Current Epidemiological Models: Scientific Basis and Evaluation

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Issues, Approaches, and Consequences of the COVID-19 Crisis

https://www.cits.ucsb.edu/spring2020

CITS Center for Information Technology and Society

UC SANTA BARBARA Data Science Initiative

NOVIM TRUST SCIENCE

spatial@ucsb CENTER FOR SPATIAL STUDIES

CRML Center for Responsible Machine Learning

Cottage Health

Acknowledgments

Terrible impact of this pandemic

The complex, dangerous, critical work by healthcare professionals all over the world on the front line of this battle

All essential frontline workers, including first responders, grocery-store workers, and transit workers

We owe them all a great deal of gratitude

Relevant links:

- "Life and Death in the 'Hot Zone"' (article and video) by Nicholas Kristof, New York Times, 4/11/2020
  https://nyti.ms/3a1GATB
- Webinar by Dr Carolina Arias Gonzales (UCSB MCDB) and Dr Lynn N. Fitzgibbons (Cottage Health), 4/14/2020
  https://www.cits.ucsb.edu/spring2020

With COVID-19,
modeling takes on life and death importance

"But on March 16th, the Imperial College group published a dramatically revised model that concluded [...] that even a reduced peak would fill twice as many intensive care beds as estimated previously." Science, March 27th

PANDEMIC Qs

Fauci's straight talk

To many watching the White House press briefings on the coronavirus pandemic, veteran public health expert Anthony Fauci has become the voice of science and reason on how the country should respond. He made national news this week for his careful but candid assessment to *Science*'s Jon Cohen of the challenges of working for President Donald Trump during the crisis. "When you're dealing with the White House, sometimes you have to say things one, two, three, four times, and then it happens. So, I'm going to keep pushing," says Fauci, longtime director of the National Institute of Allergy and Infectious Diseases. His full interview is at https://scim.ag/QAFauci.

Q: The first question everyone has is how are you?
A: Well, I'm sort of exhausted. But other than that, I'm good. I mean, I'm not, to my knowledge, coronavirus infected. To my knowledge, I haven't been fired [laughs].

Q: How are you managing to not get fired?
A: To [Trump's] credit, even though we disagree on some things, he listens. He goes his own way. He has his own style. But on substantive issues, he does listen to what I say.

Q: You've been in press conferences where things are happening that you disagree with, is that fair to say?
A: Well, I don't disagree in the substance. It is expressed in a way that I would not express it, because it could lead to some misunderstanding about what the facts are about a given subject.

Dutch models of COVID-19 are designed to help prevent overloading of hospitals and the need to transfer patients.

CORONAVIRUS

With COVID-19, modeling takes on life and death importance

Epidemic simulations shape national responses

By **Martin Enserink** *and* **Kai Kupferschmidt**

Jacco Wallinga's computer simulations are about to face a high-stakes reality check. Wallinga is a mathematician and the chief epidemic modeler at the National Institute for Public Health and the Environment (RIVM), which is advising the Dutch government on what actions, such as closing schools and businesses, will help control the spread of the novel coronavirus in the country.

pended on their work. Entire cities and countries have been locked down based on hastily done forecasts that often haven't been peer reviewed. "It's a huge responsibility," says epidemiologist Caitlin Rivers of the Johns Hopkins University Center for Health Security, who co-authored a report about the future of outbreak modeling in the United States that her center released this week.

Just how influential those models are became apparent over the past 2 weeks in the United Kingdom. Based partly on modeling

Outline

1. **historical notes**
2. introduction to mathematical epidemiology
   1. the simplest SIR model
   2. stochastic SIR models
   3. direct statistical estimation
3. summary evaluation
4. conclusion on non-pharmaceutical interventions (NPIs)

Warnings: elementary intro, no new model

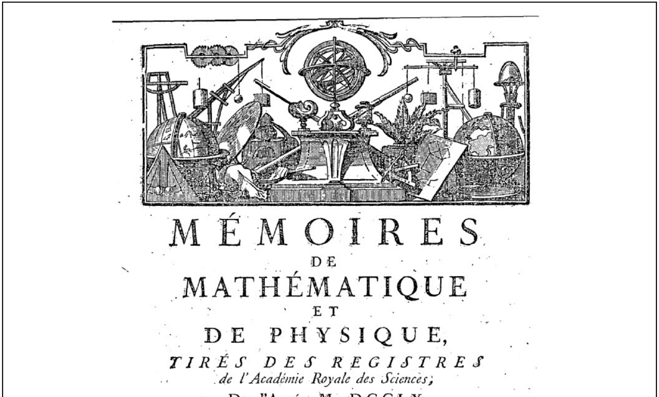
My qualifications:

- F. Bullo. *Lectures on Network Systems*. Kindle Direct Publishing, 1.3 edition, July 2019. URL: http://motion.me.ucsb.edu/book-lns
- W. Mei, S. Mohagheghi, S. Zampieri, and F. Bullo. On the dynamics of deterministic epidemic propagation over networks. *Annual Reviews in Control*, 44:116–128, 2017. doi:10.1016/j.arcontrol.2017.09.002

Daniel Bernoulli 1760: controversial smallpox variolation

- "the greatest killer in history"
- variolation, i.e., inoculation with a mild strain
- controversy: long-term benefit vs risk of immediate death

using empirical data, mathematical proof that inoculation could increase life expectancy at birth up to three years


MÉMOIRES
DE
MATHÉMATIQUE
ET
DE PHYSIQUE,
TIRÉS DES REGISTRES
de l'Académie Royale des Sciences;

W. Hamer 1906: nonlinear incidence

- **compartments**: S, I and R
- incidence = number of new cases per unit time
  depends on the **product of the densities of S and I**

THE LANCET, March 3, 1906.

The Milroy Lectures

ON

EPIDEMIC DISEASE IN ENGLAND—THE EVIDENCE OF VARIABILITY AND OF PERSISTENCY OF TYPE.

Delivered before the Royal College of Physicians of London,

By W. H. HAMER, M.A., M.D. Cantab., F.R.C.P. Lond.

LECTURE I.[1]

Delivered on March 1st.

Mr. President and Gentlemen,—Changes of type in epidemic diseases was the subject chosen by Dr. B. A Whitelegge for the Milroy lectures of 1893, to which the reader perforce returns again and again, as if increase of appetite had grown by what it fed on. The same topic has been variously approached and in recent years more particularly from the evolutionary standpoint. Already towards the close of the seventeenth century Sydenham had been accorded a Pisgah sight of the land to be explored, but prior to the Registrar-General and to Darwin no considerable advance into this new territory was possible. Even in the "fifties" there was much speculation which now seems strangely out of date. Murchison contended, on the one hand, for the *de novo* origin of typhoid fever and he notes, "No mention is made of specific disease in the Mosaic account of the Creation, when we are told that every living creature and herb of the field was created and it would be absurd to imagine that all of them have sprung from Adam." On the other hand, he observes that "although typhus varies in its severity and duration at different times and under different circumstances, there is no evidence of any change in type or essential characters. The typhus of modern times is the same as that described by Frascatorius and Cardanus." The pages of the *Edinburgh Medical Journal*, 1856–58, contain a discussion on the tran-

epidemiologist are unfortunately still of this primitive character; there is no standard case of typhus fever deposited at Kew and no one proposes to test strains of small-pox by their ability to kill unvaccinated vagrants of given weights in specified times.

Murchison has remarked that "in distinguishing the different forms of continued fever too much reliance has been placed on their symptoms and pathology, while there has been a want of sufficient investigation of their causes." With elaboration of the germ theory the pendulum has swung to the other extreme and it is now quite orthodox doctrine to hold that the presence of a particular germ spells specific disease; indeed, it may be questioned whether some modern bacteriologists, in the light of the demonstration of diphtheria, cholera, and enteric fever bacilli in persons presenting no symptoms of illness, would not feel that Murchison much exaggerated the difficulties inherent in a hypothesis requiring the co-existence of all pathogenic organisms in one individual. "The germ," Sir William Collins says, "has perhaps been too much with us, and the paramount importance of soil has been absurdly underrated." Or, to quote Dr. G. Newman, "The early school of preventive medicine declared for the health of the individual and laid the emphasis upon predisposition; the modern school have declared for the infecting agent and have laid emphasis upon the bacillus. The truth is to be found in a right perception of the action and interaction of the tissues and the bacillus." Or as Dr. F. G. Clemow expresses it, "Though constantly spoken of as if it were a material tangible entity disease is, in fact, no such thing. It is only a morbid phenomenon, or rather a group of morbid processes, in the tissues of a particular animal organism. In the language of logic it is not even a phenomenon but an epiphenomenon."

Here is a fertile source of difficulty and misapprehension. It has been suggested that rhythmical evolutionary changes in the life-history of micro-organisms may prove explanatory of waves of disease, but is the rhythm manifested in the micro-organism or in that epiphenomenon the interaction between germ and tissues? If the latter we dispose at once of a difficulty. The fossils in the strata do not recur cyclically; a species once extinguished never reappears. Is this true also in the case of disease organisms or must these "lowly organisms, on the borderland of the animal and vegetable kingdoms, on the threshold, as it were, of the organic and inorganic, ... whose cycle may be less than an hour and whose rate of propagation is incalculable" (Collins) be-

Kermack and McKendrick 1927: epidemic thresholds and outbreaks

- **epidemic threshold**: the density of susceptibles must exceed a critical value in order for an **epidemic outbreak** to occur
- differential equations, calculus

A Contribution to the Mathematical Theory of Epidemics.

By W. O. Kermack and A. G. McKendrick.

(Communicated by Sir Gilbert Walker, F.R.S.—Received May 13, 1927.)

(From the Laboratory of the Royal College of Physicians, Edinburgh.)

Introduction.

(1) One of the most striking features in the study of epidemics is the difficulty of finding a causal factor which appears to be adequate to account for the magnitude of the frequent epidemics of disease which visit almost every population. It was with a view to obtaining more insight regarding the effects of the various factors which govern the spread of contagious epidemics that the present investigation was undertaken. Reference may here be made to the work of Ross and Hudson (1915–17) in which the same problem is attacked. The problem is here carried to a further stage, and it is considered from a point of view which is in one sense more general. The problem may be summarised as follows: One (or more) infected person is introduced into a community of individuals, more or less susceptible to the disease in question. The disease spreads from

Hethcote's leading survey in 2000

motivated by a range of infectious diseases and outbreaks,

one thousand and one models have been analyzed mathematically

e.g., models with age structure, heterogeneity, and spatial structure

threshold theorems for **epidemic outbreaks**

SIAM REVIEW
Vol. 42, No. 4, pp. 599–653

The Mathematics of Infectious Diseases*

Herbert W. Hethcote†

Abstract. Many models for the spread of infectious diseases in populations have been analyzed mathematically and applied to specific diseases. Threshold theorems involving the basic reproduction number R_0, the contact number σ, and the replacement number R are reviewed for the classic SIR epidemic and endemic models. Similar results with new expressions for R_0 are obtained for MSEIR and SEIR endemic models with either continuous age or age groups. Values of R_0 and σ are estimated for various diseases including measles in Niger and pertussis in the United States. Previous models with age structure, heterogeneity, and spatial structure are surveyed.

Key words. thresholds, basic reproduction number, contact number, epidemiology, infectious diseases

AMS subject classifications. Primary, 92D30; Secondary, 34C23, 34C60, 35B32, 35F25

PII. S0036144500371907

1. Introduction. The effectiveness of improved sanitation, antibiotics, and vaccination programs created a confidence in the 1960s that infectious diseases would soon be eliminated. Consequently, chronic diseases such as cardiovascular disease and

Historical review of mathematical epidemiology

- Daniel Bernoulli. Essai d'une nouvelle analyse de la mortalité causée par la petite vérole, et des avantages de l'inoculation pour la prévenir. *Mémoires de Mathématiques et de Physique, Académie Royale des Sciences*, pages 1–45, 1760

- W. H. Hamer. On epidemic disease in England. *The Lancet*, 167(4305):569–574, 1906. doi:10.1016/S0140-6736(01)80187-2

- W. O. Kermack and A. G. McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society A*, 115:700–721, 1927. doi:10.1098/rspa.1927.0118

- N. T. J. Bailey. *The Mathematical Theory of Infectious Diseases*. Griffin, 1957

- H. W. Hethcote. The mathematics of infectious diseases. *SIAM Review*, 42(4):599–653, 2000. doi:10.1137/S0036144500371907

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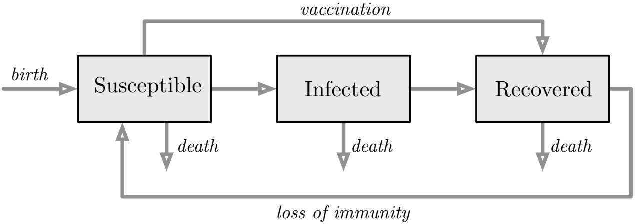
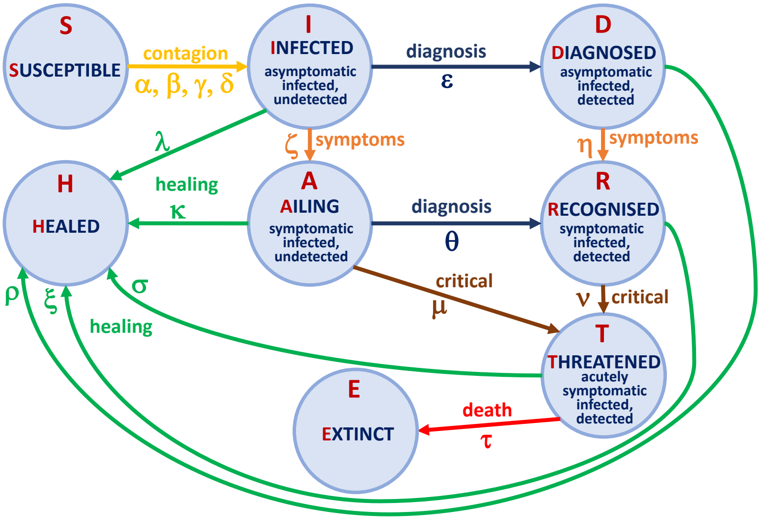
Concept #1: Compartmental Models

each individual is in one of multiple possible states:

Two types of transitions:

1. $S \rightarrow I$: interaction between a susceptible and an infected
2. $I \rightarrow R$: spontaneous, independent of interactions


vaccination
birth
Susceptible
Infected
Recovered
death
death
death
loss of immunity



G. Giordano, F. Blanchini, R. Bruno, P. Colaneri, A. Di Filippo, A. Di Matteo, and M. Colaneri. A SIDARTHE model of COVID-19 epidemic in Italy, 2020. Arxiv preprint. URL: https://arxiv.org/pdf/2003.09861

Concept # 2: Simplest SIR model

$$\boxed{s = \text{fraction susceptible}} \xrightarrow{\beta s x} \boxed{x = \text{fraction infected}} \xrightarrow{\gamma x} \boxed{r = \text{fraction recovered}}$$

differential equation = fundamental mechanism to compute an evolution

given infection rate β and recovery rate γ,
given initial values $s(0)$, $x(0)$, $r(0)$:

$$\begin{aligned}
\dot{s} &= -\beta s x \\
\dot{x} &= \beta s x - \gamma x \\
\dot{r} &= \gamma x
\end{aligned}$$

Scope of simplest SIR model

In a population of n individuals, on average:

1. contacts between uniformly randomly selected individuals
   - **contact rate** $\beta_{\mathbf{c}} > 0$ so that

     during $(t, t + \Delta t)$, $n\beta_c\Delta t$ *individuals meet other* $n\beta_c\Delta t$

     i.e., each individual meets $\beta_c\Delta t$
   - **transmission fraction** $0 < \beta_{\mathbf{t}} < 1$ resulting in infection

2. **recovery rate** $\gamma > 0$ so that

   during $(t, t + \Delta t)$, $n\gamma\Delta t$ *individuals recover*

   i.e., **infective period** $= 1/\gamma$

Therefore, on average

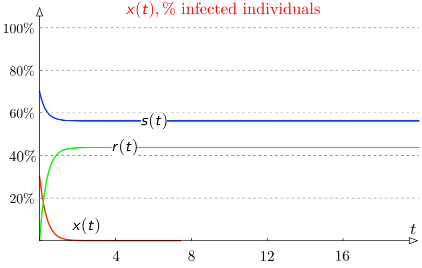
$$\frac{x(t + \Delta t) - x(t)}{\Delta t} = + \underbrace{2\beta_t\beta_c}_{\textbf{rate } \beta} \quad \underbrace{x(t)s(t)}_{\text{Hamer's product}} \quad - \quad \gamma x(t)$$

Predictions of simplest SIR model

$$\dot{s} = -\beta s x$$

$$\dot{x} = \beta s x - \gamma x \qquad = \gamma\left(\frac{\beta}{\gamma} s - 1\right) x$$

$$\dot{r} = \gamma x$$



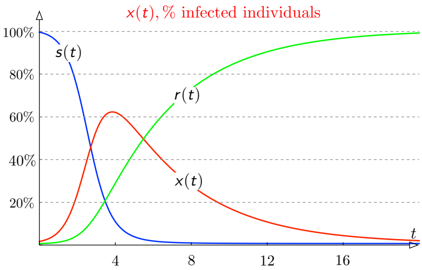
(a) NO OUTBREAK (b) OUTBREAK

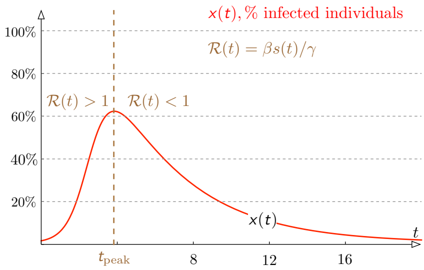
Concept #3: Reproduction number and epidemic threshold

Basic reproduction number $\mathcal{R}_0 =$ expected number of secondary cases produced by a typical infective individual, at start of epidemic

$$\mathcal{R}_0 = \beta \times 1/\gamma \times s(0)$$
$$\approx \Big(\text{(contacts/day)} \times \text{(transmission)}\Big) \times \text{(infective days)} \times s(0)$$

$$\mathcal{R}_0 > 1 \quad \Longrightarrow \quad \text{exponential growth}$$





Speculations about uncertain COVID-19 parameters

Values before: social distancing, other NPI measures, and fear

Quantity	Value	Explanation
$\mathcal{R}_0$	2.2-2.7 persons	Highly dependent upon region, age group, etc. Some estimates are much higher. (source: wikipedia)
incubation period	5 days	(median), between exposure and first symptoms, 97.5% before 12 days. (source: wikipedia)
infective period $1/\gamma$	5 days	"people can test positive for COVID-19 from 1-3 days before they develop symptoms" (source: Report WHO China Joint Mission). includes asymptomatic infective people.
doubling time	2-7 days	(source: Imperial College report and "Epidemic doubling time of the COVID-19 epidemic by Chinese province")
asymptom cases	5% - 80%	

Question 1: what are individual factors in $\mathcal{R}_0$? For thought experiments – without further evidence – imagine

$$\underbrace{\mathcal{R}_0}_{2.5\text{ persons}} \approx \Big(\underbrace{(\text{contacts/day})}_{2\text{ persons/day}} \times \underbrace{(\text{transmission})}_{25\%} \Big) \times \underbrace{(\text{infective days})}_{5\text{ days}} \times \underbrace{s(0)}_{100\%}$$

Question 2: how to compute the doubling time? While $s \approx 1$,

$$t_{\text{doubling}} \approx \frac{\ln(2)}{(\beta - \gamma)} = \frac{\ln(2)}{1/2 - 1/5} \approx 2.3\text{days}$$

Question 3 (Herd Immunity): what percentage of the population x^* needs to have immunity in order for $\mathcal{R}(t) = 1$? Assume all population is susceptible $s(0) = 100\%$, then

$$1 = \mathcal{R}(t) = \mathcal{R}_0 s(t^*) \quad \Longrightarrow \quad x^* = 1 - s(t^*) = 1 - \frac{1}{\mathcal{R}_0} = 60\%$$

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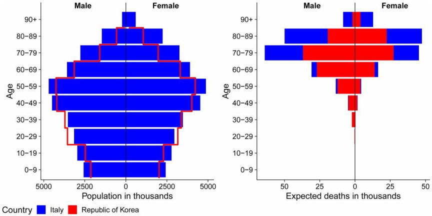
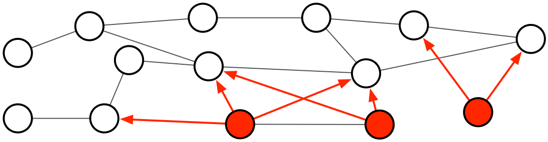
(c) Heterogeneity by spatial position



(d) Heterogeneity by age structure

More accurate models: Structured Multi-group SIR

$n =$ number of homogeneous groups in heterogeneous population
based on spatial position, age, social behavior



1. for each group, s_i susceptible, x_i infected, or r_i recovered
2. heterogeneous recovery rate γ_i
3. heterogeneous meeting/contact rate $(\beta_c)_{ij}$ between i and j

$$\dot{x} = \beta s x - \gamma x \qquad \Longrightarrow \qquad \dot{x}_i = \sum\nolimits_{j=1}^{n} \beta_t (\beta_c)_{ij} s_i x_j - \gamma_i x_i$$

Parameters: infection matrix $\beta_t \beta_c$, recovery rates γ_i

Stochastic SIR models

- In the spirit of "simplest SIR" = compartments with transitions
- No explicit estimation/computation of contact rates
- From differential equations to **stochastic virtual worlds**

Imperial College model, Report March 16th, 2020

1. synthetic individuals – by spatial position, age, social behavior
2. synthetic contacts at: (1) home/residence, (2) central hubs (work, schools, markets, churches), (3) local neighborhoods
3. parameters of person-to-person contact based on large tuning data
4. **stochastic individual-based simulation**
   large-scale Monte-Carlo simulations on HPC clusters

Stochastic simulation + visualization: https://youtu.be/gxAaO2rsdIs

N. M. Ferguson et al. Impact of non-pharmaceutical interventions (NPIs) to reduce COVID19 mortality and healthcare demand. Technical report, Imperial College, March 2020. doi:10.25561/77482

Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand

Neil M Ferguson, Daniel Laydon, Gemma Nedjati-Gilani, Natsuko Imai, Kylie Ainslie, Marc Baguelin, Sangeeta Bhatia, Adhiratha Boonyasiri, Zulma Cucunubá, Gina Cuomo-Dannenburg, Amy Dighe, Ilaria Dorigatti, Han Fu, Katy Gaythorpe, Will Green, Arran Hamlet, Wes Hinsley, Lucy C Okell, Sabine van Elsland, Hayley Thompson, Robert Verity, Erik Volz, Haowei Wang, Yuanrong Wang, Patrick GT Walker, Caroline Walters, Peter Winskill, Charles Whittaker, Christl A Donnelly, Steven Riley, Azra C Ghani.

On behalf of the Imperial College COVID-19 Response Team

WHO Collaborating Centre for Infectious Disease Modelling
MRC Centre for Global Infectious Disease Analysis
Abdul Latif Jameel Institute for Disease and Emergency Analytics
Imperial College London

Correspondence: neil.ferguson@imperial.ac.uk

Summary

The global impact of COVID-19 has been profound, and the public health threat it represents is the most serious seen in a respiratory virus since the 1918 H1N1 influenza pandemic. Here we present the results of epidemiological modelling which has informed policymaking in the UK and other countries in recent weeks. In the absence of a COVID-19 vaccine, we assess the potential role of a number of public health measures – so-called non-pharmaceutical interventions (NPIs) – aimed at reducing

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Statistical model estimation

- do not impose a mechanism for transmission, do not model micro-interactions and micro-transitions between compartments
- direct interpolation of signals
- model empirically-observed death rate curves

Institute for Health Metrics and Evaluation (IHME), lead by Dr Murray. MedRxiv paper March 30th, 2020.

1. collected: age-specific deaths by day, start date for NPIs, hospital beds and ICU capacity, & (starting April 17) mobile phone data
2. indirect standardization of age structure
3. only "admin 1 locations" with .31 death/million and time-referenced
4. curve-fitting: cumulative death rate as Gaussian error function
5. statistical covariate: # days from .31 threshold to NPI day estimated from Wuhan data (before and after NPI impositions)

IHME COVID-19 health service utilization forecasting team, C. Murray. Forecasting COVID-19 impact on hospital bed-days, ICU-days, ventilator-days and deaths by US state in the next 4 months. *medRxiv*, 2020. URL: https://covid19.healthdata.org/, doi:10.1101/2020.03.27.20043752

Forecasting COVID-19 impact on hospital bed-days, ICU-days, ventilator-days and deaths by US state in the next 4 months

IHME COVID-19 health service utilization forecasting team

Key Points

Question: Assuming social distancing measures are maintained, what are the forecasted gaps in available health service resources and number of deaths from the COVID-19 pandemic for each state in the United States?

Findings: Using a statistical model, we predict excess demand will be 64,175 (95% UI 7,977 to 251,059) total beds and 17,380 (95% UI 2,432 to 57,955) ICU beds at the peak of COVID-19. Peak ventilator use is predicted to be 19,481 (95% UI 9,767 to 39,674) ventilators. Peak demand will be in the second week of April. We estimate 81,114 (95% UI 38,242 to 162,106) deaths in the United States from COVID-19 over the next 4 months.

Meaning: Even with social distancing measures enacted and sustained, the peak demand for hospital services due to the COVID-19 pandemic is likely going to exceed capacity substantially. Alongside the implementation and enforcement of social distancing measures, there is an urgent need to develop and implement plans to reduce non-COVID-19 demand for and temporarily increase capacity of health facilities.

IHME

COVID-19 Projections

GHDX VIZ HUB

Current social distancing assumed until infections minimized and containment implemented

Last updated April 17, 2020 (Pacific Time).

FAQ | Update Notes | Article

All dates below are calculated based on the local time of the selected location.

California

Government-mandated social distancing

Mass gathering restrictions **March 11, 2020**	Initial business closure **March 19, 2020**	Educational facilities closed **March 19, 2020**
Non-essential services closed **March 19, 2020**	Stay at home order **March 19, 2020**	Travel severely limited **Not implemented**

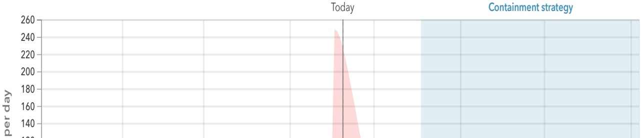
Deaths per day

4 days since projected peak in daily deaths

96 COVID-19 deaths projected on April 16, 2020

Containment strategy

After May 18, 2020, relaxing social distancing may be possible with containment strategies that include testing, contact tracing, isolation, and limiting gathering size.



References

Sample references about SIR models

1. N. M. Ferguson et al. Impact of non-pharmaceutical interventions (NPIs) to reduce COVID19 mortality and healthcare demand. Technical report, Imperial College, March 2020. doi:10.25561/77482
2. Jose Lourenco, Robert Paton, Mahan Ghafari, Moritz Kraemer, Craig Thompson, Peter Simmonds, Paul Klenerman, and Sunetra Gupta. Fundamental principles of epidemic spread highlight the immediate need for large-scale serological surveys to assess the stage of the SARS-CoV-2 epidemic. *medRxiv*, 2020. doi:10.1101/2020.03.24.20042291
3. A. J. Kucharski et al. Early dynamics of transmission and control of COVID-19: a mathematical modelling study. *The Lancet Infectious Diseases*, 2020. doi:10.1016/S1473-3099(20)30144-4

Sample references about statistical models

1. IHME COVID-19 health service utilization forecasting team, C. Murray. Forecasting COVID-19 impact on hospital bed-days, ICU-days, ventilator-days and deaths by US state in the next 4 months. *medRxiv*, 2020. URL: https://covid19.healthdata.org/, doi:10.1101/2020.03.27.20043752
2. G. Sotgiu, G. A. Gerli, S. Centanni, M. Miozzo, G. W. Canonica, J. B. Soriano, and C. Virchow. Advanced forecasting of SARS-CoV-2 related deaths in Italy, Germany, Spain, and New York State. *Allergy*. doi:10.1111/all.14327

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Evaluation of SIR models by IHME

From IHME MedRxiv on March 30th, criticism of SIR model:

1. "random mixing between all individuals in a given population"
2. given current estimates of $\mathcal{R}_0$, SIR models "generally" over-predict
3. "results of these models are sensitive to starting assumptions"
4. "SIR models with assumptions of random mixing can overestimate [...] by not taking into account behavioral change and government-mandated action"

Evaluation of Statistical models by UK scientists

From CNN article on April 9th:

- From IHME website as of April 9th, prediction of 66K deaths in the UK by early August. (As of April 20th, IHME predicts 37.5K deaths)
- Imperial College model predicts 20K-30K, if NPIs are imposed

Professor Sylvia Richardson, Cambridge University and co-chair of the Royal Statistical Society Task Force on Covid-19, says

1. IHME's projections are based on "very strong assumptions about the way the epidemic will progress."
2. "based mostly on using the experience in other countries to fit a smooth curve to the counts of deaths reported so far in the UK, rather than any modeling of the epidemic itself."
3. "Methods like this are well known for being extremely sensitive, and are likely to change dramatically as new information comes in"

https://www.cnn.com/2020/04/08/health/uk-death-toll-coronavirus-estimate-gbr-intl/index.html

Summary

- 260 years old mathematical journey. Results have been stellar.
- simplest SIR model explains emerging phenomena
  salient features: $\mathcal{R}(t)$, growth/decay, and explains NPIs
- more realistic, but still extremely data-dependent, models:
  1. stochastic structured/multi-group SIR models
  2. statistical models based on data fitting

name	description	scope
simplest SIR	low complexity explanation	crucial basic understanding
Stochastic SIR	mechanistic explanation	assessment of existing and novel NPIs
Statistical models	direct data fitting	prediction

- **from data to parameters and state** – next webinars in series

Outline

1. historical notes
2. introduction to mathematical epidemiology
    1. the simplest SIR model
    2. stochastic SIR models
    3. direct statistical estimation
3. summary evaluation
4. **conclusion on non-pharmaceutical interventions (NPIs)**

Non-pharmaceutical interventions

Recall

$$
\begin{aligned}
\mathcal{R}(t) &= \Big(\beta_{\mathrm{m}} \times \beta_{\mathrm{t}}\Big) \times 1/\gamma \times s(t) \\
&\approx \Big((\text{contacts/day}) \times (\text{transmission})\Big) \times (\text{infective days}) \times s(t)
\end{aligned}
$$

Non-pharmaceutical interventions aimed at decreasing $\mathcal{R}(t)$:

NPI	effect
washing hands and wearing masks	decrease infection transmission β_{t}
social distancing and travel restrictions	decrease contact rates β_{m}
testing leading to quarantine	decreases infective duration $1/\gamma$
contact tracing leading to quarantine	decreases infective duration $1/\gamma$

Concluding Question: how can we safely reopen UCSB?

- What if we were to perform extensive testing, contact tracing and other measures — for those students willing to consent?
- What models and what data would we need?
- What would a comprehensive approach entail?
  campus infrastructure = health center, classroom, dining
  digital infrastructure = mobile app, backend ...