immunopathol*. 2017;39(5):529–539. doi:10.1007/s00281-017-0629-x

VIROLOGIC PROFILE OF COVID-19

- Pathogenicity – capability of causing disease
  - 14% of those infected have severe disease
  - 81% have mild symptoms
  - 5% have no symptoms
- Virulence – severity of disease
  - 2.3% case fatality rate
- Infectivity – likelihood of transmission
  - R_0 of 1.4 - 2.5

$$R_0 = c \times p \times d$$

c = number of susceptible contacts per day
p = per-contact probability of infection
d = duration of infectiousness

S2: ORIGINS

- Origin of SARS-2
- How do we know this?
- Why it is definitely not man-made

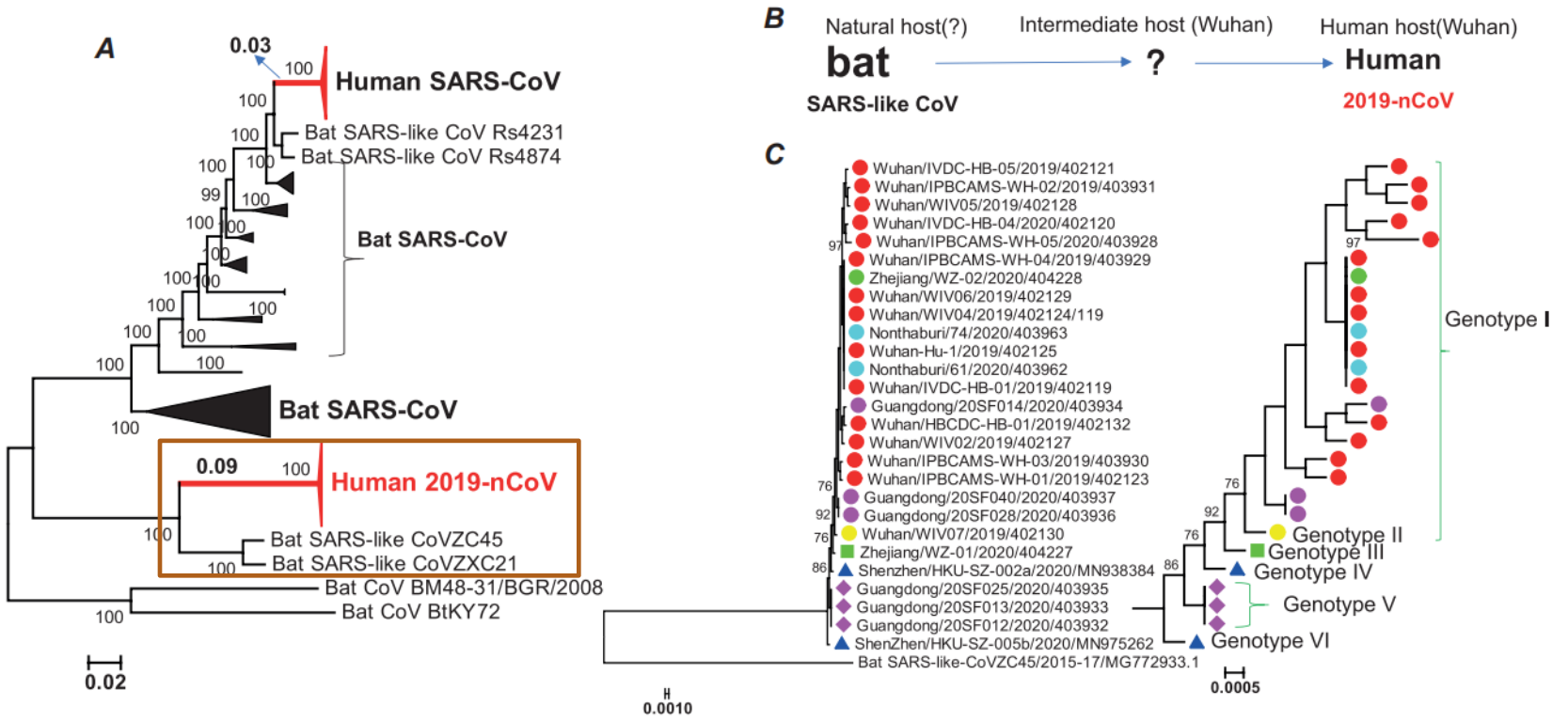
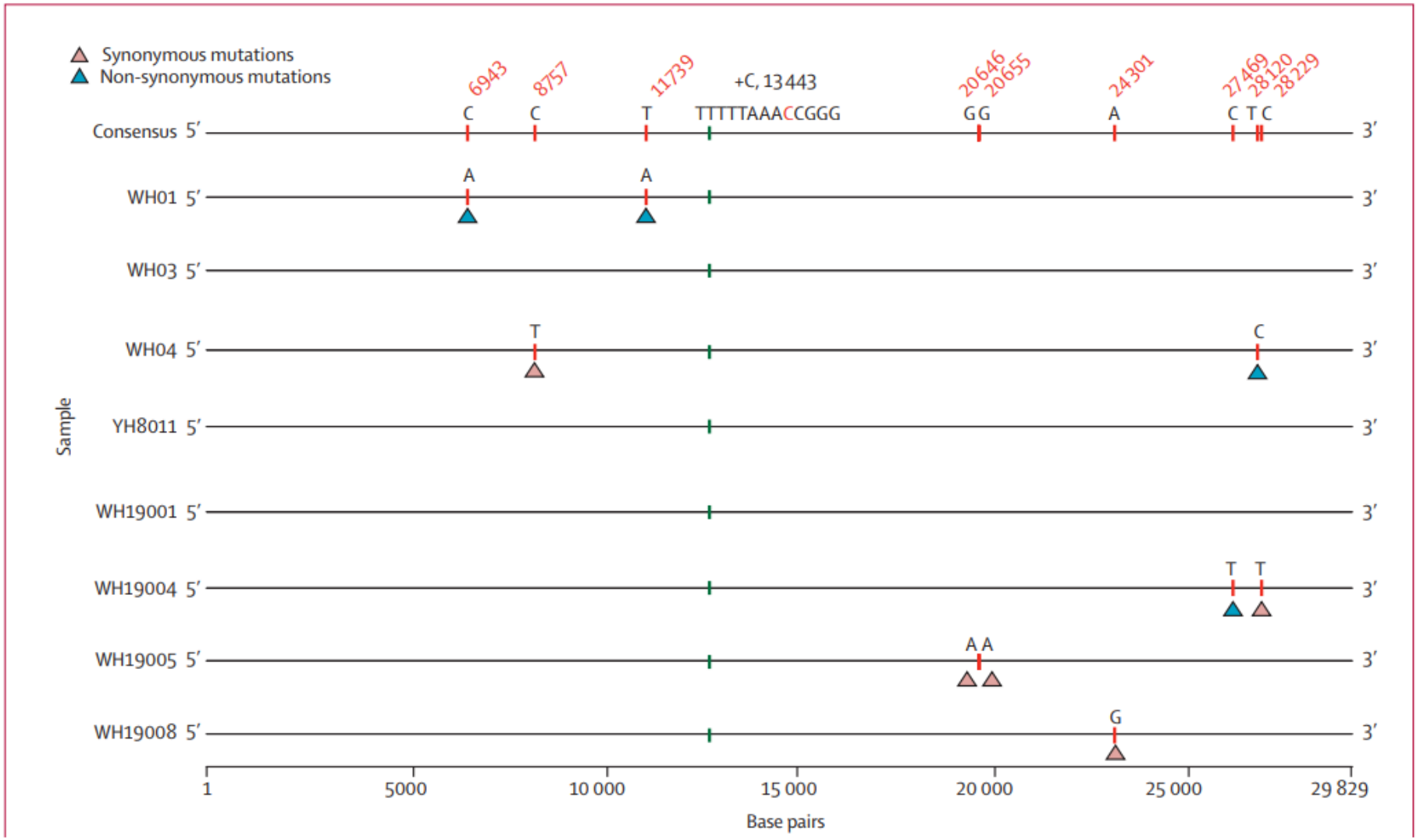
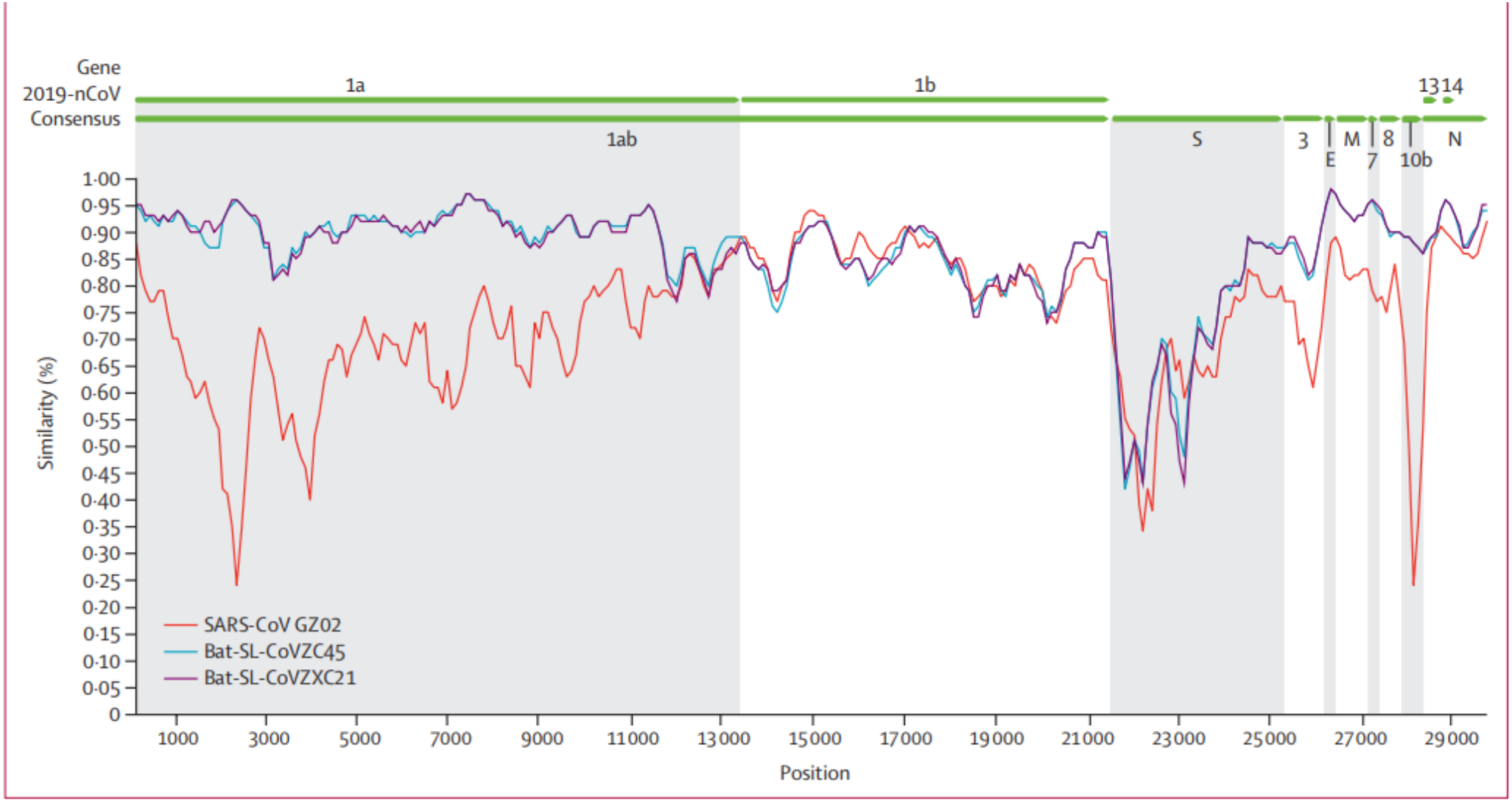


Figure 1. The phylogenomic trees of the coronavirus and the genotypes of 2019-nCoV strains. *A*, The maximum likelihood (ML) phylogenetic trees of the coronavirus with the approximate ML method by FastTree. This phylogenetic tree is a rooted tree with CoV BM48–31/BGR/2008 and BtKY72 as an outgroup. Coronaviruses from different sources are represented on the right side of the tree. The 2019-nCoV is labeled in red. The detailed sequence information used in this figure is shown in Supplementary Figure 1. *B*, A simple model of the 2019-nCoV origin inferred from the phylogenomic tree. *C*, The ML phylogenetic tree of 2019-nCoV with bat-SL-CoVZC45 as the root, with the approximate ML method by FastTree. The 27 isolates of 2019-nCoV can be divided into 6 genotypes. The 6 genotypes of 2019-nCoV strains are represented with different symbol shapes. Abbreviations: 2019-nCoV, 2019 novel coronavirus; SARS, severe acute respiratory syndrome.

Zhang L, Shen FM, Chen F, Lin Z. Origin and evolution of the 2019 novel coronavirus [published online ahead of print, 2020 Feb 3]. *Clin Infect Dis*. 2020;ciaa112. doi:10.1093/cid/ciaa112



Lu R, Zhao X, Li J, et al. Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. *Lancet*. 2020;395(10224):565–574. doi:10.1016/S0140-6736(20)30251-8



Lu R, Zhao X, Li J, et al. Genomic characterisation and epidemiology of 2019 novel coronavirus: implications for virus origins and receptor binding. *Lancet*. 2020;395(10224):565–574. doi:10.1016/S0140-6736(20)30251-8

Table 3. Amino acid identity between the 2019 novel coronavirus and bat SARS-like coronavirus or human SARS-CoV.

Amino acid identity (%)	2019-nCoV vs. bat-SL-CoVZXC21	2019-nCoV vs. SARS-CoV
NSP1	96	84
NSP2	96	68
NSP3	93	76
NSP4	96	80
NSP5	99	96
NSP6	98	88
NSP7	99	99
NSP8	96	97
NSP9	96	97
NSP10	98	97
NSP11	85	85
NSP12	96	96
NSP13	99	100
NSP14	95	95
NSP15	88	89
NSP16	98	93
Spike	80	76
Orf3a	92	72
Orf3b	32	32
Envelope	100	95
Membrane	99	91
Orf6	94	69
Orf7a	89	85
Orf7b	93	81
Orf8/Orf8b	94	40
Nucleoprotein	94	94
Orf9b	73	73

Chan JF, Kok KH, Zhu Z, et al. Genomic characterization of the 2019 novel human-pathogenic coronavirus isolated from a patient with atypical pneumonia after visiting Wuhan. *Emerg Microbes Infect*. 2020;9(1):221–236. doi:10.1080/22221751.2020.1719902

IS THIS THE RESULT OF BIOWARFARE RESEARCH?

There are rumors/conspiracy theories regarding the origins of the virus being somehow related to the only biosafety level 4 facility in China, coincidentally located in Wuhan. They span from being genetically modified as a bioweapon to more innocuous research and accidentally being let out of the lab. How would you characterize these theories—obviously wrong, very likely wrong but one can't be sure, or a real possibility?

I've also seen people claiming that this was a manufactured virus, is there any truth to this or is this just the US spreading anti-China sentiments?

- No.
- If it were, it's a terrible bioweapon. There are much better choices
- We know the sequence homology with related species
- Occam's razor says that the easiest explanation is the correct one

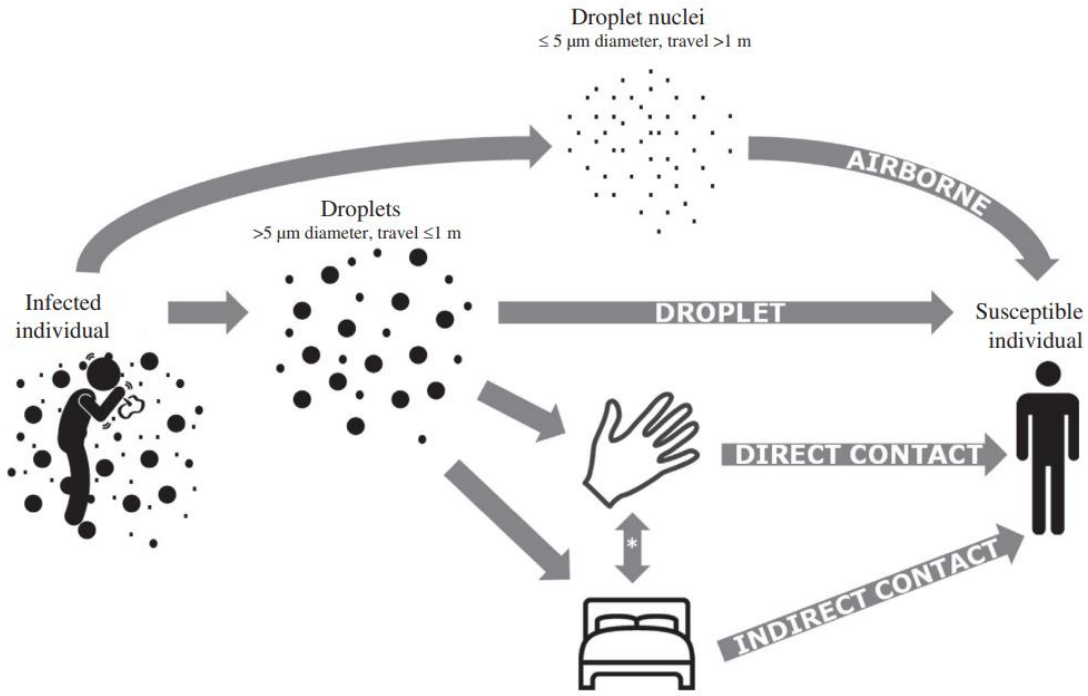
PUTTING IT TOGETHER

1) COVID-19 is a SARS-like virus and can be classified as SARS-2
2) It is closer phylogenetically to Bat SARS-CoV than is Human SARS
3) It's similarity with two Bat coronaviruses implies a direct common ancestor
4) Since the early cases were nearly identical in genetic sequence (99.98% identical), virus recently emerged in humans
    1) Late-October 2019 – Mid-November 2019
5) Anyone claiming that this is a bioweapon has clearly never studied virology, worked in a research lab, or read a scientific paper. You are encouraged to publicly shun people spreading idiotic conspiracy theories since they are HARMING the public health response. This is yelling "fire" in a crowded movie theater.

S3: TRANSMISSION

HOW IS COVID-19 SPREAD?

TRANSMISSION OF CORONAVIRUSES



* Transmission routes involving a combination of hand & surface = indirect contact.

Figure 1. Transmission routes: droplet, airborne, direct contact, and indirect contact. (Indirect contact: routes involving a combination of hand and surface.) Definitions of 'droplet' and 'droplet nuclei' are from Atkinson *et al.*[5]

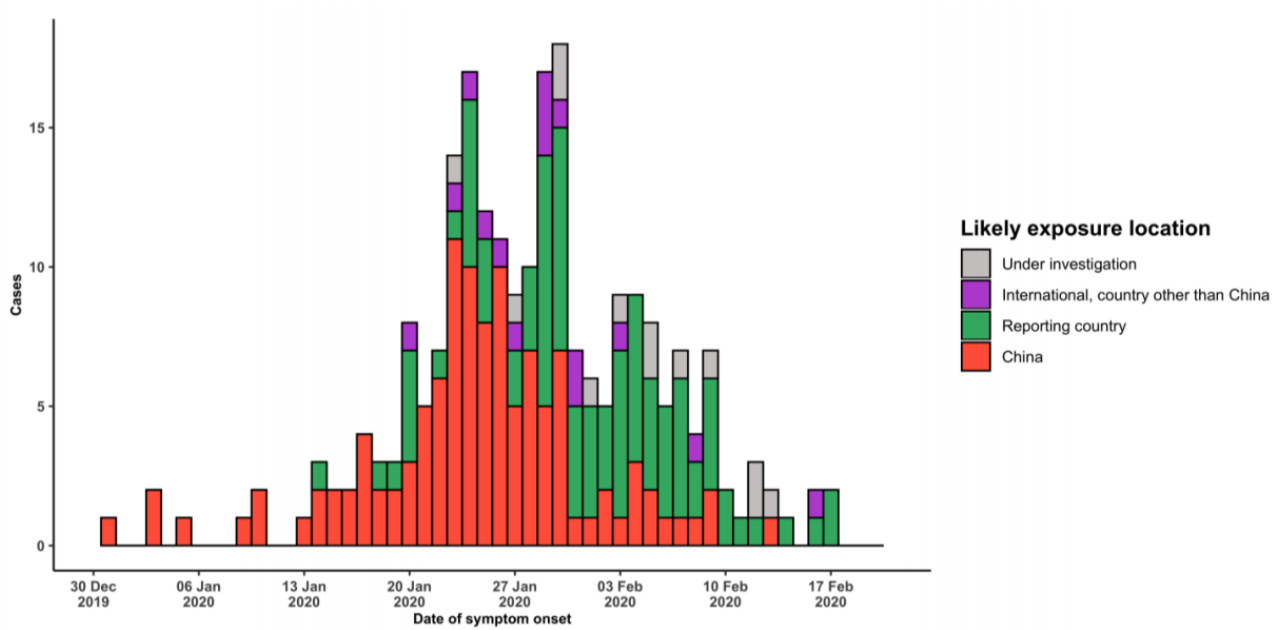
Otter JA, Donskey C, Yezli S, Douthwaite S, Goldenberg SD, Weber DJ. Transmission of SARS and MERS coronaviruses and influenza virus in healthcare settings: the possible role of dry surface contamination. *J Hosp Infect*. 2016;92(3):235–250. doi:10.1016/j.jhin.2015.08.027

TRANSMISSION REQUIREMENTS

Route	Size	Environment	CoV Survival	CoV Likelihood
Indirect Contact	Any	Temperature and RH-dependent	7.5% of frequently-touched items in Jeddah airport 3.5% in Toronto SARS Units 38.1% in SARS units in Bangkok and Taipei	Very likely
Direct Contact	Any	Independent	WASH YOUR HANDS	Very likely
Droplets	>10 μm	Independent	No more than 1 meter in all directions	Likely
Aerosol	<10 μm	Temperature and RH-dependent	7% reduction over 10 minutes	Less likely

Otter JA, Donskey C, Yezli S, Douthwaite S, Goldenberg SD, Weber DJ. Transmission of SARS and MERS coronaviruses and influenza virus in healthcare settings: the possible role of dry surface contamination. *J Hosp Infect*. 2016;92(3):235–250. doi:10.1016/j.jhin.2015.08.027

Figure 2. Epidemic curve of COVID-19 cases (n=233) identified outside of China, by date of onset of symptoms and likely exposure location, 20 February 2020

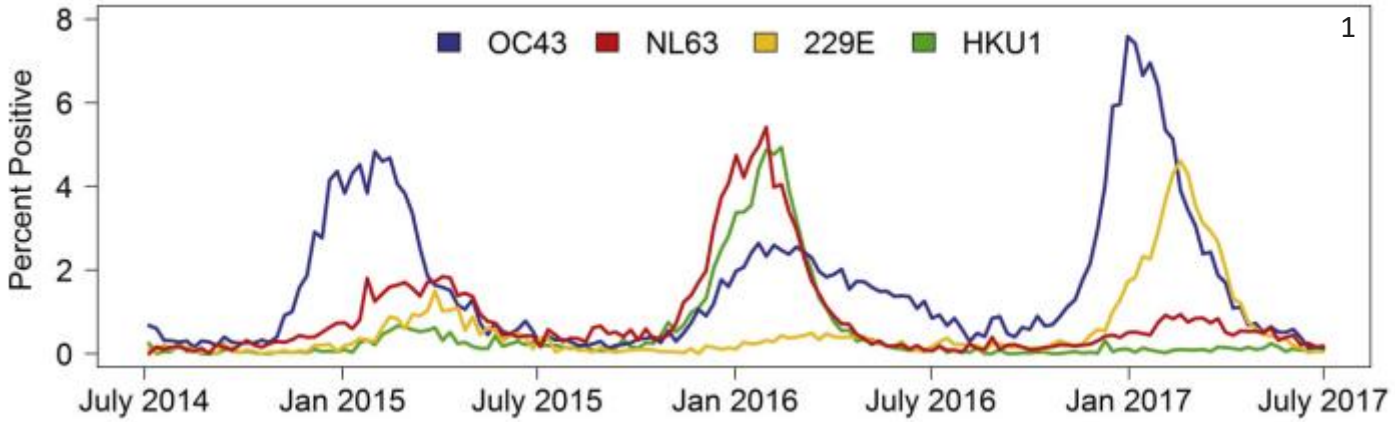


Note for figure 2: Of the 1073 cases reported outside China, 30 were detected while apparently asymptomatic. For the remaining 1043 cases, information on date of onset is available only for the 233 cases presented in the epidemiologic curve.

S4: EPIDEMIOLOGY

IT ISN'T THE STUDY OF SKIN

Name	Type	Origin	Prevalence	CFR	Incubation Period	Time to Death
NL63	Alpha	Bats	1.0%[1]	--	2-4 Days[3]	--
229E	Alpha	Bats	0.8%[1]	--	2-5 Days[3]	--
OC43	Beta	Rodents	2.2%[1]	--	2-5 Days[3]	--
HKU1	Beta	Rodents	0.6%[1]	--	2-4 Days[3]	--
SARS	Beta	Bats	0.0%[2]	9%[3]	2-11 Days[3]	23 Days[3]
MERS	Beta	Bats	0.15%[6]	36%[3]	2-13 Days[3]	14 Days[3]
COVID-19	Beta	Bats	---	2.3%[4]	6-10 Days	14 Days[5]



[1]Killerby ME, Biggs HM, Haynes A, et al. Human coronavirus circulation in the United States 2014-2017. *J Clin Virol*. 2018
[2]Centers for Disease Control and Prevention. "SARS (10 Years After)" https://www.cdc.gov/dotw/sars/index.html Last reviewed 3-3-2016. Accessed 2-26-2020
[3]Su S, Wong G, Shi W, et al. Epidemiology, Genetic Recombination, and Pathogenesis of Coronaviruses. Trends Microbiol. 2016
[4]Wu Z, McGoogan JM. Characteristics of and Important Lessons From the Coronavirus Disease 2019 (COVID-19) Outbreak in China: Summary of a Report of 72 314 Cases From the Chinese Center for Disease Control and Prevention. JAMA. Published online February 24, 2020
[5]Wang W, Tang J, Wei F. Updated understanding of the outbreak of 2019 novel coronavirus (2019-nCoV) in Wuhan, China. J Med Virol. 2020
[6]Al Kahlout RA, Nasrallah GK, Farag EA, et al. Comparative Serological Study for the Prevalence of Anti-MERS Coronavirus Antibodies in High- and Low-Risk Groups in Qatar. J Immunol Res. 2019;2019:1386740. Published 2019 Feb 18.

DEMOGRAPHICS AND RISK FACTORS

COVID-19

Age Group	CFR
80+ years old	14.8%
70-79 years old	8.0%
60-69 years old	3.6%
50-59 years old	1.3%
40-49 years old	0.4%
30-39 years old	0.2%
20-29 years old	0.2%
10-19 years old	0.2%
0-9 years old	None

Sex	CFR
Male	2.8%
Female	1.7%

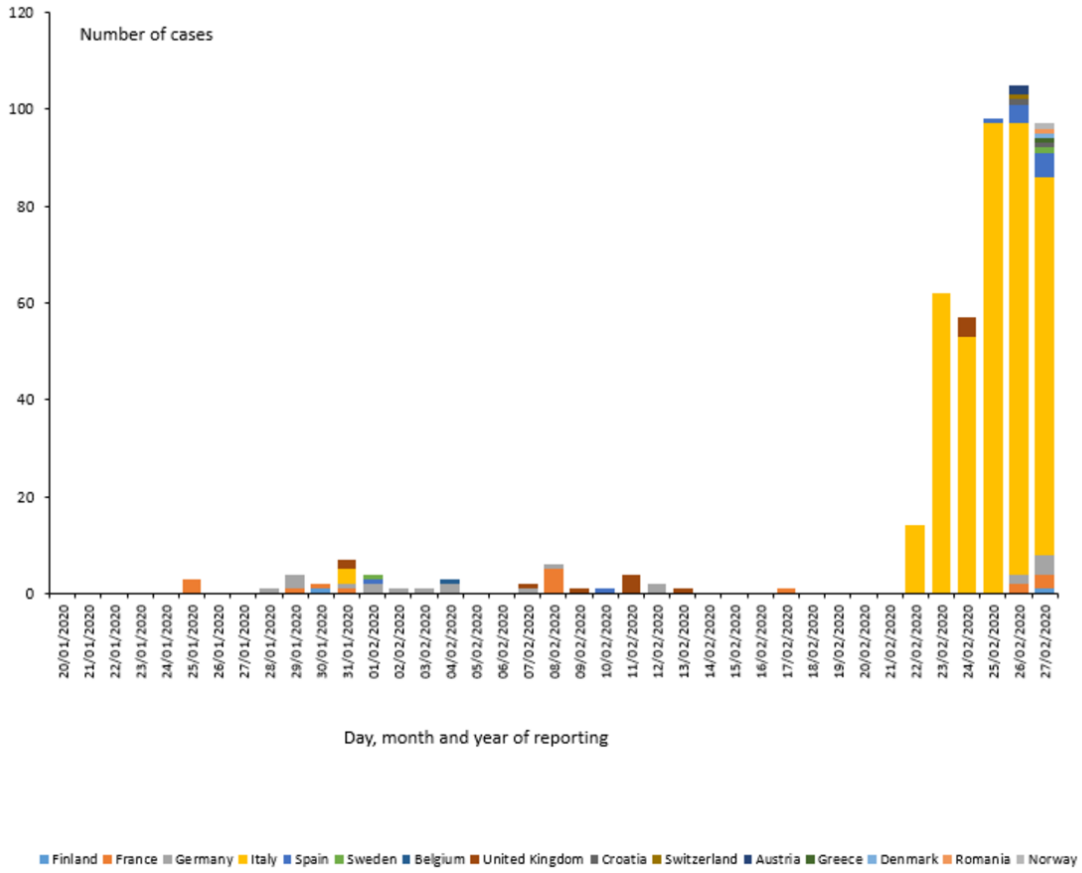
Pre-existing condition	CFR
Cardiovascular disease	10.5%
Diabetes	7.3%
Chronic respiratory disease	6.3%
Hypertension	6.0%
Cancer	5.6%
None	0.9%

ARDS

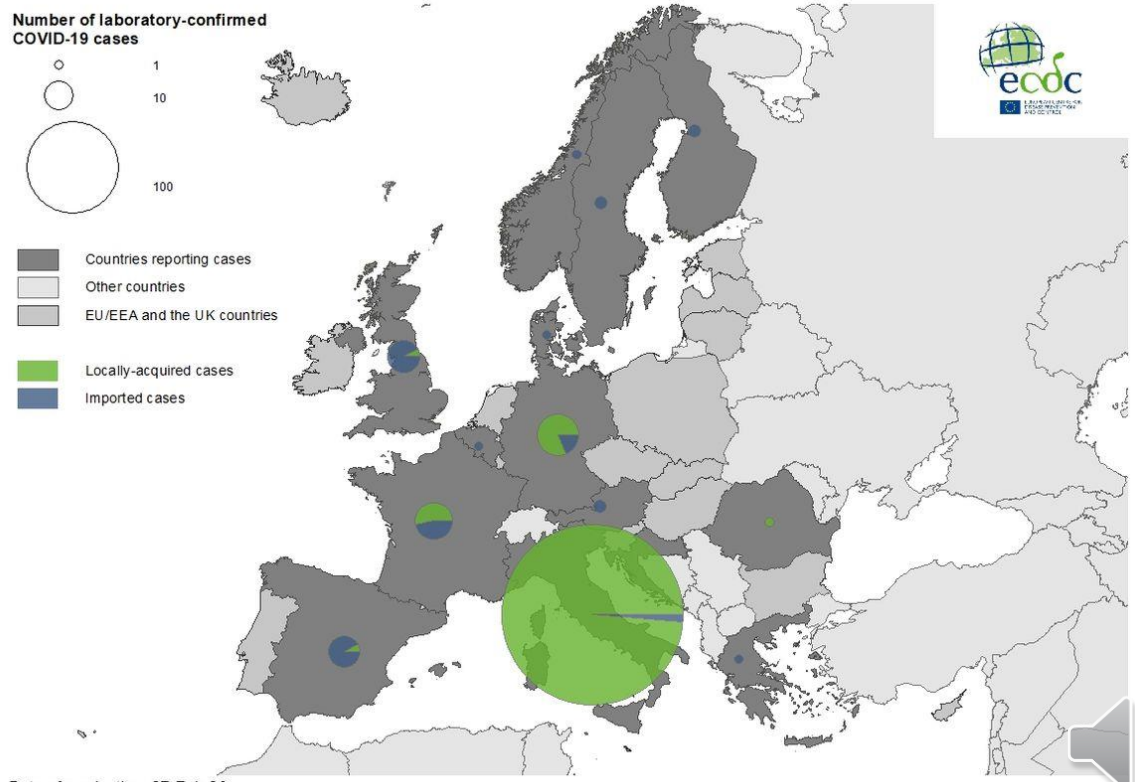
- Pre-disposing conditions
  - Recent surgery
  - Obesity
  - COPD
- Lifestyle factors
  - Chronic alcohol abuse
  - Active or passive cigarette smoking
  - Ozone exposure
  - Vitamin D deficiency is ubiquitous

BASIC EPIDEMIOLOGIC GRAPHS AND MAPS COVID-19 EUROPE

Epidemic Curve Europe (2-27-2020)

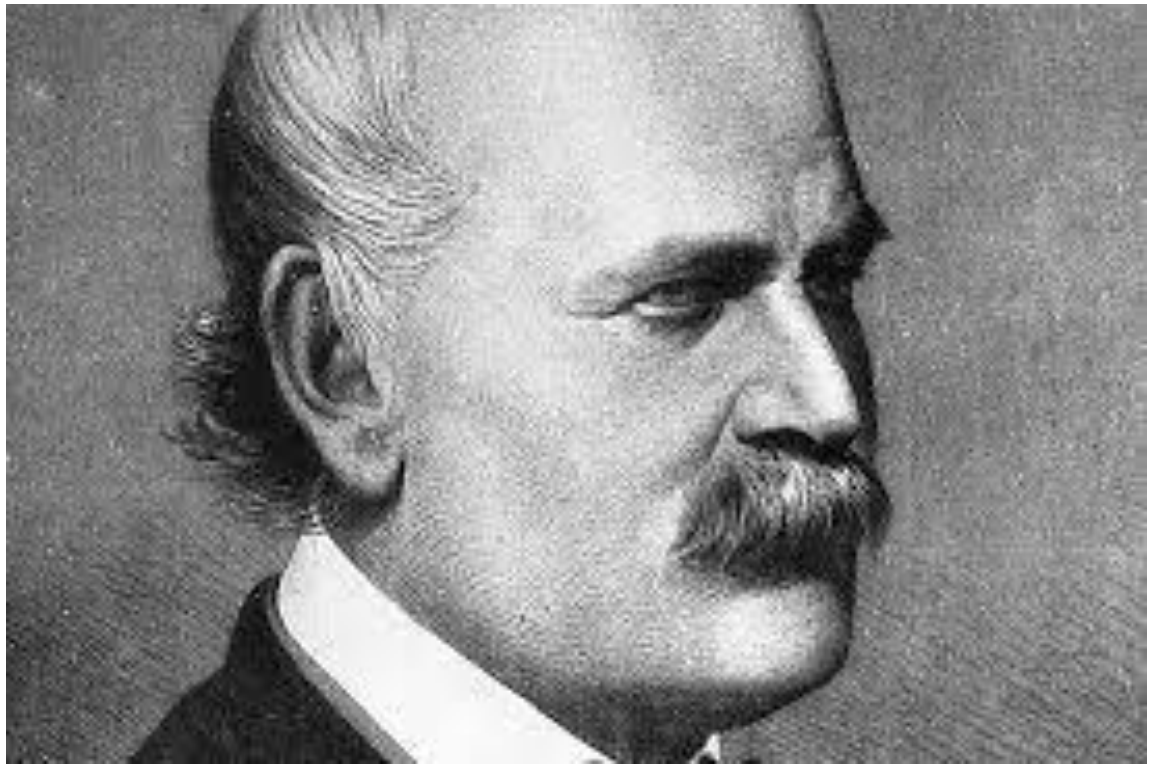


Geographic Distribution of COVID-19 (2-27-2020)



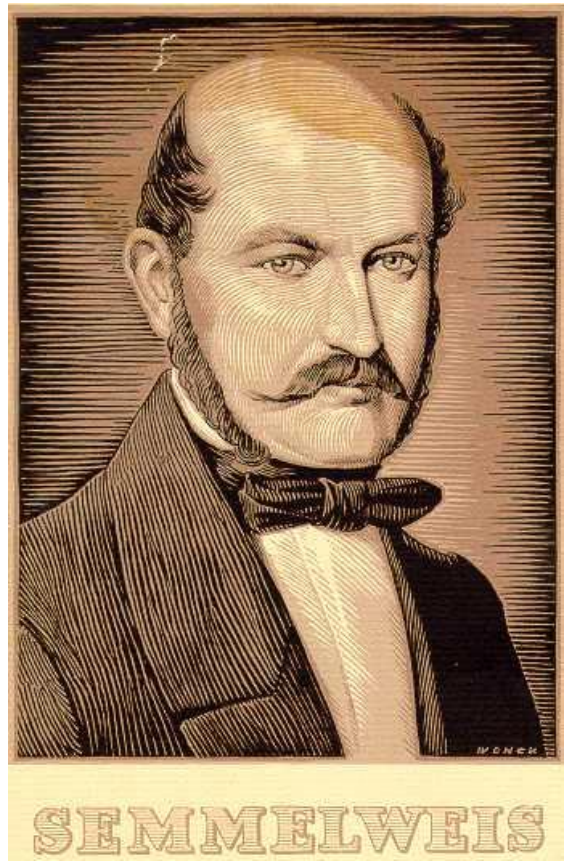
S5. PREVENTION

THE SAD DESTINY OF IGNAZ SEMMELWEIS

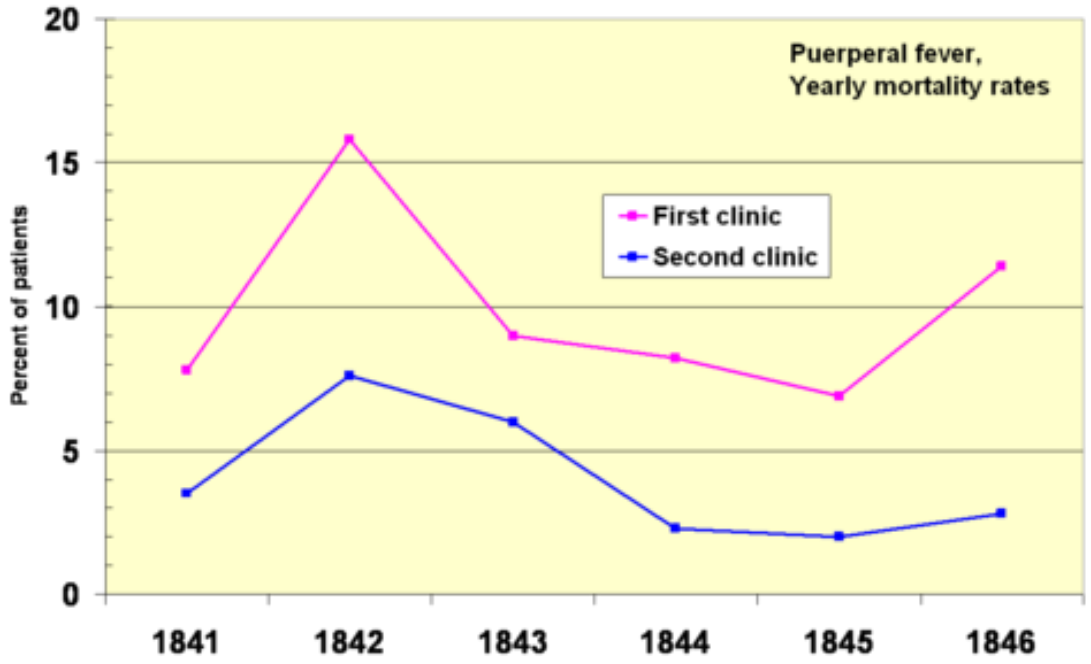


THE SAVIOR OF MOTHERS

- Semmelweis was an Austrian physician working in Vienna General Hospital
- Appointed as the equivalent of Chief Resident of Obstetrics in 1847
- Two clinics available to women, which held admissions on alternating days
  - Clinic 1 had a maternal mortality rate of 10%
  - Clinic 2 had a maternal mortality rate of 4%
- Differential mortality was known among expectant mothers
  - Some preferred giving birth in the street rather than birth in clinic 1
- "To me, it appeared logical that patients who experienced street births would become ill at least as frequently as those who delivered in the clinic. [...] What protected those who delivered outside the clinic from these destructive unknown endemic influences?"



1818 (b) – 1865 (d)
Vienna General Hospital

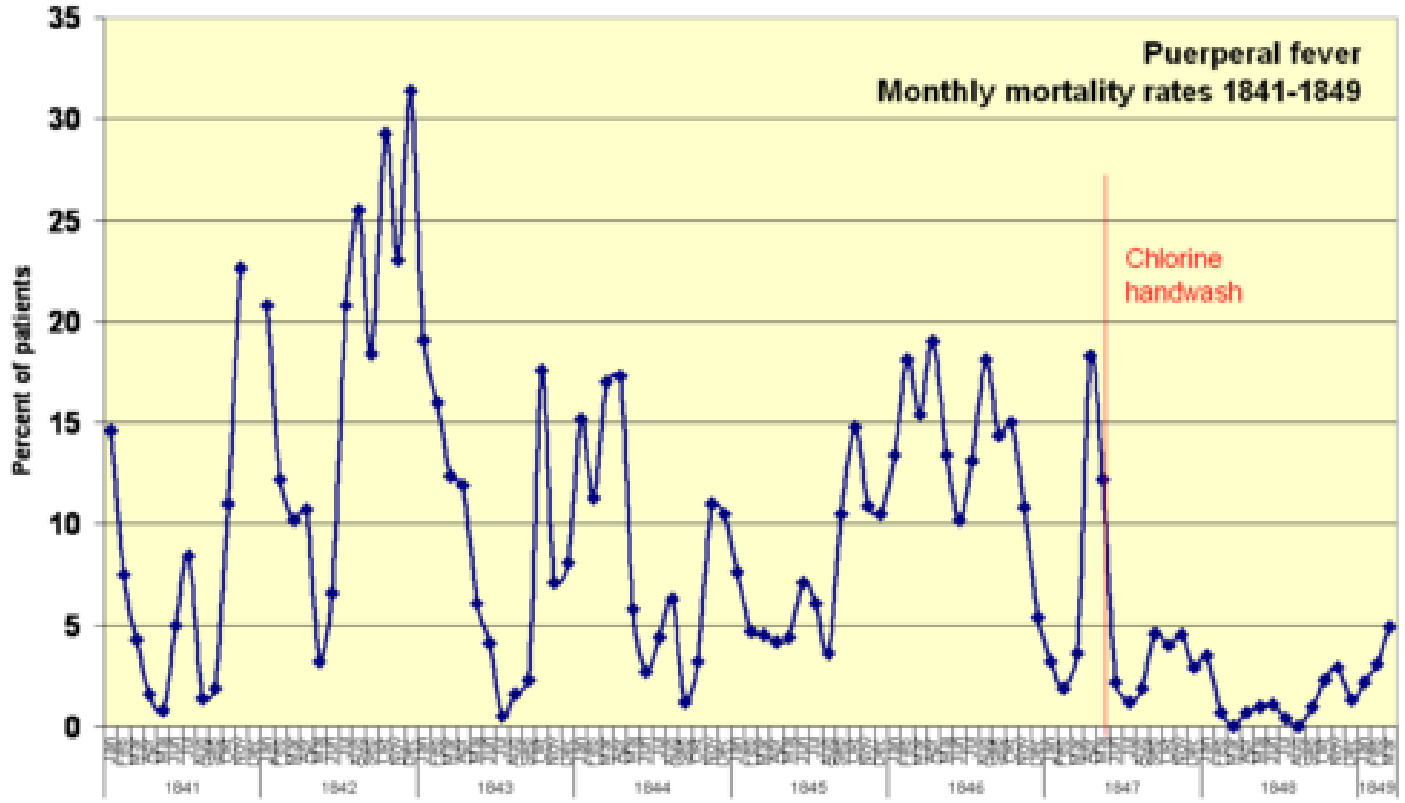


A TALE OF TWO CLINICS

- Both clinics had similar protocols and only really differed in staffing
- Clinic 1 – staffed by medical students
- Clinic 2 – staffed by midwives
- Theories:
  - Overcrowding – Clinic 2 was more crowded
  - Climate – climates were similar
  - Cadaveric contamination

ORIGIN OF HAND HYGIENE

- Implemented a protocol for medical students to wash hands before delivering
  - Solution of chlorinated lime (calcium hypochlorate)
  - Instituted mid-May 1847
  - Mortality rate dropped from 18.4% in April to 2.2% in June



RECEPTION

- Germ theory of disease hadn't been accepted in Vienna yet
  - Galen's miasma theory was the paradigm
  - The work of Pasteur (1870) and Koch (1880)
- Semmelweis' hypothesis was largely ignored and he was ridiculed by the medical community
- He was dismissed from the hospital and forced to move to Budapest

LEGACY OF THE SAVIOR OF MOTHERS

CDC Method

1) Wet hands
2) Apply good amount of soap
3) Lather up
4) Front, back, around and underneath nails
5) ~20 seconds (“Happy Birthday Twice”)

WHO Method

1) Wet hands with water
2) Apply enough soap to cover all hand surfaces
3) Rub hands palm to palm
4) Right palm over left with interlaced fingers and vice-versa
5) Palm to palm with fingers interlaced
6) Backs of fingers to opposing palms with fingers interlocked
7) Rotational rubbing of left thumb clasped in right palm and vice versa
8) Rotational rubbing backwards and forwards with clasped fingers of right hand in left palm and vice versa

ADDITIONAL PREVENTION PRACTICES

Hand sanitizer (at least 60% alcohol)

Cough/Sneeze into elbow

Wave instead of hugging/hand shaking

Universal precaution – everyone and everything is infected

Clean your commonly used items regularly (alcohol swabs)

Avoid touching your face

Stay home if sick


Softsoap
crisp cucumber & melon
HAND SOAP
SAVON POUR LES MAINS
Alcohol Prep Pads
120 PADS - 1.1 IN x 1.3 IN (2.8 cm x 3.3 cm)
CVS Health
STERILE
Alcohol Prep Pads
70% ISOPROPYL ALCOHOL
Safe & easy to use
120 PADS - 1.1 IN x 1.3 IN (2.8 cm x 3.3 cm)
CVS Health
STERILE
Alcohol Prep Pads
70% ISOPROPYL ALCOHOL
Safe & easy to use
120 PADS - 1.1 IN x 1.3 IN (2.8 cm x 3.3 cm)
CVS Health
Original Scent
Moisturizing
Hand Sanitizer
60 FL OZ (1.77
CVS Health
Prebiotic
Hand Sanitizer
CVS Health
Isopropyl
Rubbing Alcohol
FIRST AID ANTISEPTIC
70%
32 FL OZ (1 QT) 946 mL

YOU DIDN'T MENTION FACEMASKS, MIKE. WHAT KIND OF EXPERT ARE YOU?

- N95 facemasks are great for people often in close contact with infected individuals
  - Doctors
  - Nurses
  - EMTs
  - Poll workers
  - Those who are sick (maybe?)
- Probably won't protect you and is more likely to be a source of infection.

S6. EPIDEMIOLOGIC FORECAST

WHAT TO EXPECT WHEN YOU'RE EXPECTING A PANDEMIC

TERMS TO UNDERSTAND

$$R_0 = c \times p \times d$$

c = number of susceptible contacts per day
p = per-contact probability of infection
d = duration of infectiousness

- Sustained transmission or Community spread – infection is spreading beyond clusters of patients
  - Epidemic – ($R_0 > 1$) the average infected person will infect more than one person
  - Endemic – ($R_0 = 1$) the average infected person will infect one other person
  - Elimination – ($R_0 < 1$) the average infected person will infect less than one other person
- Mortality rate – $\frac{number\ who\ have\ died}{total\ population}$
- Case fatality rate – $\frac{number\ who\ have\ died}{number\ of\ cases}$
- Pandemic – community-level human-to-human transmission in at least 2 countries in different WHO areas

PANDEMIC STAGES

Phase 1: viruses circulating among animals have NOT caused human infection

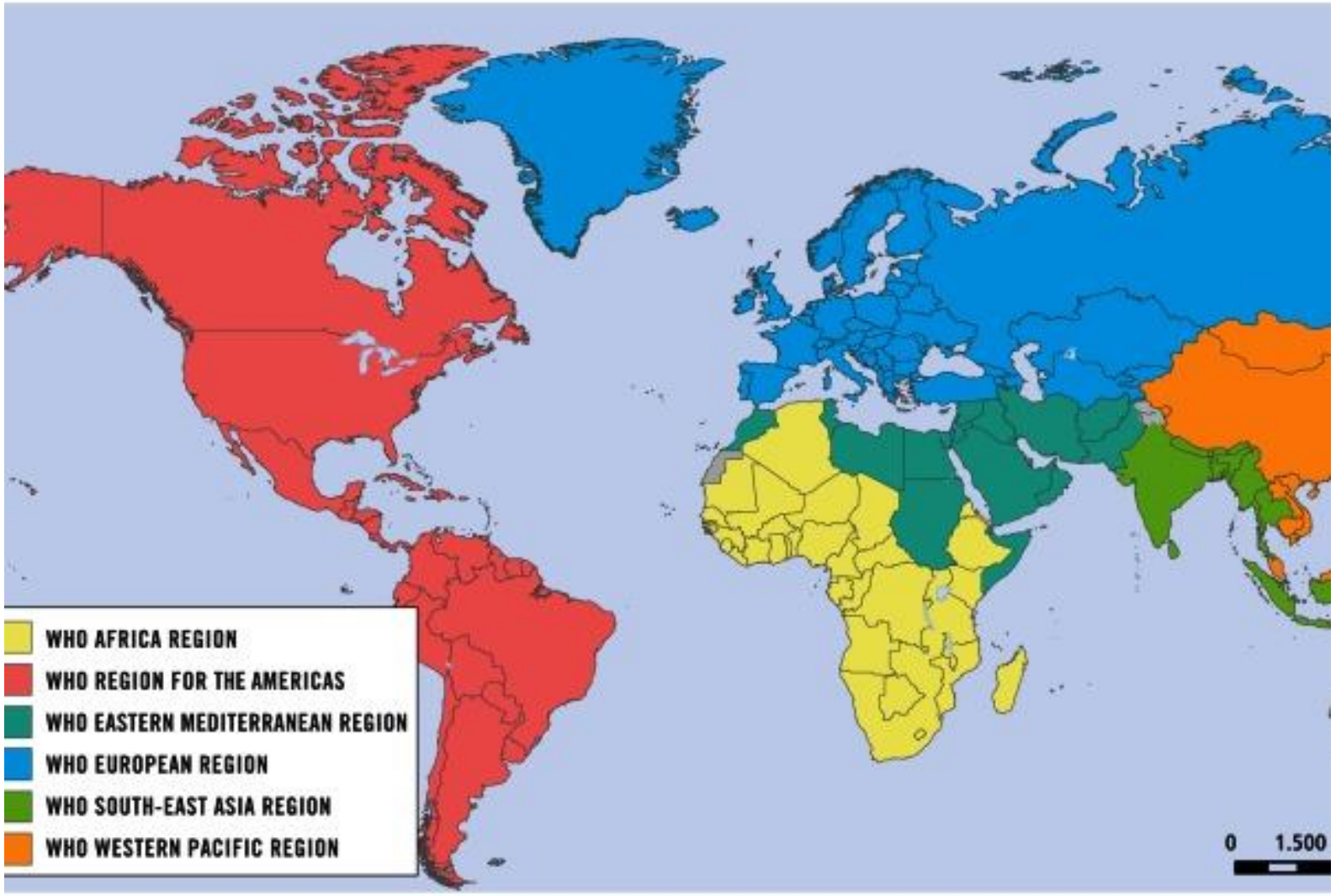
Phase 2: an infection has caused an infection in humans

Phase 3: small clusters of cases in humans, but has not resulted in human-to-human transmission sufficient to sustain community-level outbreaks

Phase 4: verified human-to-human transmission, ability to cause sustained community-level transmission

Phase 5: human-to-human spread in at least two countries within one WHO region

Phase 6: community-level outbreaks in at least one other country in a different WHO region. This indicates a pandemic is occurring.



Pandemic Influenza Preparedness and Response: A WHO Guidance Document. Geneva: World Health Organization; 2009. 4, THE WHO PANDEMIC PHASES. Available from: https://www.ncbi.nlm.nih.gov/books/NBK143061/

CLINICAL COURSE OF INFECTION

- Most cases are mild (81%)
- Symptoms develop 4-10 days after exposure
- Length of illness
  - Can't find anything in the literature
  - Median hospital stay is 12 days
- Typical symptoms:
  - Fever (98%)
  - Cough (67.8%)
  - Sputum production (33.7%)
  - Fatigue (38.1%)

Characteristics of COVID-19 infected individuals in hospitals in China[1]

	Non-severe Cases (n=926)	Severe Cases (n=173)
Median Age	45.0	52.0
Current Smoker	11.8%	16.9%
Incubation period (IQR)	4 (2-7)	4 (2-7)
Days to Pneumonia Dx	3 (1-6)	5 (2-7)

Infections in Infants

"Based on the sources of data used in this study, 9 infants were infected with COVID-19 and were hospitalized in China between December 2019 and February 6, 2020. Given the number of infections reported, the number of infected infants identified was small. This may be due to a lower risk of exposure or incomplete identification due to mild or asymptomatic disease, rather than resistance to infection."

[1]Guan W-j, Ni Z-y, Hu Y, et al. Clinical Characteristics of Coronavirus Disease 2019 in China. New England Journal of Medicine. 2020.
[2]Wei M, Yuan J, Liu Y, Fu T, Yu X, Zhang ZJ. Novel Coronavirus Infection in Hospitalized Infants Under 1 Year of Age in China [published online ahead of print, 2020 Feb 14]. JAMA. 2020;10.1001/jama.2020.2131. doi:10.1001/jama.2020.2131

WILL THERE BE AN EPIDEMIC IN THE UNITED STATES?

IT IS INEVITABLE GIVEN THE GLOBAL SPREAD OF THE INFECTION.

WHAT WILL THE UNITED STATES' RESPONSE BE?

I DON'T KNOW.

S7. ADDITIONAL QUESTIONS

INTERESTING QUESTIONS I'VE RECEIVED FROM FRIENDS

The Media

- Is the media obsession based on real danger, or is this just a way to generate website hits?
- Is this virus scary because it's new and spreading quickly or does it have other properties that make it particularly dangerous or that's all hype?
- Russian media says many other types of flu impact/kill a lot more people than Coronavirus every year. Could that be true?
- I definitely would like to know more about the infographics used to downplay corona with comparisons to common flu. Is this just cancel culture and a way of non-science minded folks to sound superior? Or should I indeed worry more about the flu than corona? To me, it seems that a novel virus that can still be contained and eradicated should matter a bit more than something that's so mature and worldwide and has a vaccine already popularized?

Geopolitical

- Is the downswing in the markets due to Chinese supply chains slowing going to take us into a recession?
- The market tanked today, apparently because of corona fears. Does that make sense, or is it just an excuse to hide behind a long-expected market correction?
- Other parts of the world that are on Corona-watch are currently experiencing other epidemics such as Dengue, Ebola to name a couple. Will countries that are currently dealing with other epidemic issues be better positioned or worse-off if there is a Coronavirus outbreak in their country? Is there any transferability of knowledge or resources from fighting off one epidemic as the new one seems to settle in?

INTERESTING QUESTIONS FROM MRS. KUEGLER'S FRESHMEN HISTORY CLASS AT WATERTOWN HIGH SCHOOL IN CONNECTICUT

Jack: Why does this disease affect some patients worse than others, are certain people more prone to getting affected based off of genetics or other diseases?

Emily: How can the virus originate from animals but CDC believes pets can't transmit the disease?

Greg: Is it possible that the coronavirus can evolve to become more dangerous?

Amarta: If the disease leads to a respiratory sickness that already exists, why isn't there any treatment?

Karina: Was a factor in the spreading of coronavirus, overcrowded populations?

Jasmine: How is the education of young people affected by the coronavirus?

Eva: How is traveling being impacted by the coronavirus?

Ian: Are the death rates for the virus (around 4%) good or bad compared to other outbreaks of viruses?

Sam: Could the virus affect the Tokyo Olympics, if so, can they be moved to a different country or will it have to be delayed?

Bertin: Are there ways to prevent/cure the coronavirus?

Erin: Is it likely for a cure to be found? How close are they to finding this solution?

THANK YOU FOR WATCHING THIS AND CONTRIBUTING YOUR QUESTIONS. IF YOU HAVE MORE QUESTIONS, I'LL DO MY BEST TO RESPOND. AND DON'T FORGET TO WASH YOUR HANDS