Court File No. CV-22-00691880-0000

ONTARIO
SUPERIOR COURT OF JUSTICE

B E T W E E N:

Dr. BYRAM BRIDLE

Plaintiff

and

UNIVERSITY OF GUELPH, JEFFREY WICHTEL, LAURIE ARNOTT, CHARLOTTE YATES, SCOTT WEESE, GLEN PYLE, ANDREW PEREGRINE, DOROTHEE BIENZLE, AMY GREER, DAVID FISMAN, NICK DULEY, JANE OR JOHN DOE JUNIOR SCIENTIST

Defendants

MOTION RECORD OF DEFENDANT DAVID FISMAN
(Returnable November 19, 2024)
Vol. I of II

June 30, 2023

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AND TO: **JANE OR JOHN DOE JUNIOR SCIENTIST**

Defendant

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NOTICE OF MOTION

The Defendant, David Fisman will make a Motion to a Judge on November 19, 2024, at 10:00 a.m., or as soon after that as the Motion can be heard at the courthouse, 330 University Avenue, Toronto, Ontario, M5G 1R7.

PROPOSED METHOD OF HEARING: The Motion is to be heard in person.

THE MOTION IS FOR*:*

(a) An Order dismissing the action against Dr. David Fisman ("Dr. Fisman") pursuant to section 137.1(3) of the *Courts of Justice Act*, RSO 1990, c C.43;

(b) Costs of this motion and of the action on a full indemnity basis pursuant to section 137.1(7) of the *Courts of Justice Act;*

(c) Such further and other Relief as to this Honourable Court may deem just.

THE GROUNDS FOR THE MOTION ARE*:*

Overview

(a) The plaintiff, Dr. Byram Bridle ("Dr. Bridle") commenced the action on December 19, 2022, by Statement of Claim;

(b) The plaintiff, Dr. Bridle, is a veterinarian and Associate Professor of Viral Immunology in the Department of Pathobiology at the Ontario Veterinary College at the University of Guelph;

(c) The plaintiff, Dr. Bridle is a high-profile critic of the COVID-19 public health response and advice. In 2021, he participated in a series of interviews and speeches and authored a number of articles criticizing the safety and efficacy consensus of COVID-19 vaccines;

(d) The defendant, Dr. David Fisman, is a physician in Toronto, Ontario specializing in infectious diseases. Dr. Fisman is a professor in epidemiology at the University of Toronto's Dalla Lana School of Public Health;

(e) Between March 2020 – August 2021, Dr. Fisman was part of Ontario's COVID-19 Science Advisory Table. Dr. Fisman regularly provided information and insights to

the public regarding the COVID-19 pandemic, including through his Twitter account @DFisman;

(f) Following a radio interview on May 28, 2021 in which Dr. Bridle claimed COVID-19 vaccinations were unsafe, Dr. Fisman posted three tweets in which he expressed his disagreement with Dr. Bridle's claims;

(g) In his tweets, he directed his followers to the website byrambridle.com, which provides responses to the claims of Dr. Bridle. Dr. Fisman is not the author or creator of the website;

(h) Dr. Bridle's interview gained international media attention, as the claims he made were contrary to the overwhelming majority of scientific opinion at the time. Dr. Fisman was contacted by a reporter at USA TODAY about Dr. Bridle's claims. Dr. Fisman responded, providing his opinion, which was that Dr. Bridle's claims were not evidence-based;

(i) Dr. Fisman's intention in posting his tweets and responding to an inquiry from a reporter was to warn against the spreading of misinformation to the public in regards to COVID-19 vaccines;

(j) Through this action, the Plaintiff claims to have suffered a total of up to $2,500,000 in joint and several damages from the 11 Defendants, including Dr. Fisman. Dr. Fisman does not personally know Dr. Bridle. He has never met him.

(k) The other Defendants are the University of Guelph and several of its senior administrators, including the President and Dean of the Department of Veterinary Medicine, professors, research chairs and human resource specialists;

(l) The Plaintiff alleges that Dr. Fisman is jointly and severally liable with the other Defendants in the torts of online harassment, conspiracy, interference with economic interest and endangerment of Plaintiff's life;

The SLAPP

(m) The causes of action asserted against Dr. Fisman arise from:

(i) A tweet posted by Dr. Fisman on May 29, 2021;

(ii) A tweet posted by Dr. Fisman on May 30, 2021;

(iii) A tweet posted by Dr. Fisman on May 31, 2021 (incorrectly dated as June 2021 in the Statement of Claim); and

(iv) An email written to a USA TODAY journalist on June 2, 2021 (collectively the "Words Complained Of");

(n) The Words Complained Of were said during a one week time frame. They were said in response to statements made by Dr. Bridle and were made by Dr. Fisman in good faith and based on a sincere concern about the potential for harm arising from an immunologist spreading misinformation;

(o) The Words Complained of relate to matters of profound public interest, being health measures in response to the global COVID-19 pandemic, public health, medical science, and professional ethics;

(p) The Words Complained Of, consisting of three tweets and one email cannot, at law constitute online harassment, as alleged in the Statement of Claim:

(i) The communications and conduct in the Words Complained Of occurred over one week;

(ii) Dr. Fisman did not engage in the Words Complained Of maliciously or recklessly. Dr. Fisman's sole purpose in writing the Words Complained Of was to direct the public to evidence and data-based research on vaccine efficacy;

(iii) The Words Complained Of are neither outrageous in character or extreme in degree, and do not go beyond all possible bounds of decency and tolerance. On their face, The Words Complained of contain no unfair or inflammatory language;

(iv) Dr. Fisman had no intention to cause fear, anxiety, emotional upset or to impugn the dignity of the plaintiff. The Words Complained Of discussed statements made by Dr. Bridle but at no time did Dr. Fisman contact Dr. Bride directly. Instead, the Plaintiff complains that Dr. Fisman harassed him without ever even contacting Dr. Bridle or directing any such statements to him; and

(v) The Plaintiff did not suffer any such harm;

(q) The Words Complained Of were not part of any broader conspiracy with the Defendants or with anyone whatsoever. Beyond bald allegations, there is no evidence pleaded of any conspiracy;

(r) Further, Dr. Fisman is not the author of any complained of website or the twitter handle @byrambridle.com. Dr. Fisman's sole purpose in tweeting links to that website or twitter handle was to direct his followers to evidence and data-based information on vaccine efficacy;

(s) The Words Complained Of are not capable of constituting conspiracy, interference with economic interest or endangerment of the Plaintiff's life as alleged in the Statement of Claim;

(t) The Plaintiff has not plead any facts, which, if true, would prove that Dr. Fisman is liable to him for online harassment, conspiracy, interference with economic interest and endangerment of Plaintiff's life;

(u) In addition, or alternative, the Plaintiff cannot satisfy the Court that there are grounds to believe that Dr. Fisman has no valid defences, and, as such, this claim must be dismissed;

(v) The public interest in protecting the expression made through Dr. Fisman's expression significantly outweighs the public interest in permitting the proceeding to continue;

(w) Dr. Bridle has suffered no harm to his reputation as a result of the Words Complained Of;

(x) If Dr. Bridle has suffered any harm in connection with any of the Words Complained Of, which is denied, it is not caused to or contributed to by Dr. Fisman, who has no appointment or affiliation with the University of Guelph, but rather is a result of Dr. Bridle's own conduct as a result of the professional and public criticism that he has received from his statements;

(y) It is in the public interest to safeguard and encourage Dr. Fisman's speech;

(z) If this action is allowed to continue, it will have a chilling effect and deter other physicians and members of the public from engaging with matters of public health, and discussing and evaluating misinformation about public health measures;

(aa) The action is a strategic lawsuit against public participation, as contemplated by s. 137.1 of the *Courts of Justice Act*, intended to intimidate, censor and silence critics of the Plaintiffs and stifle debate about matters of critical public interest;

(bb) Section 137.1 of the *Courts of Justice Act;*

(cc) Section 2(b) of the Canadian Charter of Rights and Freedoms;

(dd) Rule 37 of the Rules of Civil Procedure;

(ee) Such further and other grounds as the lawyers may advise.

THE FOLLOWING DOCUMENTARY EVIDENCE will be used at the hearing of the Motion:

(a) The affidavit of Dr. David Fisman, on a date to be sworn; and

(b) Such further and other evidence as the lawyers may advise and this Honourable Court may permit.

June 30, 2023

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AND TO: **JANE OR JOHN DOE JUNIOR SCIENTIST**

Defendant

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-and-

UNIVERSITY OF GUELPH et al.
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Court File No. CV-22-00691880-000

ONTARIO
SUPERIOR COURT OF JUSTICE

PROCEEDING COMMENCED AT TORONTO

NOTICE OF MOTION

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Defendants

AFFIDAVIT OF DR. DAVID FISMAN

I, Dr. David Fisman, of the City of Toronto, in the Province of Ontario, MAKE OATH AND SAY:

1. I am one of the Defendants in this proceeding, and, as such, have knowledge of the matters contained in this Affidavit.

Professional Background and Activities

2. I am a physician residing in Toronto, specializing in infectious diseases. I work as a professor in epidemiology at the Dalla Lana School of Public Health at the University of Toronto and as an infection disease specialist and consultant at the London Health Sciences Centre in London, Ontario.

3. In July 2020 I was appointed to Ontario's COVID-19 Science Advisory Table (the "Science Table"), which consisted of independent scientific experts who provided advice to the Government of Ontario about COVID-19. I resigned from the Science Table in August 2021. Attached as **Exhibit "A"** to this affidavit is a copy of my curriculum vitae.

4. I use my Twitter account regularly, posting public messages under my username @DFisman, where I have approximately 128,700 followers. I use Twitter as a platform to express my views on a number of topics, including to communicate my opinions on public health measures related to the COVID-19 pandemic.

The Covid Pandemic

5. On March 11, 2020, the World Health Organization declared COVID-19 a global pandemic. It is considered to be the biggest global public health challenge since the 1918 influenza pandemic.

6. The World Health Organizations COVID-19 Dashboard states that, as of May 2023, there has been over 765,000,000 confirmed cases of COVID-19, and over 6,900,000 deaths. Deaths are believed to be under-reported globally by a factor of 3-4, such that the true death toll is likely over 20 million to date. Globally, as of May 2023, over 13,000,000 vaccine doses have been administered. A link to the WHO Coronavirus (COVID-19) interactive Dashboard is included here: https://covid19.who.int/

7. In Canada, the COVID-19 vaccine rollout began in December 2020. As reported by Health Canada, in the week ending on May 22, 2021, over 6,600,000 first doses of the vaccine were

administered. A link to the Government of Canada's COVID-19 vaccination: Doses administered page is available here: https://health-infobase.canada.ca/covid-19/vaccine-administration/

8. A publication in the journal *Lancet* estimated that vaccination against COVID-19 had prevented approximately 20 million excess deaths worldwide as of December 2021. A copy of the *Lancet* article published June 23, 2022 is attached hereto as **Exhibit "B"**.

Statements at Issue

9. Dr. Byram Bridle's claim against me is based on three tweets and an article that appeared in the online edition of USA TODAY.

10. In 2021, I became aware that Dr. Bridle was conducting a series of speaking engagements, interviews and television appearances in which he publicly criticized the development and use of COVID-19 vaccines.

11. On May 28 2021, Dr. Bridle participated in a radio interview with Alex Pierson, a radio host with AM640 in Toronto, in which he claimed:

> We made a big mistake. We didn't realize it until now, we thought the spike protein was a great target antigen. We never knew the spike protein itself was a toxin and was a pathogenic protein so by vaccinating people we are inadvertently inoculating them with a toxin.

12. In the same interview, Dr. Bridle also claimed the spike proteins generated by the vaccines do not stay in the shoulder muscle, but spread and cause "so much damage in other parts of the bodies of the vaccinated." Dr. Bridle further claimed that the spike protein had been unexpectedly found in the bloodstream, and was linked to blood clots, heart and brain damage. A link to Dr.

Bridle's interview is available here: https://podcasts.apple.com/ca/podcast/new-peer-reviewed-study-on-covid-19-vaccines-suggests/id1318830191?i=1000523346577

13. Dr. Bridle's interview with Ms. Pierson gained international attention because the claims he made were contrary to the overwhelming majority of scientific opinion at the time. Attached hereto as **Exhibit "C"** is a copy of an Associated Press News article titled "Spike Protein Produced by Vaccine Not Toxic" by Beatrice Dupuy dated June 9, 2021.

14. Attached hereto as **Exhibit "D"** is an article from Reuters Fact Check titled "Fact Check – No evidence spike proteins from COVID-19 vaccines are toxic" dated June 15, 2021.

15. In my opinion, Dr. Bridle's comments had the potential to harm or halt Ontario's vaccine roll out. Late May and early June 2021 marked a crucial timeframe in the vaccine rollout. At this time efficacy of vaccines against Wuhan-variant COVID-19 as well as the emerging alpha variant remained high, and the apparent reproduction number of COVID-19 remained low enough, in combination with high vaccine efficacy, that establishment of herd immunity was thought to be possible. I attach two articles which support my opinion on this. Attached hereto as **Exhibit "E"** is an article by Nicholas G. Davies et al. titled "Estimated transmissibility and impact of SARS-CoV-2 lineage B.1.1.7 in England" dated April 9, 2021. Attached hereto as **Exhibit "F"** is an article by Yaki Saciuk et al. titled "Pfizer-BioNTech vaccine effectiveness against Sars-Cov-2 infection: Findings from a large observational study in Israel".

16. Issues around waning protection from vaccination were starting to be recognized but their significance was unclear. Attached hereto as **Exhibit "G"** is an article by Yair Goldberg et al. titled "Waning Immunity after the BNT162b2 Vaccine in Israel" dated December 9, 2021.

17. Mortality from COVID-19 had declined dramatically in the face of high vaccine uptake by this time, but the impact of COVID-19 remained concerning, with Ontario having had to establish field hospitals in March 2021 due to overflowing intensive care units. A copy of an article titled "New Toronto field hospital prepared to accept COVID-19 patients as ICUs overflow" by Sannah Choi and Muriel Draaisma dated April 20, 2021 is attached hereto as **Exhibit "H"**.

18. It was my opinion at the time that COVID-19 vaccination represented an extremely important tool for the health and safety of the Canadian population in the face of a major health crisis.

19. On May 29, 2021, I posted the following tweet:

> I've had questions over the past 48h about vaccine safety concerns aired Dr Byram Bridle at @UofGuelphOAC in some recent interviews. I don't know Dr Bridle but he's a legit immunologist. Some claims, however are not data based, and are answered here: byrambridle.com

A copy of the May 29, 2021 tweet is attached hereto as **Exhibit "I"**.

20. My intention with this tweet was to warn against the spreading of misinformation to the public in regards to COVID-19 vaccines.

21. Byrambridle.com is a website that, based on internet archives, was created in or around May 2021. I am not the author or creator of the website byrambridle.com, nor do I play any role in its maintenance. Byrambridle.com is still in operation. It presently contains a list of Dr. Bridle's claims, followed by responses to these claims from Dr. Bridle's colleagues, the media and the legal system. The website explicitly states that the author is a "Concerned Scientist" who created the website in response to Dr. Bridle's claims. I directed users to the website as it contains data-based

information on COVID-19 vaccine safety concerns. A copy of the current homepage of ByramBridle.com is attached hereto as **Exhibit "J"**. A copy of the homepage of ByramBridle.com as of May 29, 2021, is attached hereto as **Exhibit "K"**

22. I am also not involved in the creation or maintenance of the @byrambridle Twitter account.

23. On May 30, 2023, Dr. Pyle, another scientist active on Twitter wrote the following tweet seemingly in response to Dr. Bridle's interview with Ms. Pierson:

> The paper Byram cited doesn't support his claim. That's pretty telling that a study cited to support his claims actually goes against those claims.

24. I quoted Dr. Pyle's tweet and posted the following tweet:

> An excellent follow for good immune science from @UofGuelphOAC is Dr @glenpyle, who has addressed some of the misinformation in his own tweets.

A copy of my May 30, 2021 tweet quoting Dr. Pyle's tweet is attached hereto as **Exhibit "L"**.

25. The purpose of my May 30, 2021 tweet was to direct the public to evidence-based information related to the COVID-19 vaccine.

26. On May 31, 2021, I posted the following tweet:

> The website debunking Dr. Bridle's covid-19 vaccine claims has been updated with lots of peer-reviewed science that attests to the safety of vaccines.
>
> Byrambridle.com
>
> And for those who think I made or organized this website: nope. But grateful to the scientist who did.

27. I then responded to that tweet writing:

> A friend indicates that Dr Bridle's interview caused his parents to cancel their vaccine appointments. This is not ok.

A copy of my Twitter thread from May 31, 2021 is attached hereto as **Exhibit "M"**.

28. The purpose of my May 31, 2021 Twitter thread was to direct members of the public to a website which contained peer-reviewed science-based information on the safety of vaccines, and to express my frustration that members of the public were being dissuaded from getting vaccinated against COVID-19 because of, what was, in my opinion, misinformation on the efficacy of vaccines.

29. Contrary to Dr. Bridle's assertions, I was not referring to his parents in this tweet. I was referring to the parents of the friend referred to.

My sole intention in commenting on Dr. Bridle's claims on Twitter was to direct my Twitter followers to evidence and data-based information to show that vaccines were safe, contrary to Dr. Bridle's statements.

30. On June 2, 2021, I received an email from Daniel Funke, a reporter for USA TODAY, who asked what I made of Dr. Bridle's claims in general. I responded giving my opinion, which is that Dr. Bridle's claims are implausible and not data based. In my response, I copied the Defendants Dr. Scott Weese, Dr. Amy Greer and Dr. Glen Pyle, as I believed they could provide Mr. Funke with relevant information as well. A copy of my email dated June 2, 2021 is attached hereto as **Exhibit "N"**.

31. This email is the basis of the comments made in the USA TODAY article complained of by Dr. Bridle in his Statement of Claim, a copy of which is attached hereto as **Exhibit "O"**

Retaliation by Dr. Bridle

32. Throughout the pandemic, I was often emailed by members of the public who sought information or voiced their opposition to topics related to the pandemic, vaccinations, masking and lockdown efforts.

33. In the Statement of Claim, Dr. Bridle pleads that he sent emails to me requesting that I debate him directly. I have no recollection of receiving these emails. I have reviewed my emails and am unable to find any such emails from Dr. Bridle.

34. Beginning in May 2021, Dr. Bridle began blind copying me, and apparently hundreds of others, on emails setting out his views on COVID-19. A copy of one such email dated May 31, 2021 is attached hereto as **Exhibit "P"**. I never responded to any of Dr. Bridle's mass emails.

35. On April 25, 2022 a paper I co-authored titled "Impact of population mixing between vaccinated and unvaccinated subpopulations on infectious disease dynamics: implications for SARS-CoV-2 transmission" was published in the Canadian Medical Association Journal. A copy of this article dated April 25, 2022 is attached hereto as **Exhibit "Q"**.

36. The following day, Dr. Bridle posted an article on the blog website he maintains: viralimmunologist.substack.com, titled "Fiction Disguised as Science to Promote Hatred", a copy of which is attached hereto as **Exhibit "R"**.

37. Following Dr. Bridle's article on his blog, I began receiving emails from individuals I did not know which all had the same subject line: "Retract Fisman et al. 2022!". These emails were also sent to the editorial board of the Canadian Medical Association Journal. A sample of these emails are attached hereto as **Exhibit "S"**.

38. While I deleted the majority of these emails as I found them upsetting, I have reviewed by inbox and still have 22 emails with that subject line.

39. On January 20, 2023, I received a notification on Twitter that I was being invited to participate in a "respectful debate" on "Covid-19 science" by Bright Light News, a self-described media organization "shining a light on the science and data of COVID-19". One of the panelists was Dr. Bridle. I did not attend the panel debate which was scheduled to occur on January 28th in Hamilton, Ontario. A copy of the invitation is attached hereto as **Exhibit "T"**.

40. It was never my intention to harass or otherwise harm Dr. Bridle. At the time, I was concerned about the public health impact of an immunologist, like Dr. Bridle, spreading misinformation on COVID-19 vaccines to the public.

41. I felt the information Dr. Bridle was sharing could result in Canadians choosing not to get vaccinated for COVID-19. As the information was, and is, in my opinion, not scientifically sound, I wanted to ensure Canadians had access to evidence and data-based information so they could make informed decisions.

42. I believed in May and June 2021 that Dr. Bridle's speaking engagements, interviews and articles, posed a risk to the public. Given my professional responsibilities as a medical doctor and an advocate for public health measures on social media, I felt a moral and professional duty to respond to provide my own views and to direct the public to evidence and data-based resources.

43. I do not know Dr. Bridle personally, have never met him, and have no personal animosity towards him. It was never my intention to cause fear, anxiety, emotional upset or to impugn the dignity of Dr. Bridle.

44. I was not involved in any conspiracy against Dr. Bridle. I did not act in agreement or co-ordinated action with any other Defendant or anyone else outside this litigation to cause injury to Dr. Bridle.

45. I do not have any knowledge of such an agreement to conspire against Dr. Bridle between any of the co-Defendants or any agreement whatsoever in relation to Dr. Bridle, nor do I have knowledge of any groups conspiring against Dr. Bridle via social media.

46. In addition, I had no involvement whatsoever in any of the prohibitions allegedly enacted by the University of Guelph against Dr. Bridle. I have an "adjunct" (unpaid) appointment at the University of Guelph for the purpose of co-supervision of University of Guelph graduate students with Dr. Amy Greer. I have no ability to influence any decision the University of Guelph may have made about Dr. Bridle's employment.

47. Since June 2021, I understand from various press releases, that Dr. Bridle has continued to participate in a number of speaking engagements and opportunities related to his stance on COVID-19 vaccinations.

48. In February 2022, Dr. Bridle supported the Truckers' Freedom Convoy in Ottawa. Along with two other doctors, Dr. Bridle invited federal senior health officials to participate in a "health discussion". A copy of the Toronto Sun's article dated February 8, 2022 is attached hereto as **Exhibit "U"**.

49. In November 2022, it was reported by Guelphtoday.com that Dr. Bridle was used as an expert in a family law case in which parents could not agree on whether to vaccinate their 11 year-old son. A copy of this article is attached hereto as **Exhibit "V"**.

50. On March 2, 2023, GuelphMercuryTribune.com reported that Dr. Bridle was a featured speaker at a Toronto event headlined by Christine Anderson, a member of the European Parliament for Alternative for Germany. A copy of the St. Catharines Standard's article dated March 2, 2023 is attached hereto as **Exhibit "W"**.

51. On December 22, 2022, the Constitutional Rights Centre Inc. published an article about Dr. Bridle and this lawsuit, a copy of which is attached hereto as **Exhibit "X"**.

Procedural History

52. On March 10, 2023, my lawyer, Jaan Lilles, sent a Request to Inspect to Rocco Galati, counsel to Dr. Bridle. A copy of the Request to Inspect dated March 10, 2023 is attached hereto as **Exhibit "Y"**.

53. On May 3, 2023, Mr. Galati responded, refusing to produce all but three documents, which were to be delivered "very shortly". I have been advised by Mr. Lilles that, to date, these documents have not been produced. A copy of Mr. Galati's letter dated May 3, 2023 is attached hereto as **Exhibit "Z"**.

54. I swear this affidavit in support of my motion to dismiss the Claim under s. 137.1 of the *Courts of Justice Act* and for no improper purpose.

SWORN BEFORE ME by Dr. David Fisman of the City of Toronto, in the Province of Ontario, on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits
(or as may be)

(Signature of deponent)

This is Exhibit "A" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Curriculum Vitae

DAVID N. FISMAN M.D., M.P.H., F.R.C.P.(C)

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A. DATE CURRICULUM VITAE REVISED:

April 4, 2023

B. BIOGRAPHICAL INFORMATION

1. Academic Background

Education:

2000	MPH, Harvard School of Public Health (Clinical Effectiveness), Boston, MA. USA
1994	MD, University of Western Ontario, London, Ontario, Canada

Postdoctoral Training:

Internships and Residencies

1996 - 1997	Senior Assistant Resident, Rhode Island Hospital, Providence
1995 - 1996	Junior Assistant Resident, Royal Victoria Hospital, Montreal
1994 - 1995	Intern in Medicine, Royal Victoria Hospital, Montreal

Clinical and Research Fellowships

1998	Fellow in Clinical Effectiveness, Harvard School of Public Health, Boston, MA
1999 – 2001	Agency for Healthcare Policy and Research Postdoctoral Fellow, Center for Risk Analysis, Harvard School of Public Health, Boston, MA
1997 - 1999	Clinical Fellow in Medicine, Infectious Diseases, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA

Licensure and Certification:

2000	Subspecialty Certification in Infectious Diseases, American Board of Internal Medicine
1998	Fellow of the Royal College of Physicians of Canada #514086 (active)
1997	Specialty Certification in Internal Medicine, American Board of Internal Medicine
1995	Licentiate of the Medical Council of Canada #79306

2. Academic Employment

Current principal appointment

2021-	Lead, Preparedness Stream, University of Toronto Institute for Pandemics
2013-present	Professor, Tenured (July 1, 2012), Dalla Lana School of Public Health, University of Toronto

Current academic appointments

2019-present	Adjunct Professor, School of Public Health, University of Waterloo
2020-present	Adjunct Professor, Department of Medicine, Western University
2016-present	Adjunct Professor, Department of Population Medicine, University of Guelph
2010-present	Full member of School of Graduate Studies, University of Toronto
2013-present	Professor, Department of Health Policy, Management and Evaluation, University of Toronto

Current hospital/public health agency appointments

2020-	Attending Physician (Locum), London Health Sciences Centre, London, Ontario, Canada
2020-	Attending Physician, Michael Garron Hospital, Toronto, Ontario, Canada

Previous academic appointments

2017-2020	Division Head, Epidemiology, Dalla Lana School of Public Health, University of Toronto
2013-2020	Professor of Medicine, Department of Medicine, Faculty of Medicine, University of Toronto
2007-2010	Associate Member of School of Graduate Studies, University of Toronto
2008-2013	Associate Professor, Tenured (July 1, 2012), Dalla Lana School of Public Health, University of Toronto
2007-2013	Associate Professor, Department of Health Policy, Management and Evaluation, University of Toronto
2009-2013	Adjunct Associate Professor of Medicine, Department of Medicine, Faculty of Medicine, University of Toronto

2005-2006	Visiting Research Scholar and Visiting Assistant Professor of Public Affairs, Center for Health and Wellbeing, Woodrow Wilson School, Princeton University
2004-2006	Assistant Professor, Department of Medicine, Division of Infectious Diseases, Drexel University College of Medicine
2004-2006	Associate Scholar, Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania
2003 - 2006	Assistant Professor, Department of Epidemiology and Biostatistics, Drexel University School of Public Health
2002 - 2003	Associate Member, Clinical Health Sciences (Health Research Methodology) Graduate Programme, McMaster University, Hamilton, Ontario
2001 – 2004	Assistant Professor (Part Time), Department of Clinical Epidemiology and Biostatistics, McMaster University, Hamilton, Ontario

Hospital/public health agency appointments

2020-	Physician, Departments of Medicine and Pediatrics (Infectious Diseases), London Health Sciences Centre, London, Ontario
2020-2022	Physician, Department of Medicine, Michael Garron Hospital, Toronto, Ontario, Canada
2021-2020	Attending Physician, Toronto Western Hospital, Toronto, Ontario, Canada
2010-2020	Assistant Physician, Department of Medicine, University Health Network, Toronto, Ontario, Canada
2010-2013	Attending Physician, Department of Medicine, North York General Hospital, Toronto, Ontario, Canada
2009-2010	Adjunct Scientist, Ontario Agency for Health Protection and Promotion, Toronto, Ontario, Canada
2008-2009	Medical Epidemiologist, Ontario Agency for Health Protection and Promotion, Toronto, Ontario, Canada
2006-2009	Scientist, Child Health Evaluative Sciences, Research Institute of the Hospital for Sick Children, Toronto, Ontario, Canada
2006-2008	Medical Epidemiologist, Ontario Central Public Health Laboratory, Toronto, Ontario, Canada
2004-2006	Attending Physician, Department of Medicine, Hahnemann Hospital, Philadelphia, PA
2002 - 2003	Attending Physician, St. Joseph's Healthcare, TB Clinic, Hamilton, Ontario, Canada
2002 - 2003	Medical Advisor, Phoenix Association (Herpes Support Group), Toronto, Ontario, Canada
2001 - 2003	Associate Medical Officer of Health, City of Hamilton Department of Social and Public Health Services, Hamilton, Ontario, Canada
2001 - 2003	Medical Director for Sexually Transmitted Diseases and Information and Sexual Health Services, City of Hamilton Department of Social and Public Health Services, Hamilton, Ontario, Canada

2001 - 2003	Associate Staff Physician and Director, Hamilton General Hospital Sexually Transmitted Diseases Clinic, Hamilton Health Sciences, Hamilton, Ontario, Canada
2000 - 2001	Staff Physician, Department of Medicine, Division of Infectious Diseases, Beth Israel Deaconess Medical Center, Boston, MA
1998 - 2000	Assistant in Medicine, McLean Hospital, Belmont, MA
1997 - 2000	Fellow, Division of Infectious Diseases, Beth Israel Deaconess Medical Center, Boston, MA

3. Awards and honours

2022	Laureate Award, Ontario Chapter, American College of Physicians
2022	Citizenship Award, University of Toronto Faculty Association
2020	Maclean's Magazine "Power List"
2019	John Hastings Award for Excellence in Service to the University and the Community. Dalla Lana School of Public Health, June 2019.
2009	Schwartz-Reisman Hospital for Sick Children Canada-Israel Scholars Program ($8000) [declined].
2005	Drexel University School of Public Health Class of 2005 "Golden Apple" Award for Excellence in Teaching
2003	International Herpes Management Forum Elion Young Investigator Award
2003	Outstanding Teacher, McMaster University International Medical Graduate Program
2001	Distinguished Reviewer, Journal of General Internal Medicine
1997	Winner, Associates Vignette Poster Competition, American College of Physicians 78th Annual Session, Philadelphia
1996	Merck-Frosst Resident Research Award, Royal Victoria Hospital
1994	Medical Honor Society, University of Western Ontario
1993	First Prize, Alpha Omega Alpha Medical Student Essay Competition
1992	Alexander Hotson Memorial Scholarship, University of Western Ontario
1988	CFPL-TV Scholarship, University of Western Ontario

4. Professional Affiliations and Activities

Professional Associations

2021-	Fellow, Canadian Academy of Health Sciences
2005-	Fellow, College of Physicians of Philadelphia
2008-2020	International Society for Infectious Diseases
2002- 2021	Canadian Infectious Disease Society—Association of Medical Microbiology and Infectious Disease (AMMI) Canada
1997-2006	Infectious Disease Society of America

2005-2007	Society for Epidemiologic Research
2006-2009	American Society for Microbiology
2002-2011	Society for Hospital Epidemiology of America
2004-2006	Pennsylvania Public Health Association
2001-2013	Society for Medical Decision Making
2001-2003	Association of Local Public Health Agencies (alPHa)
1998-2003	Massachusetts Medical Society
1997 - 1998	Society of General Internal Medicine
1994 - 1997	Associate, American College of Physicians
1994 - present	Member, Canadian Medical Association

Journal Peer Review

2018-2022	Statistical Consultant, Annals of Internal Medicine
2017-	Editorial Board Member, Infectious Disease Epidemiology
2015-2017	Editorial Board Member, Nature Scientific Reports
2016-	Lancet Public Health (Elsevier)
2014-	New England Journal of Medicine, Massachusetts Medical Society
2011	PLoS ONE, Public Library of Science
2010	American Society for Microbiology mBIO
2009-	American Journal of Epidemiology, (Oxford University Press)
2009-2012	Section Co-editor (with Dr. Kevin Laupland), Adult Infectious Disease Notes, Canadian Journal of Medical Microbiology and Infectious Diseases.
2008-	PLoS Medicine, Public Library of Science
2008-	Canadian Journal of Infectious Diseases and Medical Microbiology, Association of Medical Microbiology and Infectious Diseases Canada
2008-	Epidemiology and Infection, Cambridge University Press
2008-	JAMA, American Medical Association
2007-	Journal of Infection, Elsevier
2007-	Archives of Internal Medicine, American Medical Association
2007-	International Journal of Public Health, Birkhäuser Basel
2006-	Canadian Medical Association Journal, Canadian Medical Association
2006	Environment and Development Economics, Beijer Institute of Ecological Economics, Royal Swedish Academy of Sciences/Cambridge University Press.
2006-	BMC Infectious Diseases. BioMed Central.

2006-	The Lancet, Elsevier Ltd.
2005-	Infection Control and Hospital Epidemiology, Society for Hospital Epidemiology of America.
2005	Occupational and Environmental Medicine, Faculty of Occupational Medicine of the Royal College of Physicians of London (BMJ).
2005	Lancet Infectious Diseases, Elsevier, Inc.
2005-	British Medical Journal (BMJ), British Medical Association
2005-	Archives of Pediatrics and Adolescent Medicine, American Medical Association
2005-	Tropical Medicine and International Health, Belgian Society of Tropical Medicine
2004-	Sexually Transmitted Diseases, American Sexually Transmitted Diseases Association.
2004-	Pharmacoepidemiology and Drug Safety, Wiley Interscience
2004-	Sexually Transmitted Infections, British Association of Sexual Health and HIV
2003-	Annals of Internal Medicine, American College of Physicians
2003-	Vaccine, Elsevier Science
2002 -	American Journal of Epidemiology, Society for Epidemiologic Research
2002 -	American Journal of Infection Control, Association for Professionals in Infection Control and Epidemiology, Inc.
2002 -	Society for Medical Decision Making Journal for the Society of Medical Decision Making
2001 -	Clinical Infectious Diseases, Infectious Diseases Society of America
2001 -	Emerging Infectious Diseases, U.S. Centers for Disease Control and Prevention
2001 -	Medical Care, American Public Health Association
2001	Haematologica, Ferrata Storti Foundation, Pisa, Italy
2000	Journal of General Internal Medicine, Society of General Internal Medicine

External Peer Review

2019-2020	Chair, Public Health 1 Study Section, CIHR Institute of Health
2019	Canada Research Chairs Program
2017-2023	Canadian Institutes of Health Research College of Reviewers and Member, PH1 Study Section
2015-	Reviewer, CIHR Foundation Grants Competition
2015-	Reviewer, Canadian Immunization Research Network Grants Competition
2011-	Member, Institute of Population and Public Health Study Section (PH1), Canadian Institutes of Health Research

2011	External Reviewer, Fonds de Recherches en Sante de Quebec (FRSQ) Program on Cancer and the Environment (GRPeC)
2011	External Reviewer, Canada Research Chairs Program
2011	Scientific Review Committee, Association for Medical Microbiology and Infectious Disease—Canada (AMMI-Canada) Annual Meeting
2011	Scientific Review Committee, International Society for Sexually Transmitted Disease Research Biannual Meeting
2010-	External Reviewer, Public Health Agency of Canada Field Epidemiology Training Program
2010	Reviewer, Cancer Care Ontario Position Paper on Epidemiology of Cancer and Infectious Diseases
2009	National Science and Engineering Research Council of Canada, Discovery Grants Program. Reviewer
2008	Physician's Services Incorporated Foundation Grant Program
2007-2008	Review Committee for Operating Grant: Pandemic Influenza Diagnostics, Canadian Institutes for Health Research
2007	2008 U.S. National STD Prevention Meeting (Centers for Disease Control and Prevention)
2007	External Reviewer, Washington University (St. Louis) Diabetes Research Training Center Pilot and Feasibility grants program
2007-	Canadian Institutes for Health Research
2006-	Bulletin of Mathematical Biology, Society for Mathematical Biology
2004	2004 Canadian National Sexually Transmitted Disease Guidelines, Health Canada
2004-	Proceedings of the National Academy of Sciences, National Academy of Sciences
2003	MITACS (Canadian Applied Mathematics Consortium)
2002 & 2006	Scientific Review Committee, Annual Meetings of the Society for Medical Decision Making.
2001 -	Value in Health, International Society for Pharmacoeconomics and Outcomes Research
2001	Mayo Clinic Proceedings for Mayo Clinic Rochester, Rochester, MI

Professional Service

2023-	Member, Bioaerosols Advisory Group, Canadian Association of PPE Manufacturers (unpaid)
2022-	Member, Program Committee, Canadian Academy of Health Sciences
2021-2022	Advisor to Hon. Patty Hajdu (Federal Minister of Health), on COVID-19
2020-2021	External Advisor on COVID-19, Government of Korea

2020-	Public Health Agency of Canada External Expert Advisory Group on COVID-19 Modeling
2021-	National Advisory Committee on Immunization (Canada) Expert Working Group on Pneumococcal Vaccination
2020-2022	Ontario COVID-19 Science Table Member
2020-2022	Ontario COVID-19 Modeling Table Member
2020-2021	Expert Witness, Ontario Nurses Association (*prepared a report and underwent cross-examination relative to ONA challenge to Ontario CMOH directive 5).*
2020-2021	Expert Witness, Elementary Teachers Federation of Ontario (*prepared a report on ventilation and mask use in schools as it relates to SARS transmission risk).*
2018-2022	Expert Witness, EcoJustice (*prepared a report on climate change and infectious disease risk).*
2018-2022	Organizing Committee and Session Chair, European Conference on Clinical Microbiology and Infectious Diseases
2021-	Program Committee Member, Canadian Academy of Health Sciences
2020-	Expert Advisory Group to the Federal Minister of Health on COVID-19
2020-2021	Member, Ontario COVID-19 Modeling Table
2020-2021	Member, Ontario COVID-19 Science Table
2020-	Member, Public Health Agency Ad Hoc National Modeling Group on COVID-19
2019-2021	Member, Clinical Research Network, AMMI Canada
2018-	Member, Canadian Immunization Research Network, Training and Education Committee
2018-2021	Planning Committee, European Society for Clinical Microbiology and Infectious Diseases Annual Meeting
2016	World Health Organization Guideline Working Group on Use of Mathematical Models, Geneva, Switzerland, April 27-29, 2016.
2016, 2018	Scientific Committee, International Meeting on Emerging Diseases, Vienna, Austria, November 4-7, 2016.
2015	Member, Toronto Public Health Advisory Group on Climate Change
2013-2016	Member, Ontario Chief Medical Officer's Annual Report Advisory Group. (Provided input and editorial assistance on annual reports tabled in the Ontario provincial legislature).

2015	Organizing Committee, 3rd Digital Disease Detection Conference, Florence, Italy, May 21-22, 2015.
2013-	Editorial Board, ID Cases, Elsevier
2011-2017	Editorial Board, Nature Scientific Reports
2011	Organizer and Chair, Plenary Session on Climate Change and Infectious Diseases, International Meeting on Emerging Diseases, Vienna, Austria, February 4-7, 2011.
2011	International Society for Pharmacoeconomics and Outcomes Research—Society for Medical Decision Making. Expert Panel on Health Economic Evaluation of Communicable Disease Control Programs. Chair: Richard Pitman, Oxford Outcomes.
2010-	Honorary Advisory Board, One Health Initiative (http://www.onehealthinitiative.com)
2010	Organizing Committee (Chair Susan Lett), Canadian Pandemic Influenza Planning Meeting: Assumptions. Public Health Agency of Canada, Winnipeg, Manitoba, February 2-3, 2011.
2010	Organizing Committee (with Drs. Jan Sargeant, Zvonimir Poljak, Amy Greer, Javier Sanchez, and Bruce McNab), "One Health One Model: Modeling at the Animal-Human Interface". 4 day meeting on applying mathematical modeling to the "One Health" paradigm. University of Guelph, November 1-4, 2010.
2010	Organizing Committee, International Society for Infectious Diseases International Meeting on Emerging Diseases (IMED, Vienna, Austria, February 2011)
2010	Co-organizer (with Profs. Jianhong Wu and Troy Day), Fields Institute Thematic Program in the Mathematics of Antimicrobial Resistance, Toronto, Ontario, Canada, July-August 2011
2010	Co-organizer (with Profs. David Earn and Jonathan Dushoff), Banff International Research Station Meeting on Persistent Infectious Diseases (Banff, AB, February 2011)
2009	Co-organizer (with Dr. Emery Leger, Canadian Food Inspection Agency; Dr. Javier Sanchez, University of Prince Edward Island; and Dr. Babak Pourbohloul, British Columbia Centre for Disease Control), Canadian Food Inspection Agency Meeting on Animal-Human Modeling of Influenza. Montreal, Quebec, Canada, November 18-19, 2009.
2009-2011	Member, Society for Hospital Epidemiology of America External Affairs Committee (Ms. Barbara Soule, Chair).
2009	Organizer, Signal Detection 2009: An International Conference on Modeling and Surveillance. Ontario Agency for Health Protection and Promotion, Toronto, Ontario, Canada. October 8-9, 2009.
2009	Organizing Committee, Mitigating the spread of influenza A (H1N1) (Part II): An International Mathematical Modelling Meeting. British Columbia Centre for Disease Control (BCCDC), Vancouver, BC, Canada, September 14 – 16, 2009.
2009	Ontario Agency for Health Protection and Promotion Medical Officers of Health "Scientific Webinar" on Mathematical Modeling and Influenza, May 6, 2009.

2009	Organizer and Co-host (with Ontario Emergency Management Unit): Mathematical Modeling and Pandemic Influenza Control. Sutton Place Hotel, January 30-31, 2009.
2008	Organizer and Host, Ontario Agency for Health Protection and Promotion --- University of Guelph Center for Public Health and Zoonosis meeting on collaborative efforts in human-veterinary health research, Ontario Central Public Health Laboratory, November 26, 2008.
2007-2009	Ontario Vaccine Evaluation Center Planning Committee (Dr. Craig Laferriere, Chair)
2007	Co-organizer, Public Health Agency of Canada—MITACS Joint Symposium on Modeling Sexually Transmitted and Blood-Borne Infections (with Dr. Jianhong Wu, York University and Dr. Tom Wong, PHAC). Banff International Research Station for Mathematical Innovation and Discovery, Banff, Alberta, Canada, August 10-12, 2007.
2007	Introduction to Decision Analysis, Cost-Effectiveness Analysis, and Dynamic Transmission Modeling. Merck Frosst Health Sciences Associates Mentorship Program. Montreal, PQ, September 26, 2007.
2007-2008	Program Committee, U.S. Centers for Disease Control National STD Prevention Meeting (Chicago, IL, May 10-13 2008).
2006-2007	CDC Expert Panel on Chlamydia Screening in Males (Chair, Dr. Tom Gift, CDC).
2006	Ontario Ministry of Health and Long-term Care Research Paper Editorial Board (Healthy and Responsible Consumers). (document available via the Internet at http://www.ourplanforhealth.ca/moh/research/Healthy_and_Responsible_Consumers.pdf).
2006-2009	Ontario Public Health Laboratory Research Ethics Board Member (Dr. Steve Drews, Chair)
2006-2008	Clinician-Scientist Training Program Committee, Hospital for Sick Children Research Institute (Dr. Neil Sweezy, Chair)
2005	New Jersey State Department of Public Health Task Force on Antimicrobial Resistance (New Jersey CAUSE) (Dr. Corey Robertson, Chair)
2005	Society for Hospital Epidemiology of America (SHEA), Working Group on Management of Invasive Group A Streptococcal Infections in Long-Term Care (Chair, Dr. Andrew Simor, University of Toronto).
2003-2004	Adjunct Member, Hahnemann Hospital SARS Planning Committee
2004	Society for Hospital Epidemiology of America, Working Group on Economic Evaluation in Infection Control (Chair, Dr. Eli Perencevich, University of Maryland).
2003	Ontario SARS Science Committee, March 30-April 16, 2003.
2002-2004	Canadian Infectious Disease Society, STD/HIV Committee
2002	McMaster University Community Medicine Residency Training Committee
2002 - 2003	Hamilton Regional Microbiology Committee
2002 - 2003	Emergency Preparedness Group, City of Hamilton, Hamilton, Ontario
2002	Ontario Ministry of Health, Health Canada Advisory Group on Smallpox Vaccine, Toronto, October 11, 2002.

2001 - 2003	Hamilton Regional Infection Control Committee, Hamilton, Ontario (Dr. Maureen Cividino, Chair)
2001 - 2003	Hamilton City Nuclear/Biological/Chemical Planning Committee, Hamilton, Ontario
2001 - 2003	Hamilton Health Sciences Center Nuclear/Biological/Chemical Planning Committee, Hamilton, Ontario
2001 - 2003	Consultant, Ontario Public Health Research, Education, Development (PHRED) Program, City of Hamilton Department of Social and Public Health Services, Hamilton, Ontario
1994 - 1996	Residency Training Committee, Royal Victoria Hospital, Montreal, Quebec (Dr. Sam Benaroya, Chair)

D. RESEARCH GRANTS AND CONTRACTS

(Principal investigator(s) underlined)

Principal investigator

2020-2022 **Fisman DN (PI)** Understanding, forecasting, and communicating risk during the COVID-19 epidemic. Canadian Institutes of Health Research. Canadian 2019 Novel Coronavirus (2019-nCoV) Rapid Research. OVA-170360. Collaborators: Lee N, Mazzola E, McGeer A. $331,700 CDN. *This project aims to use mathematical and statistical modelling to characterize COVID-19 pandemic transmission dynamics in the Canadian population and to develop tools to communicate modelling results and forecasts to the general public.*

2018-2020 **Fisman DN (PI),** Greer AL, Ellen M, Lofmark S, Hulth A, Daneman N. An Online Platform for Expanding Antibiotic Stewardship: OPEN Stewardship. Joint International Program on Antimicrobial Resistance/CIHR. 460,564 to Canadian site. $800,000 total.

2015-2018 **Fisman DN (PI),** Greer AL (co-PI)**.** One Health In Action: Linking Human and Animal Data Sources to Understand and Prevent Enteric Disease in Ontario (#343143). CIHR Operating Grant ($100,000)

2015-2019 **Fisman DN (PI)**, Tuite AR, Hatchette T, Drews S, Gubbay J**.** Seasonal Influenza Forecasting in Real Time using the IDEA Model. Canadian Immunization Research Network. ($37,076).

Fisman DN (PI). Cost Effectiveness of Decennial Booster Dosing of Acellular Pertussis Vaccine: A Dynamic Modeling Approach. Canadian Immunization Research Network. ($38,021).

2013 **Fisman DN (PI).** FitzGerald Seminar Series in Communicable Disease Epidemiology (unrestricted educational grant). Novartis Pharma Canada Inc., Merck Canada Inc. and GlaxoSmith Kline Inc. ($40,000)**.**

2012 **Fisman DN (PI).** FitzGerald Seminar Series in Communicable Disease Epidemiology (unrestricted educational grant). Novartis Pharma Canada Inc. ($31,000)**.**

2011 **Fisman DN (PI).** FitzGerald Seminar Series in Communicable Disease Epidemiology (unrestricted educational grant). Novartis Pharma Canada Inc. ($38,400)**.**

2011-2014 **Fisman DN,** Allen VG, Gesink D, Garay JR, Greer A. *Untangling the web: understanding the abrupt increase in chlamydia risk in Ontario through applied epidemiology and mathematical modeling.* Canadian Institutes of Health Research Operating Grant. ($247, 423).

2010-2012 **Fisman DN**, Kwong J, McGeer A, Drews S, Pourbohloul B, Buckeridge D. *Wintertime Seasonality of Influenza and Invasive Bacterial Disease: Influence of Environment, Pathogen Interactions, Time Scales, and Geography*. Canadian Institutes for Health Research Institute of Infection and Immunity and Population and Public Health. ($227,612).

2009 **Fisman DN**, Wu J, Crowcroft N, Moore K. *Signal Detection 2009* (conference at OAHPP on linkage between public health surveillance and mathematical modeling). Mathematics of Information Technology and Complex Systems (MITACS) grant. ($7,500).

2007-2010 **Fisman DN**. *Keeping Vulnerable Children Safe from Pertussis: Cost-Effective Strategies for Ontario Hospitals as Whooping Cough Returns.*Ontario Ministry of Innovation Early Researcher Award. ($150,000).

2006-2008 **Fisman DN**, Johnson C. *Seasonality, environment, and infectious disease occurrence.* National Institute of Allergy and Infectious Diseases (R21AI065826-01A1). ($200,000).

2002-2003 **Fisman DN**, Cividino M, Harris AD, Mittleman MA. *Case-crossover study of sharps related injuries*. City of Hamilton, Social and Public Health Services Department, Public Health Research, Education and Development Program, Hamilton, Ontario. ($11,400).

2002 **Fisman DN**, Sheehan D. *Assessment of health-related quality of life in individuals with symptomatic and asymptomatic genital herpes infection.* City of Hamilton, Social and Public Health Services Department, Public Health Research, Education and Development Program, Hamilton, Ontario. ($6,000).

1999-2001 **Fisman DN**. Agency for Healthcare Research and Quality. National Research Service Award #5-T32-HS00020-15. ($50,000).

1995 **Fisman DN**, Tamblyn R. *Survival after percutaneous endoscopic gastrostomy in the elderly.* Department of Medicine, Royal Victoria Hospital, Montreal, Quebec. ($500).

1992 **Fisman DN**. *Medicine, Fraud, and Puritanism in 17th century England*. Osler Studentship in the History of Medicine. McGill University, Montreal, Quebec.($5,000).

1990 **Fisman DN**, LaChance M. *Taxonomic evaluation of yeasts through protein gel-electrophoresis.* Natural Science and Engineering Research Council Scholarship. Western University, London, Ontario. ($5,000).

Co-investigator

2020-2021 **Co-Investigator**. Population-based seroprevalence of prior infection with COVID-19 in Canada: implications for testing, economic revitalization and population health. Principal Investigator: Drews S. Co-Investigators: Evans D, Fisman D, Gingras A, Hobman T, O'Brien S. $1,055,700 CDN. *This project will use banked samples from blood donors across Canada to measure seroprevalence of COVID-19 infection over time and geography to better understand population immunity.*

2020-2021 Co-Investigator. Surveillance and Modeling of COVID-19 infection.
PI: Ashleigh Tuite. Health Canada, $200,000.

2013-2017 Burchell A, Allen V, Tan D, Cooper C, Fisman D, Gardner S, Gough K, MacPherson P, Raboud J, Rachlis A, Remis RS, Rourke SB, Walmsley S. Enhanced syphilis screening among HIV-positive men who have sex with men: Evaluation of a clinic-based intervention. Canadian Institutes for Health Research (#300246). ($411,244).

2011-2014 Tien J, **Fisman DN**, Eisenberg M. Modeling the effects of heterogeneity in water quality on cholera disease dynamics. National Science Foundation (US) ($978,123).

2010-2013 Wu J, **Fisman DN**, Moghadas S, Sahai B, Dean C, Brauer F, Webb G, Zhu H, Belair J, Watmmough J, Heffernan J, Khan K, Arino J, Wang L, Rioux M, Gardam M, Li M, Madras N, Yan P, van den Driessche P, Ruan S, Day T, Jacobson Z. York-MITACS Centre for Disease Modeling. *Transmission Dynamics and Spatial Spread of Infectious Diseases: Modelling, Prediction and Control.* Mathematics, Information Technology and Complex Systems Nation al Centre of Excellence. ($198,000).

2010-2012 Mishra S, **Fisman DN**. *Assessing the Impact of Undiagnosed Syphilis on the Transmission of Syphilis and HIV in Ontario: Epidemiological evaluation of co-infection and development of a disease transmission model.* Canadian Institutes for Health Research Public Health Fellowship. ($100,000), (deferred).

2009-2010 Pourbohloul B, **Fisman DN**, Buckeridge D, Arino J, Dushoff J, Earn DJD, Moghadas S, Wu J. *Pan-Canadian Decision-Making Support Network for Pandemic Preparedness. ("CanPan").* Emergency Supplementary Funding, Canadian Institutes for Health Research Catalyst Grant (Pandemic Preparedness). ($700,000).

2009 Wu J, **Fisman D**, Moghadas S. MITACS *Accelerate Internship in Mathematical Modeling of Infectious Diseases* ($45,000 with $45,000 match from Ontario Agency for Health Protection and Promotion). ($90,000).

2008-2009 Pourbohloul B, Bauch C, Beauchemin C, Brauer F, Buckeridge D, Dean CB, Dushoff J, Earn DJD, **Fisman DN**, Khan K, McGeer AJ, Tellier R, Moghadas S, Wu J. *Pan-Canadian Decision-Making Support Network for Pandemic Preparedness ("CanPan").* Canadian Institutes for Health Research Catalyst Grant (Pandemic Preparedness). ($100,000).

2008-2009 Moghadas S, Wu J, Pizzi N, **Fisman DN**, Yan P, Driedger M, Roos L, Alexander M. *Evaluation of Mitigation Strategies for Pandemic Preparedness in Canada.* Canadian Institutes for Health Research Catalyst Grant (Pandemic Preparedness). ($94,750).

2007-2009 To T, Stanbrooke M, Crichton E, Guttman A, **Fisman DN**, Wang C. *Respiratory population-based outcomes network: Studies and evaluations (RESPONSE).* Canadian Institutes of Health Research (CIHR) Partnerships for Health System Improvement (PHSI). ($87,715).

2003-2004 Abrutyn E, Kirchner C, **Fisman DN**, Kim Y, Dhond AJ. *Center for study of hospital acquired infections.* Tenet Healthcare Foundation, Dallas TX (GFW 11595). ($1,004,000 US).

2002-2006 Mittleman MA, **Fisman DN**, Harris AD, Sorock G. *A case-crossover study of sharps-related injuries.* Centers for Disease Control and Prevention (CDC). National Institute for Occupational Safety and Health, Atlanta, GA. ($1,076,531 US).

2002 Redwood-Campbell L, Kaczorowski J, **Fisman DN**. *Improving pap smear screening in immigrant women.* City of Hamilton, Social and Public Health Services Department, Public Health Research, Education and Development Program. ($14,000).

2002 Gardam M, Tsang L, Petrich A, Jamieson F, **Fisman DN**. *Molecular epidemiology of tuberculosis in the Greater Toronto Area 1999-2001.* National Sanatorium Foundation. 2002.

2000-2001 Mittleman MA, **Fisman DN**, Sorock G, Harris AD. *Case-crossover study of sharps-related injuries in healthcare workers.* Harvard-Liberty Department of Occupational and Environmental Health. Harvard School of Public Health. ($100,000 US)

Supervisor

2015-2016 Tahmina Nasserie, Canadian Immunization Research Network Studentship ($10,000). Using IDEA to forecast seasonal influenza.

2015-2016 Ashleigh McGirr, Canadian Immunization Research Network Studentship ($25,000). Agent based model of pertussis transmission in Ontario.

2014-2016 Dr. Derek MacFadden, CIHR Doctoral Award ($275,000). ResistanceOpen: Developing a Global Map of Regional Antimicrobial Resistance.

2012-2015 Ashleigh McGirr, CIHR Doctoral Award (Banting and Best), ($105,000). Evaluating vaccination strategies to contain the spread of pertussis in Canada.

2012-2015 Ashleigh Tuite, CIHR Doctoral Award (Banting and Best), ($70,000). Evaluating vaccination strategies to contain the spread of pertussis in Canada.

2010-2013 Kevin A. Brown, CIHR Doctoral Award (Banting and Best), ($105,000). Developing a clinical prediction rule for hospital-acquired Clostridium difficile infection to enable inter-institution comparisons of incidence rates and promote quality improvement.

Research Contracts

2012-2013 **Fisman DN**, Tuite AR. Toronto Unvaccinated: Estimating the Impact of Vaccination on Toronto's Health. Toronto Public Health, ($25,000).

2011-2012 **Fisman DN**, Tuite AR. *Mathematical modeling of novel partner notification strategies for communicable disease control.* National Collaborating Centre for Communicable Diseases, ($25,000).

2011-2012 **Fisman DN**, Mishra S, Tuite AR. *Mathematical modeling of syphilis/HIV testing strategies in Ontario*. Ontario AIDS Bureau/Public Health Agency of Canada/Hassle Free Clinic (Toronto), ($25,000).

2011 **Fisman DN**, Tuite AR. *Estimation of the health and economic burden of Chlamydia trachomatis infection in Canada.* Public Health Agency of Canada, ($10,000).

2011 **Fisman DN**. *Health economic evaluation of rotavirus vaccine in Canada*. Public Health Agency of Canada, ($6,000).

2010-2011 **Fisman DN,** Tuite AR. *Mathematical modeling of the impact of an adjuvanted influenza vaccine*. Novartis Vaccines Canada. ($35,000).

2010 **Fisman DN**, Tuite AR. *Mathematical modeling of pertussis under-reporting in Ontario.* GlaxoSmithKline Canada. ($50,000).

2009-2010 **Fisman DN**, Greer A. *Mathematical modeling of optimal control strategies for Chlamydia trachomatis in Canada*. Public Health Agency of Canada. ($25,000).

Career summary of research funding

SUMMARY OF RESEARCH FUNDING (PEER-REVIEWED GRANTS AND CONTRACTS)

	Past	Current	Career Total
Total Grants as Principal Investigator	$732,412	$287,423	$1,019,835
Total Grants as Co-Investigator	$3,366,996	$1,281,123	$4,648,119
Total Grants	$3,840,796	$1,787,158	$5,667,954
Total Contracts	$176,000	$25,000	$201,000
Total Grants and Contracts	**$4,016,796**	**$1,812,158**	**$5,868,954**

ALL GRANTS – PRINCIPAL INVESTIGATOR

Funder	Years	Role	Team	Amount	Title
Novartis Pharma Canada Inc.	01.2013-12.2013	Nominated PI	Vasilevska, M	$40,000	*FitzGerald Seminar Series in Communicable Disease Epidemiology (Knowledge Translation Activity)*
Novartis Pharma Canada Inc.	01.2012-12.2012	Nominated PI	Vasilevska, M	$31,000	*FitzGerald Seminar Series in Communicable Disease Epidemiology (Knowledge Translation Activity)*
Novartis Pharma Canada Inc.	07. 2011-12. 2011	Nominated PI	Vasilevska, M	$38,400	*FitzGerald Seminar Series in Communicable Disease Epidemiology (Knowledge*

Funder	Years	Role	Team	Amount	Title
					Translation Activity)
CIHR, Operating Grant	10.2011-09.2014	Nominated PI	Allen AG, Gesink D, Garay JR, Greer A.	$247, 423	*Untangling the web: understanding the abrupt increase in chlamydia risk in Ontario through applied epidemiology and mathematical modeling.*
CIHR, Operating Grant	10.2010-09.2012	Nominated PI	Kwong J, McGeer A, Drews S, Pourbohloul B, Buckeridge D.	$227,612	*Wintertime Seasonality of Influenza and Invasive Bacterial Disease: Influence of Environment, Pathogen Interactions, Time Scales, and Geography*
MITACS – Mathematics of Information Technology and Complex Systems	2009	Nominated PI	Wu J, Crowcroft N, Moore K.	$7,500	*Signal Detection 2009* (conference at OAHPP on linkage between public health surveillance and mathematical modeling).
Ontario Ministry of Innovation Early Researcher Award.	10.2008-09.2010	Nominated PI	N/A	$150,000	*Keeping Vulnerable Children Safe from Pertussis: Cost-Effective Strategies for Ontario Hospitals as Whooping Cough Returns.*
National Institute for Allergy and Infectious Diseases (R21AI065826-01A1).	09.2006-08.2008	PI	Johnson C.	$200,000	*Seasonality, environment, and infectious disease occurrence*

Funder	Years	Role	Team	Amount	Title
City of Hamilton, Social and Public Health Services Department, Public Health Research, Education and Development Program, Hamilton, Ontario	2002-2003	Nominated PI	Cividino M, Harris AD, Mittleman MA.	$11,400	*Case-crossover study of sharps related injuries*
City of Hamilton, Social and Public Health Services Department, Public Health Research, Education and Development Program, Hamilton, Ontario	2002	Nominated PI	Sheehan D.	$6,000	*Assessment of health-related quality of life in individuals with symptomatic and asymptomatic genital herpes infection*
Agency for Healthcare Research and Quality	1999-2001	Nominated PI	N/A	$50,000	National Research Service Award #5-T32-HS00020-15
Department of Medicine, Royal Victoria Hospital, Montreal, Quebec	1995	Nominated PI	Tamblyn R	$500	*Survival after percutaneous endoscopic gastrostomy in the elderly*
Osler Studentship in the History of Medicine. McGill University, Montreal, Quebec	1992	Nominated PI	N/A	$5,000	*Medicine, Fraud, and Puritanism in 17th century England*

Funder	Years	Role	Team	Amount	Title
Natural Science and Engineering Research Council Scholarship. Western University, London, Ontario	1990	Nominated PI	LaChance M.	$5,000	*Taxonomic evaluation of yeasts through protein gel-electrophoresis*
				$1,019,835 AWARDED	

ALL GRANTS – CO-INVESTIGATOR

Funder	Years	Role	Team	Amount	Title
National Science Foundation (US)	08.2011-07-2014	CI	Tien J (PI), Fisman DN, Eisenberg M.	$978,123	*Modeling the Effects of Heterogeneity in Water Quality on Cholera Disease Dynamics*
MITACS Centre for Disease Modeling	04.2010-03.2013	CI	Wu J (PI), Moghadas S, Sahai B, Dean C, Brauer F, Webb G, Zhu H, Belair J, Watmmough J, Heffernan J, Khan K, Arino J, Wang L, Rioux M, Gardam M, Li M, Madras N, Yan P, van den Driessche P, Ruan S, Day T, Jacobson Z.	$198,000	*Transmission Dynamics and Spatial Spread of Infectious Diseases: Modelling, Prediction and Control*
CIHR Doctoral Award—Banting and Best	09.2010-08-2013	Supervisor	Daneman, N Brown, K	$105,000	*Epidemiology of Clostridium difficile in Canada*
CIHR Fellowship-Health Professionals	06.2010-06.2012	Supervisor	Mishra S.	$100,000	*Assessing the Impact of Undiagnosed Syphilis on the Transmission of Syphilis and HIV in Ontario:*

Funder	Years	Role	Team	Amount	Title
					Epidemiological evaluation of co-infection and development of a disease transmission model.
CIHR Catalyst Grant Emergency Supplementary Funding, Pandemic Preparedness	10.2009-09.2010	CI	Pourbohloul B, (PI) Buckeridge D, Arino J, Dushoff J, Earn DJD, Moghadas S, Wu J.	$700,000	*Pan-Canadian Decision-Making Support Network for Pandemic Preparedness.* *"CanPan"*
MITACS ($45,000 with $45,000 match from Ontario Agency for Health Protection and Promotion).	2009	CI	Wu J, Moghadas S.	$90,000	*Accelerate Internship in Mathematical Modeling of Infectious Diseases*
CIHR Catalyst Grant Emergency Supplementary Funding, Pandemic Preparedness	10.2008-09.2009	CI	Pourbohloul B (PI), Bauch C, Beauchemin C, Brauer F, Buckeridge D, Dean CB, Dushoff J, Earn DJD, Khan K, McGeer AJ, Tellier R, Moghadas S, Wu J.	$100,000	*Pan-Canadian Decision-Making Support Network for Pandemic Preparedness "CanPan"*

Funder	Years	Role	Team	Amount	Title
CIHR Catalyst Grant Pandemic Preparedness	10.2008-09.2009	CI	Moghadas S (PI), Wu J, Pizzi N, Yan P, Driedger M, Roos L, Alexander M.	$94,750	*Evaluation of Mitigation Strategies for Pandemic Preparedness in Canada*
CIHR Partnerships for Health System Improvement (PHSI)	08.2007-07.2009	CI	To T (PI), Stanbrooke M, Crichton E, Guttman A, Wang C.	$87,715	*Respiratory population-based outcomes network: Studies and evaluations (RESPONSE)*
Tenet Healthcare Foundation, Dallas TX (GFW 11595).	2003-2004	CI	Abrutyn E, Kirchner C, Kim Y, Dhond AJ.	$1,004,000 ($US)	*Center for study of hospital acquired infections.*
Centers for Disease Control and Prevention (CDC). National Institute for Occupational Safety and Health, Atlanta, GA.	2002-2006	CI	Mittleman MA, Harris AD, Sorock G.	$1, 076,531 ($US)	*A case-crossover study of sharps-related injuries.*
City of Hamilton, Social and Public Health Services Department, Public Health Research, Education and Development Program	2002	CI	Redwood-Campbell L, Kaczorowski J	$14,000	*Improving pap smear screening in immigrant women*
National Sanatorium Foundation	2002	CI	Gardam M, Tsang L, Petrich A, Jamieson F	---	*Molecular epidemiology of tuberculosis in the Greater Toronto Area 1999-2001*
Harvard-Liberty Department of Occupational and Environmental	2000-2001	CI	Mittleman MA, Sorock G, Harris AD	$100,000 ($US)	*Case-crossover study of sharps-*

Funder	Years	Role	Team	Amount	Title
Health. Harvard School of Public Health					*related injuries in healthcare workers.*
				$4,648,119 AWARDED	

ALL CONTRACTS

Funder	Years	Role	Team	Amount	Title
Toronto Public Health	2012-2013	Nominated PI (for Decision Centre for Infectious Disease Epidemiology (DeCIDE)	Tuite AR, McGirr A, Hum R	$25,000	*Toronto Unvaccinated: Estimating the Impact of Vaccination on Toronto's Health*
National Collaborating Centre on Infectious Diseases (NCCID)	2011-12	Nominated PI	Tuite AR	$25,000	*Mathematical modeling of novel partner notification strategies for communicable disease control.*
Ontario AIDS Bureau and Hassle-Free Clinic (Toronto)	2011-12	Nominated PI	Tuite AR, Mishra S	$25,000	*Mathematical modeling of syphilis/HIV testing.*
Public Health Agency of Canada	2011	Nominated PI	---	$6,000	*Health economic evaluation of rotavirus vaccine in Canada.*
Public Health Agency of Canada	2011	Nominated PI	Tuite AR	$10,000	*Estimation of the health and economic burden of Chlamydia trachomatis infection in Canada.*
Novartis Vaccines Canada	2010-2011	Nominated PI	Tuite AR	$35,000	*Mathematical modeling of the impact of an adjuvanted influenza vaccine*

Funder	Years	Role	Team	Amount	Title
GlaxoSmithKline Canada	2010	Nominated PI	Tuite AR	$50,000	*Mathematical modeling of pertussis under-reporting in Ontario*
Public Health Agency of Canada	2009-2010	Nominated PI	Greer A	$25,000	*Mathematical modeling of optimal control strategies for Chlamydia trachomatis in Canada*
				$201,000 AWARDED	

Legend

CIHR: Canadian Institutes for Health Research

MITACS: Mathematics of Information Technology and Complex Systems

E. PUBLICATIONS

As of 2021 I am no longer updating my publications directly. A listing of new publications can be obtained at https://pubmed.ncbi.nlm.nih.gov/?term=%22fisman+d%22&sort=pubdate&size=100

Notes regarding authorship and contributions

I believe strongly that the experience of preparing and submitting research for publication is an invaluable component of scientific training, and is a core component of my mentorship strategy. As such, I frequently encourage students, trainees, and junior research officers, where appropriate, to serve as lead authors on publications. As such, in many of the papers below, on which I am listed as senior responsible author, I have contributed in a manner that would also have made first authorship reasonable.

Peer reviewed publications (* student/trainee)

159. Chen PZ*, Bobrovitz N*, Premji Z, Koopmans M, **Fisman DN**, Gu FX. Heterogeneity in transmissibility and shedding SARS-CoV-2 via droplets and aerosols. Elife. 2021 Apr 16;10:e65774. doi: 10.7554/eLife.65774.

158. Greenhalgh T, Jimenez JL, Prather KA, Tufekci Z, **Fisman D**, Schooley R. Ten scientific reasons in support of airborne transmission of SARS-CoV-2. Lancet. 2021 Apr 15:S0140-6736(21)00869-2. doi: 10.1016/S0140-6736(21)00869-2.

157. Maharaj AS, Parker J, Hopkins JP, Gournis E, Bogoch II, Rader B, Astley CM, Ivers N, Hawkins JB, VanStone N, Tuite AR, **Fisman DN**, Brownstein JS, Lapointe-Shaw L. The effect of seasonal respiratory virus transmission on syndromic surveillance for COVID-19 in Ontario, Canada. Lancet Infect Dis. 2021 Mar 25:S1473-3099(21)00151-1. doi: 10.1016/S1473-3099(21)00151-1.

156. Brankston G, Merkley E, **Fisman DN**, Tuite AR, Poljak Z, Loewen PJ, Greer AL. Socio-demographic disparities in knowledge, practices, and ability to comply with COVID-19 public health measures in Canada. Can J Public Health. 2021 Mar 24:1-13. doi: 10.17269/s41997-021-00501-y.

155. Fitzpatrick T, McNally JD, Stukel TA, Lu H, **Fisman D**, Kwong JC, Guttmann A. Family and Child Risk Factors for Early-Life RSV Illness. Pediatrics. 2021 Mar 18:e2020029090. doi: 10.1542/peds.2020-029090.

154. Acharya KR*, Brankston G, Soucy JR*, Cohen A, Hulth A, Löfmark S, Davidovitch N, Ellen M, **Fisman DN**, Moran-Gilad J, Steinman A, MacFadden DR, Greer AL. Evaluation of an OPEN Stewardship generated feedback intervention to improve antibiotic prescribing among primary care veterinarians in Ontario, Canada and Israel: protocol for evaluating usability and an interrupted time-series analysis. BMJ Open. 2021 Jan 15;11(1):e039760. doi: 10.1136/bmjopen-2020-039760.

153. Soucy JR*, Low M, Acharya KR, Ellen M, Hulth A, Löfmark S, Garber GE, Watson W, Moran-Gilad J, **Fisman DN**, MacFadden DR. Evaluation of an automated feedback intervention to improve antimicrobial prescribing among primary care physicians (OPEN Stewardship): protocol for an interrupted time-series and usability analysis in Ontario, Canada and Southern Israel. BMJ Open. 2021 Jan 13;11(1):e039810. doi: 10.1136/bmjopen-2020-039810.

152. Shaw J, Day T, Bing NM, Barber N, Wickenheiser H, **Fisman DN**, Bogoch I, Brownstein JI, Williamson T. Les bulles de travail : Comment les entreprises peuvent-elles rouvrir en réduisant le risque d'éclosions de la COVID-19? CMAJ. 2021 Jan 11;193(2):E80-E84. doi: 10.1503/cmaj.201582-f.

151. Tuite AR, Zhu L, **Fisman DN**, Salomon JA. Ann Intern Med. 2021 Jan 5:M20-8137. Alternative Dose Allocation Strategies to Increase Benefits From Constrained COVID-19 Vaccine Supply. doi: 10.7326/M20-8137.

150. Jüni P, Rothenbühler M, Bobos P*, Thorpe KE, da Costa BR, **Fisman DN**, Slutsky AS, Gesink D. Effets du climat et des interventions de santé publique sur la pandémie de COVID-19 : une étude de cohorte prospective. CMAJ. 2020 Nov 2;192(44):E1374-E1382. doi: 10.1503/cmaj.200920-f.

149. COVID-19 Research GloPID-R Synergies Meeting Working Group; Meeting Co-chairs. Ending COVID-19: progress and gaps in research-highlights of the July 2020 GloPID-R COVID-19 Research Synergies Meetings. BMC Med. 2020 Oct 29;18(1):342. doi: 10.1186/s12916-020-01807-3.

148. Tuite AR, **Fisman DN**, Greer AL. CMAJ. Modélisation mathématique de la transmission de la COVID-19 et stratégies d'atténuation des risques dans la population ontarienne au Canada. 2020 Oct 19;192(42):E1276-E1285. doi: 10.1503/cmaj.200476-f.

147. **Fisman DN**, Greer AL, Hillmer M, Tuite R. Derivation and Validation of Clinical Prediction Rules for COVID-19 Mortality in Ontario, Canada. Open Forum Infect Dis. 2020 Oct 5;7(11):ofaa463. doi: 10.1093/ofid/ofaa463.

146. Stall NM, Wu W, Lapointe-Shaw L, **Fisman DN**, Giannakeas V*, Hillmer MP, Rochon PA . Sex- and age-specific differences in COVID-19 testing, cases and outcomes: a population-wide study in Ontario, Canada. J Am Geriatr Soc. 2020 Jul 24. doi: 10.1111/jgs.16761. Online ahead of print.

145. **Fisman DN**, Greer AL, Tuite AR. Age Is Just a Number: A Critically Important Number for COVID-19 Case Fatality.Ann Intern Med. 2020 Jul 22:M20-4048. doi: 10.7326/M20-4048. Online ahead of print.

144. **Fisman DN**, Bogoch I, Lapointe-Shaw L, McCready J, Tuite AR. Risk Factors Associated With Mortality Among Residents With Coronavirus Disease 2019 (COVID-19) in Long-term Care Facilities in Ontario, Canada. JAMA Netw Open. 2020 Jul 1;3(7):e2015957. doi: 10.1001/jamanetworkopen.2020.15957.

143. **Fisman DN**, Greer AL, Tuite AR. Bidirectional impact of imperfect mask use on reproduction number of COVID-19: A next generation matrix approach. Infect Dis Model. 2020 Jul 4;5:405-408. doi: 10.1016/j.idm.2020.06.004. eCollection 2020.

142. Ogden NH, Fazil A, Arino J, Berthiaume P, **Fisman DN**, Greer AL, Ludwig A, Ng V, Tuite AR, Turgeon P, Waddell LA, Wu J. Modelling scenarios of the epidemic of COVID-19 in Canada. Can Commun Dis Rep. 2020 Jun 4;46(8):198-204. doi: 10.14745/ccdr.v46i06a08. eCollection 2020 Jun 4.

141. Berry I, Tuite AR, Salomon A, Drews S, Harris AD, Hatchette T, Johnson C, Kwong J, Lojo J, McGeer A, Mermel L, Ng V, **Fisman DN**. Association of Influenza Activity and Environmental Conditions With the Risk of Invasive Pneumococcal Disease. JAMA Netw Open. 2020 Jul 1;3(7):e2010167. doi: 10.1001/jamanetworkopen.2020.10167.

140. Tuite AR, Bogoch II, **Fisman D**. Estimation of Coronavirus Disease 2019 Burden and Potential for International Dissemination of Infection From Iran. Ann Intern Med. 2020 Jul 7;173(1):74-75. doi: 10.7326/L20-0593.

139. Tuite AR, Greer AL, De Keninck S, **Fisman DN**. Risk for COVID-19 Resurgence Related to Duration and Effectiveness of Physical Distancing in Ontario, Canada. Ann Intern Med. 2020 May 27:M20-2945. doi: 10.7326/M20-2945. Online ahead of print.PMID: 32459528

138. Berry I*, Soucy JR*, Tuite A, **Fisman D**; COVID-19 Canada Open Data Working Group. Open access epidemiologic data and an interactive dashboard to monitor the COVID-19 outbreak in Canada. CMAJ. 2020 Apr 14;192(15):E420. doi: 10.1503/cmaj.75262.

137. Jüni P, Rothenbühler M, Bobos P, Thorpe KE, da Costa BR, **Fisman DN**, Slutsky AS, Gesink D. Impact of climate and public health interventions on the COVID-19 pandemic: a prospective cohort study. CMAJ. 2020 May 25;192(21):E566-E573. doi: 10.1503/cmaj.200920. Epub 2020 May 8.

136. Cousins M, Sargeant JM, **Fisman DN**, Greer AL. Identifying the environmental drivers of Campylobacter infection risk in southern Ontario, Canada using a One Health approachs. Zoonoses Public Health. 2020 May 4. doi: 10.1111/zph.12715. Online ahead of print.

135. Tuite AR, **Fisman DN**, Greer AL. Mathematical modelling of COVID-19 transmission and mitigation strategies in the population of Ontario, Canada. CMAJ. 2020 May 11;192(19):E497-E505. doi: 10.1503/cmaj.200476. Epub 2020 Apr 8.

134. Tuite AR, Ng V, Rees E, **Fisman D**, Wilder-Smith A, Khan K, Bogoch II. Estimation of the COVID-19 burden in Egypt through exported case detection. Lancet Infect Dis. 2020 Aug;20(8):894. doi: 10.1016/S1473-3099(20)30233-4. Epub 2020 Mar 26

133. Tuite AR, Ng V, Rees E, **Fisman D**. Estimation of COVID-19 outbreak size in Italy. Lancet Infect Dis. 2020 May;20(5):537. doi: 10.1016/S1473-3099(20)30227-9. Epub 2020 Mar 19.

132. Tuite AR, Bogoch II, Sherbo R, Watts A, **Fisman D**, Khan K. Estimation of Coronavirus Disease 2019 (COVID-19) Burden and Potential for International Dissemination of Infection From Iran. Ann Intern Med. 2020 May 19;172(10):699-701. doi: 10.7326/M20-0696. Epub 2020 Mar 16.

131. MacFadden DR, Coburn B, Břinda K, Corbeil A, Daneman N, **Fisman D**, Lee RS, Lipsitch M, McGeer A, Melano RG, Mubareka S, Hanage WP.Using Genetic Distance from Archived Samples for the Prediction of Antibiotic Resistance in *Escherichia coli*. Antimicrob Agents Chemother. 2020 Apr 21;64(5):e02417-19. doi: 10.1128/AAC.02417-19. Print 2020 Apr 21.

130. Tuite AR, **Fisman DN**. Reporting, Epidemic Growth, and Reproduction Numbers for the 2019 Novel Coronavirus (2019-nCoV) Epidemic. Ann Intern Med. 2020 Apr 21;172(8):567-568. doi: 10.7326/M20-0358. Epub 2020 Feb 5

129. Logar-Henderson C*, Ling R*, Tuite A, **Fisman DN**. Effects of Large-Scale Oceanic Phenomena on Non-Cholera Vibriosis Incidence in the United States: Implications for Climate Change. *Epidemiology and Infection* 2019; in press.

128. Nasserie T*, Tuite AR, Whitmore L, Hatchette T, Drews SJ, Peci A, Kwong JC, Friedman D, Garber G, Gubbay J, **Fisman DN**. Seasonal Influenza Forecasting in Real Time Using the Incidence Decay With Exponential Adjustment Model. Open Forum Infect Dis. 2017 Sep 27;4(3):ofx166. doi: 10.1093/ofid/ofx166. eCollection 2017 Summer.

127. Tuite AR, Shaw S, Reimer JN, Ross CP, **Fisman DN**, Mishra S. Can enhanced screening of men with a history of prior syphilis infection stem the epidemic in men who have sex with men? A mathematical

modelling study. Sex Transm Infect. 2018 Mar;94(2):105-110. doi: 10.1136/sextrans-2017-053201. Epub 2017 Jul 13.

126. Guthrie JL*, **Fisman D**, Gardy JL. Self-rated health and reasons for non-vaccination against seasonal influenza in Canadian adults with asthma. PLoS One. 2017 Feb 16;12(2):e0172117. doi: 10.1371/journal.pone.0172117. eCollection 2017.

125. Derek MacFadden*, Sarah McGough, **David Fisman**, Mauricio Santillana, and John Brownstein. Antibiotic Resistance Increases with Local Temperature. Nature Climate Change, accepted for publication. [Paper #NCLIM-17050804C]

124. Santillana M, Tuite AR, Nasserie T*, Fine P, Champredon D, Chindelevich L, Dushoff J, **Fisman DN**. Relatedness of the incidence decay with exponential adjustment (IDEA) model, "Farr's law" and SIR compartmental difference equation models. Infectious Disease Modelling. Volume 3, 2018, Pages 1-12. url: https://www.sciencedirect.com/science/article/pii/S2468042718300101?via%3Dihub

123. Brankston G*, Boughen C, Ng V, **Fisman DN,** Sargeant J, Greer AL. Assessing the Impact of Environmental Exposures and Cryptosporidium Infection in Cattle on Human Incidence of Cryptosporidiosis in Southwestern Ontario, Canada. PLoS ONE, accepted for publication [PONE-D-17-14499R1]

122. MacFadden DR*, Bogoch II, Brownstein JS, Daneman N, **Fisman D**, German M, Khan K. A passage from India: Association between air traffic and reported cases of New Delhi Metallo-beta-lactamase 1 from 2007 to 2012. Travel Med Infect Dis. 2015 Jul-Aug;13(4):295-9. doi: 10.1016/j.tmaid.2015.06.003.

121. Tuite AR*, **Fisman DN**. Go big or go home: impact of screening coverage on syphilis infection dynamics. Sexually Transmitted Infections. Sex Transm Infect. 2015 May 7. pii: sextrans-2014-052001. doi: 10.1136/sextrans-2014-052001. [Epub ahead of print]

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119. Majumder MS*, Rivers C*, Lofgren E*, **Fisman D**. Estimation of MERS-Coronavirus Reproductive Number and Case Fatality Rate for the Spring 2014 Saudi Arabia Outbreak: Insights from Publicly Available Data. PLoS Curr. 2014 Dec 18;6. pii: ecurrents.outbreaks.98d2f8f3382d84f390736cd5f5fe133c. doi: 10.1371/currents.outbreaks.98d2f8f3382d84f390736cd5f5fe133c.

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113. Brown KA*, **Fisman DN**, Moineddin R, Daneman N. The magnitude and duration of Clostridium difficile infection risk associated with antibiotic therapy: a hospital cohort study. PLoS One. 2014 Aug 26;9(8):e105454. doi: 10.1371/journal.pone.0105454. eCollection 2014.

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111. Tuite A, Burchell A, **Fisman DN**. Cost-effectiveness of enhanced syphilis screening among HIV-positive men who have sex with men: a microsimulation model. PLOS One, in press.

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2. Ng V, Tang P, **Fisman DN**. Learning to count: our evolving understanding of legionellosis epidemiology. Clinical Infectious Diseases 2008; 47(5): 600-2. *I am senior responsible author*.

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F. PRESENTATIONS AT MEETINGS

Invited presentations

International:

60. **Fisman DN.** How to Be Wrong: Reflections on the Pandemic. University of Michigan Undergraduate Medicine Branches Spring Conference. April 4, 2022.

59. **Fisman DN** and Mossong J. Zero Risk doesn't exist but low risk does. European Conference on Coronavirus Diseases (ECCVID). September 23, 2020.

58. **Fisman DN.** Solving pandemic enigmas and communicating risk to the public. European Conference on Coronavirus Diseases (ECCVID). September 25, 2020.

57. **Fisman DN.** Schrodinger's Coronavirus: Paradoxes in COVID-19 Transmission. BSL4ZNet COVID19 International Symposium: Epidemiology and Transmission Dynamics (Online). August 19, 2020.

56. **Fisman DN.** Summary of GLOPID-R Symposium on Transmission. World Health Organization Symposium on COVID-19 Transmission. (Online). August 4, 2020.

55. **Fisman DN.** Paradoxes in Transmission. GLOPID-R Symposium on COVID-19 Transmission. (Online). July 20, 2020.

54. **Fisman DN**. Modeling Novel Coronavirus. Royal College of Physicians of Ireland Masterclass. April 1, 2020.

53. **Fisman DN**. Climate Change and Infectious Diseases. Pediatric Academic Societies Annual Meeting. Toronto, Canada, May 8, 2018.

52. **Fisman DN.** Modeling and Forecasting COVID-19: A Korea-Canada Partnership. Special Report to the Government of Korea (Address to Prime Minister Chung Sye-Kyun). April 23, 2020.

51. **Fisman DN**. Impact of Herd Immunity from Influenza Vaccination of Children and Adolescents. European Conference on Clinical Microbiology and Infectious Diseases. Madrid, Spain, April 21-24, 2018.

50 **Fisman DN.** Weather, Climate and Antimicrobial Resistance. European Conference on Clinical Microbiology and Infectious Diseases. Vienna, Austria, April 21-25, 2017.

49. **Fisman DN**. Climate change and infectious diseases dynamics. European Conference on Clinical Microbiology and Infectious Diseases. Amsterdam, The Netherlands, April 11, 2016.

48. **Fisman DN.** Single equation approaches to modeling emerging infectious diseases. Harvard School of Public Health Centre for Infectious Disease Dynamics Seminar Series. Boston, MA, February 26, 2015.

47. **Fisman DN**. Forecast Model 1: Incidence Decay and Exponential Adjustment Model. Forecasting Plenary. World Health Organization--London School of Hygiene and Tropical Medicine Meeting on Ebola Modeling. London, UK, February 16-17, 2015.

48. **Fisman DN** (Moderator). With Amy Greer, Keith Klugman, Xavier Rodo. Things Are Heating Up: Emerging Infectious Diseases and Climate Change. International Meeting on Emerging Infectious Diseases. Vienna, Austria, October 31-November 3, 2014.

47. **Fisman DN.** Indirect effects of climate on disease emergence: Population stress and migration, and emerging infectious diseases. International Meeting on Emerging Diseases, Vienna, Austria, October 31-November 3, 2014.

46. **Fisman DN.** The Incidence Decay and Exponential Adjustment (IDEA) model: a new single equation model to describe epidemics and their simultaneous control. Yale University School of Public Health Epidemiology of Microbial Disease Seminar Series. New Haven CT, October 9, 2014.

45. **Fisman DN.** Climate Change and Infectious Diseases: Knowns and Unknowns. Northeast Branch—American Society of Microbiology and American Society for Clinical Chemistry

Waltham, MA March 13, 2014

44. **Fisman DN**, Cattuto C, Horvitz E, Bharti N, Buckeridge, D. Big data and predictive analytics. Moving beyond nowcasting (panel). 2nd Conference on Digital Disease Detection. San Francisco CA, September 18-20, 2013.

43. **Fisman DN.** Climate change and changing patterns of infectious diseases. Council of State and Territorial Epidemiologists Annual Meeting. Omaha, Nebraska, June 6, 2012.

42. **Fisman DN.** Is it cost-effective? Best practices on evaluating the bang-for-the-buck in communicable disease control. International Meeting on Emerging Diseases and Surveillance (IMED). Vienna, Austria, February 15-18, 2013.

41. **Fisman DN.** Economic issues in the control of herpesvirus infections. Fondation Merieux Conference on Herpes and Immunity. Annecy, France, June 18-20, 2012.

40. **Fisman DN**. Catch the wave: seasonality of infectious diseases and why clinicians should care. Brown University Medical Grand Rounds. Providence, RI November 29, 2011.

39. **Fisman DN**. Bugs and bucks: infectious disease persistence is a matter of economics. Institute on Science for Global Policy, Emerging and Persistent Infectious Diseases: Focus on Prevention. La Jolla, California, June 5-8, 2011.

38. **Fisman DN**. Odd Couples: The Complex Relationship Between Influenza and Invasive Bacterial Diseases. Institut National de Veille Sanitaire (National Institute for Public Health Surveillance). Paris (Sainte-Maurice), France, March 26, 2011.

37. **Fisman DN**. The need for improved influenza vaccines in older adults. Novartis Vaccines Scientific Roundtable Meeting. Frankfurt, Germany, March 24, 2011.

36. **Fisman DN**. Odd Couples: The Complex Relationship Between Influenza and Invasive Bacterial Diseases. Centre Hopitalier Universitaire Seminar in Infectious Diseases Epidemiology. Lyon, France, February 9, 2011.

35. **Fisman DN**. Odd Couples: The Complex Relationship Between Influenza and Invasive Bacterial Diseases. Harvard School of Public Health, Lunchtime Seminars in Infectious Disease Epidemiology. Boston, MA, January 22, 2010.

34. **Fisman DN**. Climate change and disease transmission. Institute of Medicine Workshop on Indoor Air Quality and Climate Change. Institute of Medicine, Washington, DC, June 19, 2009.

33. **Fisman DN**. Residency training and fatigue: are we killing our house staff? Society for Hospital Epidemiology of America Annual Meeting; Plenary on Occupational Health and Safety, San Diego, California, March 21, 2009.

32. **Fisman DN**. Climate Change and Infectious Diseases in North America: The Road Ahead. Imperial College London Division of Infectious Disease Epidemiology Seminar Series, February 17, 2009.

31. **Fisman DN**. "'Sneezonality': What we know (and don't) about the seasonality of respiratory infections". AstraZeneca Epidemiology Seminar, Wilmington, Delaware, May 17, 2006.

30. **Fisman DN**. "'Sneezonality': What we know (and don't) about the seasonality of respiratory infections". University of Pennsylvania Infectious Disease Grand Rounds, Philadelphia, PA. March 23, 2006.

29. **Fisman DN**. A vaccine that works even when it fails: effect of prior pneumococcal vaccination on survival and morbidity in community acquired pneumonia. Harvard School

of Public Health, 5th Annual Jonathan Freeman Seminar in Infectious Diseases Epidemiology, Boston, MA, February 10, 2006.

28. **Fisman DN**. "Bugs and Bucks: The Economics of STD Control". University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics Seminar Series, Philadelphia, January 12, 2006.

27. **Fisman DN**. Economic costs of antibiotic resistance: identification, measurement, and valuation. New Jersey State Department of Health and Senior Services Antimicrobial Resistance Symposium. West Windsor, NJ, October 31, 2005.

26. **Fisman DN**. "Sharps related injuries and their prickly precipitants". Association of Professionals in Infection Control Philadelphia Chapter Meeting, Philadelphia, December 10, 2004.

25. **Fisman DN**. "Seasonality, weather, and acute infectious disease occurrence" University of Pennsylvania, Center for Clinical Epidemiology and Biostatistics Seminar Series, Philadelphia, October 21, 2004.

24. **Fisman DN**. "Seasonality, weather, and acute infectious disease occurrence" Emory University, Rollins School of Public Health Epidemiology Seminar Series, Atlanta GA, October 14, 2004.

23. **Fisman DN**. "Genital Herpes: Epidemiology and Cost-Effectiveness of Emerging Vaccines". Fox Chase Cancer Center Population Sciences Seminar. Philadelphia, PA, September 14, 2004.

22. **Fisman DN**. "The response to SARS". SARS and Emerging Infectious Diseases: Lessons Learned. New Jersey Hospital Association. Princeton, NJ. June 15, 2004.

21. **Fisman DN**. "Syndromic surveillance: rationale and implementation". Capitol Hill Steering Committee on Telehealth and Healthcare Informatics, "Advances in Biosurveillance, Early Warning, and Effective Response Toward Protecting Providers and the Public". Washington, DC. June 9, 2004.

20. **Fisman DN**. "Genital herpes: what's new?". Infectious Disease Grand Rounds, University of Pennsylvania. April 8, 2004.

19. **Fisman DN**. "Seasonality and infection: meningococcus runs hot and cold." Harvard School of Public Health Freeman Seminar in Infectious Disease Epidemiology. Boston, April 29, 2004.

18. **Fisman DN**. "Modeling genital herpes: Biological and economic considerations" (Elion IHMF Junior Investigator Award Lecture). 11th Annual Meeting of the International Herpes Management Forum. Amsterdam, The Netherlands, February 26-29, 2004.

17. **Fisman DN**. "SARS in Toronto: Lessons Learned (and Already Forgotten?)". Preparing Your Hospital for SARS. Florida Hospital Association, Orlando, FL, March 5 2004.

16. **Fisman DN**. How good is good enough? Modeling approaches to the effectiveness and cost-effectiveness of vaccines for herpes simplex virus type 2. Sexually Transmitted Diseases in Philadelphia: Linking Clinicians and Researchers. University of Pennsylvania and Centers for Disease Control and Prevention. Philadelphia, PA, January 28, 2004.

15. **Fisman DN**. Dynamic projection of cost-effectiveness of HSV-2 vaccines for young women. University of Tampere/Finnish National Public Health Institute International Symposium in Medicine. Lääkäripäivat 2004 (2004 Finnish Medical Association Annual Meeting). Helsinki, Finland, January 8, 2004.

14. **Fisman DN**. Prickly precipitants: epidemiology of needlesticks, and cost-effectivness of prevention. Johns Hopkins Bloomberg School of Public Health, Center for Injury Research and Policy, Graduate Seminar in Injury Research and Policy. Baltimore, MD, December 1, 2003.

13. **Fisman DN**. SARS in Toronto: the good, the bad, and the ugly. Southeastern Pennsylvania Regional Bioterrorism Preparedness Working Group. Philadelphia PA, August 6, 2003.

12. **Fisman DN**. Cold and rainy, with scattered relative risks: seasonality, weather, and invasive group A streptococcal disease. 3rd Annual Jonathan Freeman Memorial Seminar in Infectious Diseases Epidemiology, Harvard School of Public Health, Boston, MA, May 15, 2003.

11. **Fisman DN**. Novel tools for prevention of HSV-2 transmission: implications for HSV-2 testing. International Herpes Management Forum "Strategies for Interrupting the Transmission of HSV" Workshop, Seattle, WA, May 5, 2003.

10. **Fisman DN**. Prickly precipitants: epidemiology of needlesticks, and cost-effectivness of prevention. Drexel University School of Public Health. Philadelphia PA, January 11, 2003.

9. **Fisman DN**. Prickly precipitants: epidemiology of needlesticks, and cost-effectivness of prevention. Division of General Internal Medicine Rounds, University of Pennsylvania. Philadelphia PA, January 10, 2003.

8. **Fisman DN**. Control of herpes simplex virus type 2: using modeling to inform policy. Special Rounds, Department of Epidmiology and Preventive Medicine, University of Maryland. Baltimore MD, January 8, 2003.

7. **Fisman DN**. Precipitants of needlestick injuries in health care workers: a case-crossover study. Infectious Disease Grand Rounds, Washington University, St. Louis, MO. June 25, 2002.

6. **Fisman DN**, Mandl L. Medical management of osteoarthritis of the knee: cost-effectiveness of American College of Rheumatology guidelines. Partners Healthcare Arthritis Research Centre, Brigham and Women's Hospital. Boston, MA. May 1, 2002.

5. **Fisman DN**. Cost-effectiveness of directly observed therapy for the prevention of maternal-fetal HIV transmission. Lifespan/Tufts/Brown Center for AIDS Research Forum on Directly Observed Therapy for Treatment of HIV. Providence, Rhode Island. April 30, 2002.

4. **Fisman DN**. Virulent outbreak of severe group A streptococcal disease in a long-term care facility: control with mass antibiotic prophylaxis. 2nd Annual Jonathan Freeman Memorial Symposium in Infectious Disease Epidemiology. Harvard School of Public Health, Boston, MA. April 26, 2002.

3. **Fisman DN**. Control of herpes simplex virus type 2: using modeling to inform policy Sexually Transmitted Diseases Seminar Series, Johns Hopkins School of Medicine, Baltimore, Maryland. June 2001.

2. **Fisman DN**. A case-crossover study of sharps-related injuries in healthcare workers. Institute for Healthcare Improvement, Boston, MA. June 2001.

1. **Fisman DN**. Modeling genital herpes. 1st Annual Jonathan Freeman Memorial Symposium in Infectious Disease Epidemiology. May 2001.

National:

43. **Fisman DN,** keynote speaker. Andre Aisenstadt Memorial Clinical Day, Jewish General Hospital, University of Manitoba. October 28, 2021.

42. **Fisman DN** (co-organizer), COVID-19 pre-budget consultation for Hon. Chrystia Freeland. February 4, 2022.

41. **Fisman DN**, Epidemiology of COVID-19: Journal of a Plague Year, Dean's Grand Rounds, University of Manitoba. March 22, 2022.

40. **Fisman DN**, Prather K, Conly J. Is airborne transmission an important and mitigable aspect of the COVID-19 pandemic? – A panel discussion. O'Brien Institute for Public Health, University of Calgary, April 9, 2021.

39. **Fisman DN**. Appearance before Canadian House of Commons Select Committee on Health. COVID-19. March 8, 2021.

38. **Fisman DN.** Climate Change, Emerging Infections, and Pandemics. Jewish National Fund Symposium on Environmental Change and Pandemics (with Dr. Jacob Moran-Gilad, Ben Gurion University, Israel). June 8, 2020.

37. **Fisman DN**. Address to Canadian House of Commons on COVID-19. May 20, 2020.

36. **Fisman DN**. Modeling and Forecasting COVID-19. CANCOVID Seminar Series. May 15, 2020.

35. **Fisman DN** (co-organizer), Verrall A, Young Chun A, Leung G, Moran-Gilad J, Lipsitch M, Horgan M. Global best practices in SARS-CoV-2 control. Presentation to Deputy Prime Minister Chrystia Freeland. May 7, 2020.

34. **Fisman DN**. Climate change and infectious diseases. Canadian Infectious Diseases and Microbiology Annual Retreat. Toronto, Ontario, Canada, August 11, 2014.

33. **Fisman DN.** Economic issues in vaccine decision making. Vaccine Decision Making—Beyond the Science. Policy Panel 1. Canadian Immunization Conference. Ottawa, Canada. December 2-4, 2014.

32. **Fisman DN.** The Incidence Decay and Exponential Adjustment (IDEA) model: a new single equation model to describe epidemics and their simultaneous control. University of Montreal School of Veterinary Medicine. McGill University Department of Epidemiology, Biostatistics and Occupational Health, 50th Anniversary Seminar Series, October 27, 2014.

31. **Fisman DN.** The Incidence Decay and Exponential Adjustment (IDEA) model: a new single equation model to describe epidemics and their simultaneous control. University of Montreal School of Veterinary Medicine. Ste. Hyacinthe, Quebec. June 17, 2014.

30. **Fisman DN**. A whirlwind introduction to health economic analysis. Canadian Public Health Association. Economic Evaluation of New Influenza Vaccine Options (Plenary). Canadian Public Health Association Annual Meeting, Toronto, Canada, May 28, 2014.

29. **Fisman DN.** The impact of climate and environmental change on infectious diseases. Keynote Address. AMMI-CACMID Annual Meeting, Victoria, BC, April 3, 2014.

28. **Fisman DN.** Pharmacoeconomic Evaluation of Vaccines: How They're Different. Vaccine Industry of Canada Pharmacoeconomic Workshop. Toronto, Ontario, Canada, November 25, 2013.

27. **Fisman DN.** Severity of influenza in remote and isolated First Nations communities. Possible mechanisms and implications for control. Banff International Research Station Workshop on Mathematical Modeling of Indigenous Populations Health, Banff, AB, Canada, Sep 27-29, 2013.

26. **Fisman DN**. Economic evaluation of vaccines. Canadian Immunization Conference. Vancouver, British Columbia, December 3-5, 2012.

25. **Fisman DN**. One Health for Clinicians. Canadian Family Medicine Forum, Toronto, November 15, 2012.

24. **Fisman DN**. Influenza immunization in older adults: an epidemiological perspective. University of Ottawa "IDeology" Seminar, Ottawa, Ontario, Canada. October 12, 2011.

23. **Fisman DN**. Mathematical modeling: a useful tool for guidance of partner notification strategies. National Collaborating Centre on Infectious Diseases, national consultation on partner notification. Toronto, Ontario, Canada, October 4, 2011.

22. **Fisman DN** and Tuite AR. Estimation of the burden and economic costs of *Chlamydia trachomatis* infection in Canada. Public Health Agency of Canada, Ottawa, Ontario, Canada, June 30, 2011.

21. **Fisman DN** and Sargeant J. Prioritization of zoonotic diseases. Canadian Conference on Medical Education, plenary session on "One Health". Toronto, Ontario, Canada, May 10, 2011.

20. **Fisman DN.** The economics of disease persistence. Banff International Research Station Workshop on Persistent Infectious Diseases. Banff, Alberta, Canada, March 2, 2011.

19. Tuite AR, **Fisman DN**. Modeling in the real world: contribution of modeling to management of influenza pandemics. Public Health Agency of Canada—Canadian Pandemic Influenza Planning Meeting, Winnipeg, MB, February 1-2 2011.

18. **Fisman DN**, Greer A. Modeling disease spread in populations—overview and pts. One Health—One Model Zoonotic Disease Modeling Meeting. University of Guelph, November 1-4, 2010.

17. **Fisman DN**. What a Difference a Year Makes: PanINFORM, the 2009 pH1N1 Pandemic, and Mathematical Modeling in Canada. PanINFORM National Influenza Modeling Meeting (The First Influenza Pandemic of the 21st Century: Canada's Response, Lessons Learned, and Challenges Ahead). Winnipeg, MB. April 19-20, 2010.

16. **Fisman DN**. One Health: getting human health experts to think "trans-species". (Plenary address). Canadian Association of Veterinary Epidemiology and Preventive Medicine, Guelph, Ontario, Canada, May 29-30, 2010.

15. **Fisman DN**. The Flu Formula: How Math is Helping Canada Respond to H1N1. University of Western Ontario Applied Mathematics Seminar Series. London, Ontario, Canada, December 9, 2009.

14. **Fisman DN**. The Flu Formula: How Math is Helping Canada Respond to H1N1. MITACS 10th Anniversary Public Lecture. Vancouver, BC, November 6, 2009.

13. **Fisman DN.** Plenary: The Great Divide: Can Models Inform Disease-Control Policy in Real Time? Mitigating the spread of influenza A (H1N1) (Part II): An International Mathematical

Modelling Meeting. British Columbia Centre for Disease Control (BCCDC), Vancouver, BC, Canada, September 14 – 16, 2009.

12. **Fisman DN.** Age and Epidemiology of Novel Influenza A (H1N1) in Ontario. Mitigating the spread of influenza A (H1N1) (Part II): An International Mathematical Modelling Meeting. British Columbia Centre for Disease Control (BCCDC), Vancouver, BC, Canada, September 14 – 16, 2009.

11. **Fisman DN**. Invasive bacterial disease, seasonality, and climate change. 26th International Conference on Chemotherapy and Infection/2009 Annual Meeting of the Association of Medical Microbiology and Infection of Canada. Plenary session "the changing climate of infectious diseases". Toronto, Ontario, June 18, 2009.

10. **Fisman DN**, Deonandan R. An Expert Panel Discussion on Health Effects of Climate Change. University of Ottawa Health Science Students Environmental & Public Health Advocacy Group and the Student Federation of the University of Ottawa; Ottawa, Ontario, December 5, 2008.

9. **Fisman DN.** But the bugs bounce back: simple transmission models and "failure" of bacterial STD control programs. Canadian Applied and Industrial Mathematics Mini-symposium on Communicable Diseases, 2nd Canada-France Congress on Mathematics. Universite de Quebec a Montreal, Montreal, PQ, June 1, 2008

8. **Fisman DN.** There's a Bug in My Model: Using Mathematical Modeling to Inform Communicable Disease Control Policy and Practice. University of Calgary Division of Infectious Diseases Rounds, Calgary, Alberta, Canada, January 8, 2008.

7. **Fisman, DN**. Environment, Climate Change, and Infectious Diseases. Infectious Diseases Seminar, Queens University, Kingston, Ontario, Canada. November 27, 2007.

6. **Fisman DN.** High School-Based Chlamydia Screening: Projected Health and Economic Impact in Philadelphia. Public Health Agency of Canada—MITACS Joint Symposium on Modeling Sexually Transmitted and Blood-Borne Infections. Banff International Research Station for Mathematical Innovation and Discovery, Banff, Alberta, Canada, August 10-12, 2007.

5. **Fisman DN.** "Seasonality, Environment, and Infectious Disease Occurrence: A Novel Application for Case-Crossover Study Design". University of Alberta Public Health Sciences Grand Rounds. Edmonton, Alberta, Canada, October 5, 2005.

4. **Fisman DN.** "Environmental Factors and Acute Communicable Disease Occurrence: A Rediscovery". University of Western Ontario Homecoming 2004 Seminar: Political and Ecological Influences on Health. London, Ontario, Canada, October 2, 2004.

3. **Fisman DN**. Control of herpes simplex virus type 2: using modeling to inform policy. British Columbia Center for Disease Control. November 8, 2003.

2. **Fisman DN**. Prophylaxis and immunization in the emergency room. Canadian Association of Emergency Physicians. Annual Scientific Assembly. Hamilton, Ontario. April 19, 2002.

1. **Fisman DN**. Sexually transmitted diseases: an overview for the mental health professional. Department of Psychiatry, University of Western Ontario, London, Ontario, Canada. April 2001.

<u>Local:</u>

47. Fisman DN, DeVilla EP, Sinha S. Update on COVID-19 with Councillor Josh Matlow.

46. **Fisman DN**. Epidemiology of COVID-19: An Update. Hospital for Sick Children (Toronto) Perioperative Services Grand Rounds. February 19, 2021.

45. **Fisman DN**. Epidemiology of COVID-19: A Whirlwind Introduction. Medical Grand Rounds, Michael Garron Hospital, November 4, 2020.

44. **Fisman DN**. Epidemiology of COVID-19: A Whirlwind Introduction. University of Toronto Respirology Grand Rounds, May 29, 2020.

43. **Fisman DN**, Birn AE, Orbinski J, Upshur R, Lavery J. Ebola In Context Symposium, Panel Discussion. University of Toronto Student Ebola Working Group, Toronto, Ontario, April 22, 2014.

42. **Fisman DN**, Bean S, Fong G, Caulford P. Opening the medicine cabinet: Economic limitations on public health provision. Ill With Illness. Munk School Graduate Conference, University of Toronto, March 27, 2015.

41. **Fisman DN**. How epidemics grow and stop: Ebola 2014 as a case study. Ebola: Stories and Perspectives from the Frontlines. Ryerson University, Toronto, ON March 4, 2014.

40. **Fisman DN,** Upshur R, Orbinski J. Ebola in context: a conversation. University of Toronto, January 16, 2015.

39. **Fisman DN**. Moderator. Ebola: a global response. University of Toronto Faculty of Medicine Student Global Health Conference. Toronto, Ontario, February 11, 2015.

38. **Fisman DN.** Clinical case rounds: typhoid. University of Toronto Infectious Disease Conference, December 17, 2014.

37. **Fisman DN.** Ebola 2014: How did we get here? What can we expect? Public lecture, University of Toronto in Your Neighborhood. Toronto, Canada, November 13, 2014.

36. **Fisman DN**, Kamanye AM and Bogoch I. Ebola and vulnerable health systems. Amref Health Coffee House Speaker Series. Toronto, Canada, November 22, 2014. (http://www.amrefcanada.org/media-centre/stories/is-the-ebola-outbreak-a-symptom-of-poor-health-systems/)

35. **Fisman DN**. Sexy models: what math can tell us about STI in Ontario. Public Health Ontario Grand Rounds. Toronto, Canada. October 24, 2013.

34. **Fisman DN**. Is it cost-effective? Why communicable diseases are different (and why clinicians should care). University of Toronto City-Wide Infectious Diseases Conference. Sunnybrook Health Sciences Centre, Toronto, Canada. January 8, 2013.

33. Tuite AR, Mishra S, **Fisman DN**. Mathematical modeling and resurgence of sexually transmitted infections in Canada. Canadian National Infectious Disease Fellows' Retreat. University of Toronto, Canada. August 16, 2012.

32. **Fisman DN.** John Snow: Insights into Emerging Infections from the Pre-Microbiologic Era. John Snow 200th Birthday Bash. Dalla Lana School of Public Health, University of Toronto, March 15, 2013.

31. **Fisman DN**. It's Gettin' Hot in Here: Climate Change and Implications for Infectious Disease Control. University of Toronto School of the Environment Environmental Health Seminar Series. January 24, 2013

30. Tuite AR, **Fisman DN.** Understanding the Increase in Chlamydia Risk in Ontario through Applied Epidemiology and Mathematical Modeling. York Region Community and Health Services. September 26, 2012

29. Agard E, Rutty C, **Fisman DN**. Vaccines: is controversy overshadowing science? Ontario Science Centre Café Scientifique. May 26, 2012.

28. **Fisman DN**. Using mathematical models to inform syphilis prevention strategies in Ontario. Ontario Syphilis Working Group. Toronto, April 30, 2012.

27. Bell J, **Fisman DN.** Can Viruses Cure Cancer? Ontario Institute for Cancer Research Café Scientifique. February 9, 2012.

26. **Fisman DN.** Influenza immunization in older adults: an epidemiological perspective. FitzGerald Seminar Series in Communicable Disease Epidemiology, Dalla Lana School of Public Health, University of Toronto. October 13, 2011.

25. **Fisman DN.** Climate change and infectious diseases. York Region Infection Prevention and Control Education Day. Kettleby, Ontario, October 5, 2011.

24. **Fisman DN** and Tuite AR. Mathematical epidemiology of pertussis in the Greater Toronto Area: Implications for vaccine policy. GlaxoSmithKline Canada, Mississauga, Ontario, March 16, 2011.

23. **Fisman DN.** Cholera Model in Haiti, 2010—Using a Gravity Model to Explain Initial Spatial Dynamics. Toronto Public Health Epi Lunch Bunch; Toronto, Ontario, Canada, March 11, 2011.

22. Tuite AR, **Fisman DN**. Plagues past: what history teaches us about epidemics. Woodsworth College Alumni Lecture Series, University of Toronto. January 18, 2011.

21. **Fisman DN**, Greer AL, Jones N, Derry B. It's Getting' Hot in Here: Climate Change and Infectious Diseases. Canadian Institutes for Health Research Café Scientifique presented by the Research Institute of the Hospital for Sick Children. Toronto, Ontario, Canada, October 5, 2009.

20. **Fisman DN**. Flu on the Fly: Emerging Diseases, Public Policy, and the Influenza Pandemic. Woodsworth College (University of Toronto) Alumni Café. October 13, 2009.

19. **Fisman DN.** The Ontario Mathematical Epidemiology Hub ("ONTology"). Public Health Agency of Canada National Mathematical Modeling Meeting, Toronto, Ontario, Canada, July 9, 2009.

18. **Fisman DN**. Bright Ideas? Ultraviolet Radiation, Weather, and the Seasonality of Invasive Bacterial Disease in North America. Toronto Invasive Bacterial Disease Network (TIBDN) Research Day, Mt. Sinai Hospital, Toronto. November 27, 2008.

17. **Fisman DN**. Overview of Modelling as it relates to Public Health and Emergency Preparedness. Ontario Agency for Health Protection and Promotion Session on Disaster Preparedness, Canadian Critical Care Conference. Toronto, Ontario, November 11, 2008.

16. **Fisman DN**. By the numbers: math, vaccines, and the secrets of disease control. St. Michael's Hospital Center for Global Health Research, Toronto, Ontario, Canada, August 1, 2008

15. **Fisman DN**. Modeling Genital Herpes and Related Conditions: Exercises, Approaches, and Evaluation of Cost-Effectiveness. Public Health Agency of Canada-MITACS Conference on Mathematical Modeling of Herpes Simplex Viruses and Human Papillomavirus. York University, Toronto, Ontario, Canada, May 29-30, 2008.

14. **Fisman DN**. By the numbers: math, vaccines, and the secrets of disease control. University of Toronto Infectious Diseases/Microbiology Academic Day, Toronto, Ontario, Canada, May 27, 2008.

13. **Fisman DN**. "Making Best Bets: Mathematical Modeling as a Tool for Vaccine Policy". St. Michael's Hospital Clinical and Population Research Rounds, February 7, 2008.

12. **Fisman DN.** "Old Timey Diseases" in the Here and Now. Fields Institute Center for Mathematical Medicine Seminar Series, Toronto, Ontario, Canada. January 25, 2008.

11. **Fisman DN**. A high-school Chlamydia screening program. Toronto Public Health "Epi Lunch Bunch". Toronto, Ontario, Canada, January 22, 2008.

10. **Fisman DN.** "Pertussis: the disease that won`t go away". York Region Health Services Lunch and Learn. Newmarket, Ontario, June 25, 2007.

9. **Fisman DN.** "Seasonality, Environment, and Infectious Diseases". Sunnybrook and Women's Hospital Infectious Disease/Microbiology Rounds, June 19, 2007.

8. **Fisman DN.** "There's a Bug in this Model: Transmission Modeling in Epidemiology and Health Policy". York University MITACS Seminar, Toronto, Ontario, February 13, 2007.

7. **Fisman DN.** "Enhanced Screening for *Chlamydia* Control: Recent Experience and Projected Health and Economic Impact in Philadelphia". Plenary Session on Sexually Transmitted Disease Control (Chairs Edward W. Hook III and Jonathan Zenilman). 44th Annual Meeting Infectious Disease Society of America, Toronto, Ontario, Canada, October 14, 2006.

6. **Fisman DN**. "Bugs and bucks: cost-effectiveness of Philadelphia's high-school *Chlamydia* screening program." Population Health Sciences Seminar, Hospital for Sick Children Research Institute, Toronto, Ontario, Canada. March 20, 2006.

5. **Fisman DN**. Invasive group A streptococcal disease in long-term care. Toronto Public Health Infection Control Education Day. Toronto, Ontario, November 5, 2002.

4. **Fisman DN**. Needlestick injuries: identifying precipitants and evaluating the cost-effectiveness of prevention. Institute for Clinical Evaluative Sciences, Sunnybrook Hospital, Toronto, Ontario. October 9, 2002.

3. **Fisman DN**. Update on genital herpes. Phoenix Association (Herpes Support Group). Toronto, Ontario. March 20, 2002.

2. **Fisman DN**. Report of an invasive group A streptococcal outbreak investigation in a nursing home. Toronto Invasive Bacterial Disease Network Research Day. Mt. Sinai Hospital, Toronto, February 7, 2002.

1. **Fisman DN**. Bioterrorism: Simulation, Preparation, Motivation. Ontario Hospital Association Roundtable on Bioterrorism. Toronto, Ontario. December 20, 2001.

Contributed presentations, peer reviewed

43. Brown KA, Daneman N, **Fisman DN**. Above and Beyond Individual Exposure: Ward-level Antibiotic Prescribing Is the Principal Predictor of Increased *Clostridium difficile* Infection (CDI) Risk. ID Week, Philadelphia PA, October 11, 2014.

42. McGirr AA and **Fisman DN**. "Duration of Pertussis Immunity Following Childhood Immunization with DTaP: A Systematic Review and Meta-Analysis". The Canadian Society for Epidemiology and Biostatistics National Student Conference, May 2014.

41. Tuite AR, **Fisman DN.** Estimation of the burden of disease and costs of genital *Chlamydia trachomatis infection in Canada*. International Society for Sexually Transmitted Diseases Research Biannual Meeting, Quebec City, Quebec. July 10-13, 2011.

40. Chan CH, McCabe CJ, **Fisman DN.** Core Groups, Antimicrobial Resistance and Rebound in Gonorrhea. International Society for Sexually Transmitted Diseases Research Biannual Meeting, Quebec City, Quebec. July 10-13, 2011.

39. Tuite AR, **Fisman DN**. Pertussis in Ontario, Canada: a transmission dynamic model. North American Congress of Epidemiology, Montreal, Quebec. June 21-24, 2011.

38. Tuite AR, **Fisman DN**. Seasonality of influenza-attributable meningococcal disease in central Ontario, Canada: implications for targeting of influenza vaccination programs. AMMI Canada – CACMID Annual Conference 2011, Montreal, Quebec, April 7-9, 2011.

37. Brown K, **Fisman DN.** A mathematical model of nosocomial clostridium difficile infection (CDI) transmission in an acute care hospital system with seasonal variations in transmission rate. AMMI Canada – CACMID Annual Conference 2011, Montreal, Quebec, April 7-9, 2011.

36. Devault A, Poinar H, Tien J, Earn D, **Fisman DN,** Dhody A. Ancient DNA analysis of 19th century cholera. Society for American Archeology, Sacramento, CA, March 30-April 2, 2011.

35. Tuite AR, Tien J, Earn DJD, Eisenberg M, Ma J, **Fisman DN.** Use of a gravity model to reproduce spatial patterns of cholera spread in Haiti, 2010. International Meeting on Emerging Diseases, Vienna, Austria, February 4-7, 2011.

34. Tuite AR, **Fisman DN**. Cholera, commerce and contagion: rediscovering Dr. Beck's report. Pennsylvania Medical Humanities Consortium Annual Meeting. Philadelphia, PA May 19-20, 2010.

33. **Fisman DN**. Gonorrhea Ain't Gone: Dissemination of Antibiotic Resistance via Core Groups. Canadian Mathematics Society Winter Meeting 2008. Ottawa, Ontario, December 6, 2008.

32. Soverow J, Wellenius G, **Fisman D**, Mittleman MS. Infectious Disease in a Warming World: How Weather Influenced West Nile Virus in the United States (2001-2005). 20th Annual Conference of the International Society for Environmental Epidemiology, October 12-16, 2008 Pasadena, CA.

31. **Fisman DN**, Greer A, Broukhanski G, Drews S. Of Gastro and the Gold Standard: Use of Latent Class Modeling to Estimate Test Performance for a Novel PCR and EIA for Norovirus G1 and G2. AMMI Canada—CACMID Annual Conference. Vancouver, British Columbia, February 27 - March 1, 2008.

30. Kinlin L, Spain CV, Ng V, White A, Johnson C, **Fisman DN**. Seasonal Variation and Environmental Effects in Invasive Meningococcal Disease in Philadelphia, Pennsylvania. AMMI Canada—CACMID Annual Conference. Vancouver, British Columbia, February 27 - March 1, 2008.

29. **Fisman DN**, Tang P, Richardson S, Drews S, Jamieson F. Pertussis Resurgence in Toronto, 2007. The View from the Lab. Late Breaker Sessions II: 2007 Annual Meeting of the Pediatric Academic Societies. Toronto, Ontario, Canada, May 5-8, 2007.

28. Cohen E, Weinstein M, **Fisman DN**. What Is the Most Cost Effective Treatment for Pediatric Empyema? 2007 Pediatric Academic Societies Annual Meeting, Toronto, Ontario, Canada. May 5-8, 2007.

27. Drews S, **Fisman D**, Brouhanski G, Chedore P, Jamieson F. Association of histopathology and biopsy specimen type with direct detection of Mycobacterium tuberculosis by PCR.

AMMI-Canada CACMID 2007 Annual Conference, Halifax, Nova Scotia, Canada, March 14-18 2007.

26. Chedore P, **Fisman D**, Jamieson F. Current trends in extremely drug resistant (XDR) tuberculosis in Ontario. International Union against Tuberculosis and Lung Disease (IUATLD) 11th North American Regional Conference. Vancouver, British Columbia, Canada. February 22-24, 2007.

25. **Fisman DN**, Spaude KA, Kirchner C, Kim A, Abrutyn EA, Daley J. Recent influenza vaccination reduces adverse health outcomes in adults with community-acquired pneumonia. 16th Annual Meeting of the Society for Hospital Epidemiology of America (SHEA), Chicago, IL, March 18-21, 2006.

24. Johnson-Masotti, AP, **Fisman DN**, Lynd L, Sheehan D. Anonymous HIV testing in Canada: a cost-effective health intervention. Health Services Restructuring: New Evidence and New Directions. John Deutch Institute for the Study of Economic Policy. Queens University, Kingston, Ontario, Canada. November 17-18, 2005.

23. **Fisman DN**, Spain V, Asbel L, Goldberg M, Lawrence D, Newbern EC. High-school-based screening for Chlamydia in Philadelphia: identification of cost-savings using a dynamic transmission model. 27th Annual Meeting of the Society for Medical Decision Making. San Francisco, CA, October 21-24, 2005.

22. **Fisman DN**, Edmunds J. The importance of transmissibility in estimating cost-effectiveness of STI prevention: lessons from simulation studies. 16th Biennial Meeting of the International Society for Sexually Transmitted Disease Research. Amsterdam, The Netherlands. July 10-13, 2005.

21. Sorock GS, Lombardi DA, **Fisman DN**, Harris AD, Courtney TK, Evanoff B, Smith GS, Mittleman MA. Future directions for case-crossover research in injury epidemiology. 132nd Annual Meeting of the American Public Health Association. Washington, DC. November 6-10, 2004.

20. **Fisman DN**, Johnson-Masotti A, Lynd L, Sheehan D. Anonymous HIV Testing in Canada: A Cost-Effective Health Intervention. 132nd Annual Meeting of the American Public Health Association. Washington, DC. November 6-10, 2004.

19. **Fisman DN**, Spaude K, Kirchner C, Kim A, Daley J, Alexander J, Zhang J, Abrutyn E. Prior Pneumococcal Vaccination Reduces Death and Respiratory Failure Among Adults Admitted to Hospital with Community-Acquired Pneumonia. 44th Interscience Congress on Antimicrobial Agents and Chemotherapy. Washington, DC. October 30-November 2, 2004.

18. **Fisman DN**, Goldie SJ, Hook EW, Lipsitch M. Dynamic projection of ness and cost-effectiveness of HSV-2 vaccine for young women: how good is good enough? CDC STD Prevention Meeting, Philadelphia PA, March 8-11, 2004.

17. **Fisman DN**, Goldie SJ, Hook EW, Lipsitch M. Dynamic projection of the effectiveness and cost-effectiveness of HSV-2 vaccine for young women: how good is good enough? 11th Annual Meeting of the International Herpes Management Forum, Amsterdam, The Netherlands, February 26-29, 2004.

16. **Fisman DN**, Harris AD, Sorock GS, Mittleman MA. Cost-effectiveness of safer sharp medical devices for prevention of HIV and hepatitis C infection in healthcare workers. NIOSH/CDC National Occupational Injury Symposium, Pittsburgh PA, October 29 2003.

15. **Fisman DN**. The season's the reason: invasive group A streptococcal disease and weather patterns in a Canadian city. Pennsylvania Public Health Association Conference, Harrisburg, PA, October 17, 2003.

14. **Fisman DN**, Goldie SJ, Hook EW, Lipsitch M. Dynamic projection of the effectiveness and cost-effectiveness of HSV-2 vaccine for young women. Pennsylvania Public Health Association Conference, Harrisburg, PA, October 16, 2003.

13. Weir E, Taha M, Knowles L, Hart R, Haley A, **Fisman DN**, Tsang L, Li A, Sheehan D. Devil take the hindmost: A large community verotoxigenic E. Coli outbreak associated with haggis consumption. Society for Hospital Epidemiology of America 13th Annual Meeting, Arlington, VA. April 5-8, 2003.

12. **Fisman DN**. Cost-effectiveness of directly observed highly active antiretroviral therapy in pregnant HIV-infected women. Ontario HIV Treatment Network Research Day. Toronto, Ontario, November 28-29, 2002.

11. Kleiner-Fisman G, **Fisman D**, Sime E, St. Cyr J, Lozano A, Lang A. Long-term outcome of subthalamic nucleus deep brain stimulation in patients with advanced Parkinson's disease. 7th Annual International Congress on Parkinson's Disease and Other Movement Disorders, Miami, FL, November 10-14, 2002.

10. Kleiner-Fisman G, **Fisman D**, Khan F, Sime E, Lozano A, Land A. Motor cortical stimulation in patients with multi-system atrophy. 7th Annual International Congress on Parkinson's Disease and Other Movement Disorders, Miami, FL, November 10-14, 2002.

9. Mandl LA, Liang M, **Fisman DN**. Cost-effectiveness of competing strategies for management of knee osteoarthritis. American College of Rheumatology 66th Annual Scientific Meeting, New Orleans, LA, October 25-29, 2002.

8. **Fisman DN**. Cost-effectiveness of competing strategies for management of osteoarthritis of the knee. 24th Annual Meeting of the Society for Medical Decision Making Annual Meeting, Baltimore, MD, October 20–23, 2002

7. **Fisman DN**. Cost-effectiveness of post-exposure antibiotic prophylaxis in household contacts of individuals with severe invasive group A streptococcal disease. 24th Annual Meeting of the Society for Medical Decision Making Annual Meeting, Baltimore, MD, October 20–23, 2002

6. Perencevich EN, **Fisman DN**, Harris AD, Morris JG, Smith DL. Point prevalence and clinical culture positivity may be inadequate measures of an infection control intervention's effectiveness. 24th Annual Meeting of the Society for Medical Decision Making Annual Meeting, Baltimore, MD. October 20–23, 2002

5. **Fisman DN**, Smith A. Virulent outbreak of severe group A streptococcal disease in a long-term care facility: control with mass antibiotic prophylaxis. 12th Annual Meeting, Society for Hospital Epidemiology of America, Salt Lake City, UT April 6-9, 2002.

4. **Fisman DN**, Harris AD, Lipsitch M, Perencevich EN, Smith DL. Benefits of active surveillance for vancomycin-resistant enterococcus on ICU Admission assessed with a stochastic model. 41st International Congress on Antimicrobial Agents and Chemotherapy, Chicago, IL, December 16-19, 2001.

3. **Fisman DN**, Perencevich EN, Cosgrove SE, Levy DB, Goldie SJ. Cost-effectiveness of directly observed highly active antiretroviral therapy in pregnant women with asymptomatic HIV infection. Infectious Disease Society of America 39th Annual Meeting, San Francisco,

CA, 2001, and Society for Medical Decision Making Annual Meeting, San Diego, CA October 25 –28, 2001.

2. Perencevich EN, Lipsitch M, Harris AD, **Fisman DN**. Estimating the costs and benefits of active surveillance for vancomycin resistant enterococcus on ICU admission. Society for Healthcare Epidemiology of America Annual Meeting, Toronto, Ontario April 1 – 3, 2001.

1. **Fisman DN**, Goldie SJ. Estimating the costs and benefits of screening monogamous, heterosexual couples for asymptomatic infection with herpes simplex virus type 2. Society for Medical Decision Making 22nd Annual Meeting, Cincinnati, OH October 2000.

Media Interviews and Appearances (Google):

https://www.google.com/search?q=%22David+Fisman%22&rlz=1C5CHFA_enCA804CA804&source=lnms&tbm=nws&sa=X&ved=2ahUKEwjp4q_Bo4XvAhWXElkFHXfWBzQQ_AUoAXoECAQQAw&biw=1440&bih=789

Contributed poster presentations, peer reviewed

58. Tuite AR and **Fisman DN**. Go big or go home: impact of screening coverage on syphilis infection dynamics. International Meeting on Emerging Infectious Diseases and Surveillance, Vienna Austria. October 31-November 3, 2014.

57. Tuite AR and **Fisman DN**. Are screening blitzes contributing to the observed trends in syphilis outbreaks in urban men who have sex with men? ID Week, Philadelphia, PA October 8-12, 2014.

56. McGirr AA and **Fisman DN**. "Duration of Pertussis Immunity Following Childhood Immunization with DTaP: A Systematic Review and Meta-Analysis" Poster presented at the Canadian Immunization Conference. Ottawa, ON. Dec 1-4, 2014.

55. McGirr AA and Fisman DN. "Duration of Pertussis Immunity Following Childhood Immunization with DTaP: A Systematic Review and Meta-Analysis". Poster presented at The Society for Epidemiologic Research Annual Meeting. Seattle, WA. June 24-27, 2014.

54. Brown KA, Daneman N, Moinedden R, Fisman DN. The duration of effects of antibiotic exposures on the risk of Clostridium difficile infection (CDI): a cohort study. International Meeting on Emerging Diseases and Surveillance (IMED). Vienna, Austria, February 15-18, 2013.

53. Tuite AR, **Fisman DN,** Alexander D, Guthrie J, Marchand-Austin A, Lam K, Ma J, Whelan M, Lee B, Jamieson F. Epidemiological evaluation of spatio-temporal and genotypic clustering of Mycobacterium tuberculosis in Ontario, Canada. International Meeting on Emerging Diseases and Surveillance (IMED). Vienna, Austria, February 15-18, 2013.

52. Vasilevska M, Major M, McGeer A, Brown V, Greer A, Tuite A, Ulanova M, Morris S, FitzGerald J, DeAngelis F, **Fisman DN.** The FitzGerald Seminar Series - Creation of an Open Access Infectious Disease Control and Prevention Seminar Series in Ontario. 10th Canadian Immunization Conference, Vancouver, Canada, December 3-5, 2012.

51. Tuite AR, **Fisman DN.** Estimation of the health impact and cost-effectiveness of an adjuvanted influenza vaccine with enhanced effectiveness and durability of effect. Poster presented at: 3rd North American Congress of Epidemiology, June 2011.

50. Devault, Alison, Hendrik N. Poinar, Joseph H. Tien, David J.D. Earn and **David N. Fisman.** *Ancient DNA analysis of 19th century North American cholera* Multiple pandemics. Society for American Archeology 76th Annual Meeting, Sacramento, California, March 30-April 3, 2011.

49. Tuite AR, **Fisman DN.** Cost-effectiveness of an adjuvanted vaccine for prevention of influenza in Ontario, Canada. International Meeting on Emerging Diseases, Vienna, Austria, February 4-7, 2011.

48. Xiao Y, **Fisman DN**. Impact of antiviral drug use on epidemic dynamics in an isolated First Nations reserve in Ontario, 2009. International Meeting on Emerging Diseases, Vienna, Austria, February 4-7, 2011.

47. Kuster S, Tuite AR, McGeer A, Kwong J, **Fisman DN**. Influenza drives risk of invasive pneumococcal disease but not pneumococcal transmission dynamics in Toronto, Canada. International Society for Prevention of Pneumococcal Disease, Tel Aviv, Israel, March 14-18, 2010.

46. Tuite AR, Kinlin LM, **Fisman DN**. Influenza A activity and increased risk of invasive meningococcal disease in central Ontario, Canada: a case-crossover analysis. European Society for Pediatric Infectious Diseases, Nice, France, May 4-8, 2010.

45. Kinlin L, Kirchner C, Zhang H, Daley J, **Fisman DN**. Derivation and validation of a clinical prediction rule for nosocomial pneumonia following coronary artery bypass grafting surgery. Annual Meeting of the Society for Hospital Epidemiology of America. San Diego, California, March 20-22, 2009.

44. Kinlin LM, Ng V, Crowcroft N, Granerod J, Fraser G, Spain CV, Johnson CC, Jamieson F, Brown EM, **Fisman DN**. Seasonal Patterns and Environmental Predictors of Invasive Meningococcal Disease in London, England; Philadelphia, United States; Sydney, Australia; and Toronto, Canada. International Society of Infectious Diseases—International Meeting on Emerging Diseases. Vienna, Austria, February 13-16, 2009.

43. **Fisman DN.** Rate of change of Lyme disease incidence in the United States exhibits a north-south gradient consistent with climate change effect. International Society for Infectious Diseases—International Meeting on Emerging Diseases. Vienna, Austria, February 13-16, 2009.

42. Greer A, **Fisman DN**. Keeping vulnerable children safe from pertussis: preventing nosocomial pertussis transmission in the neonatal intensive care unit (NICU). *Epidemics* First Annual Conference, Asilomar, California. December 1-3, 2008.

41. Greer A, **Fisman DN**. Keeping vulnerable children safe from pertussis: preventing nosocomial pertussis transmission in the neonatal intensive care unit (NICU). 120th Anniversary Conference of the Pasteur Institute, Paris, France. November 11-13, 2008.

40. Ng V, Tang P, Jamieson F, Guyard C, **Fisman DN**. Laboratory-Based Evaluation of the Epidemiology of Legionellosis in Ontario, Canada, 1978 to 2006. 46th Annual Conference of the Infectious Disease Society of America, Washington, DC, October 25-28, 2008.

39. Kinlin L, Jamieson F, Brown E, Rawte P, Brown S, Dolman S, **Fisman DN**. Impact of Conjugate Group C Meningococcal Vaccine on Invasive Meningococcal Disease in Vaccinated and Unvaccinated Groups in Ontario, Canada, 2000 to 2006. 46th Annual Conference of the Infectious Disease Society of America, Washington, DC, October 25-28, 2008

38. Greer AL, Drews S, **Fisman DN**. Why Does the "Winter Vomiting Disease" Happen in Winter? Unravelling the Seasonality of Norovirus Outbreaks in Toronto, Canada. 46th Annual Conference of the Infectious Disease Society of America, Washington, DC, October 25-28, 2008.

37. White ANJ, Kinlin L, Johnson C, Ng V, **Fisman DN**. Let the Sun Shine In: Temperature and UV Radiation Affect the Incidence of Pneumococcal Infection in Philadelphia. 46th Annual Conference of the Infectious Disease Society of America, Washington, DC, October 25-28, 2008.

36. White ANJ, Johnson C, Ng V, **Fisman DN**. Environmental Effects on the Incidence of *Campylobacter* Infection in Philadelphia. 2008 Canadian *Campylobacter* Conference. Montreal, Quebec, September 25-26, 2008.

35. Ota K, **Fisman DN**, Jones K, Tamari I, Jamieson F, Wong T, DePrima A, Richardson, S. Prevalence and characteristics of *Neisseria gonorrhoeae* isolates in Ontario. AMMI Canada—CACMID Annual Conference. Vancouver, British Columbia, February 27 - March 1, 2008.

34. Brown E, **Fisman DN**, Brown S, Rawte P, Jamieson F. Epidemiology of invasive meningococcal disease with decreased penicillin susceptibility in Ontario, 2000 to 2006. AMMI Canada—CACMID Annual Conference. Vancouver, British Columbia, February 27 - March 1, 2008.

33. Ng-Brett V, **Fisman DN**, Moineddin R. Cute, Cuddly, Contagious: Kangaroo Density Drives Human Ross River Virus Infections. Late breaker, American Society of Tropical Medicine and Hygeine 56th Annual Meeting, Philadelphia PA, November 4-8, 2007.

32. Ng-Brett V, Tang P, Jamieson F, Drews S, Johnson C, **Fisman DN**. Hydrological factors associated with increase legionellosis risk in the Greater Toronto Area, Ontario, Canada. 47th Annual Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC), Chicago, Illinois, United States of America, September 17- 20, 2007.

31. **Fisman DN,** Tang P, Richardson S, Ng-Brett V, Drews S, Low DE, Jamieson F. Laboratory Factors in an Apparent Pertussis Resurgence, Toronto, Canada, 2005-2007. 47th Annual Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC), Chicago, Illinois, United States of America, September 17- 20, 2007.

30. **Fisman DN,** Spain V, Ng-Brett V, Johnson C. Weather, Water and Giardia in Philadelphia. 47th Annual Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC), Chicago, Illinois, United States of America, September 17- 20, 2007.

29**.** **Fisman DN**, Harris AD, Rubin M, Daley K, Mittleman, MA. Alcohol consumption and sharps-related injuries among healthcare workers: results from a case-crossover study. 16th Annual Meeting of the Society for Hospital Epidemiology of America (SHEA), Chicago, IL, March 18-21, 2006.

28. **Fisman DN**. Cost-effectiveness of the SuturTek 360° Fascia Closure Device for prevention of operative sharps-related injuries. 16th Annual Meeting of the Society for Hospital Epidemiology of America (SHEA), Chicago, IL, March 18-21, 2006.

27. **Fisman DN**, Harris AD, Sorock GS, Rubin M, Daley K, Mittleman MA. Gloves reduce the risk of sharps related injuries in healthcare workers. Results from a case-crossover study. Annual Meeting of the Society for Epidemiological Research. Toronto, Ontario, Canada. June 27-30, 2005.

26. **Fisman DN**, Harris AD, Sorock GS, Rubin M, Mittleman MA. Fatigue Increases Sharps-Injury Risk in Medical Trainees: Results from a Case-Crossover Study. 132nd Annual Meeting of the American Public Health Association. Washington, DC. November 6-10, 2004.

25. **Fisman DN**, Bamberg W, Kirchner C, Kim A, Daley J, Alexander J, Zhang J, Abrutyn E. Female Gender Increases Risk of Graft Harvest Site Infection, but not Sternal Wound Infection, After Cardiac Bypass Grafting. 44th Interscience Congress on Antimicrobial Agents and Chemotherapy. Washington, DC. October 30-November 2, 2004.

24. Perencevich EN, Kaye KS, Strasbaugh LJ, Bradham DD, **Fisman DN**, Harris AD. Acceptable failure rates for antibiotic therapy of central venous catheter associated bacteremia. 26th Annual Meeting of the Society for Medical Decision Making. Atlanta, GA, October 17-20, 2004.

23. **Fisman DN**, Kirchner C, Daley J, Ambrose JF, Kim A, Alexander J, Zhang H, Abrutyn E. Risk Factor Analysis for Deep Sternal Wound Infections after Coronary Artery Bypass Grafting in Community Hospitals. 42nd Annual Meeting of the Infectious Disease Society of America Meeting, Boston MA, Sept 30-Oct 3, 2004.

22. **Fisman DN**, Kirchner C, Daley J, Ambrose JF, Kim A, Alexander J, Zhang H, Abrutyn E Risk Factors for Saphenous Vein Graft Harvest Site Infections after Cardiac Surgery and Impact of Infection on Outcome. 42nd Annual Meeting of the Infectious Disease Society of America Meeting, Boston MA, Sept 30-Oct 3, 2004.

21. **Fisman DN**, Lim S, Wellenius G, Britz P, Gaskins M, Newbern C. Rainfall Acutely Increases the Risk of Legionellosis in Philadelphia. 42nd Annual Meeting of the Infectious Disease Society of America Meeting, Boston MA, Sept 30-Oct 3, 2004.

20. **Fisman DN**, Kirchner C, Daley J, Kim A, Zhang H, Paris M, Alexander J, Abrutyn E. Deep sternal wound infection after coronary artery bypass grafting markedly increases hospital length of stay: Estimates from community hospitals. Association of Professionals in Infection Control 31st Annual Educational Conference and National Meeting. Phoenix, AZ, June 6-10, 2004.

19. Kirchner C, Abrutyn A, Jones I, **Fisman D**, Dhond A, Kim Y, Alexander J, Daley J, Zhang H, Kim A. Using a multi-center, computer-based surveillance system: Overcoming data collection challenges with the use of technology and creative teamwork. Association of Professionals in Infection Control 31st Annual Educational Conference and National Meeting. Phoenix, AZ, June 6-10, 2004.

18. **Fisman DN**, Wellenius G, Tsang L, Mittleman MA. Meteorological factors associated with occurrence of meningococcal disease: a novel use of case-crossover study design. Society for Healthcare Epidemiology of America Annual Meeting, Philadelphia, PA, April 17-20, 2004.

17. Bamberg W, **Fisman DN**, Kirchner C, Kim Y, Kim A, Daley J, Alexander J, Zhang H, Paris M, Abrutyn E. Risk factors for nosocomial pneumonia after coronary artery bypass grafting in community hospitals. Society for Healthcare Epidemiology of America Annual Meeting, Philadelphia, PA, April 17-20, 2004.

16. **Fisman DN,** Harris AD, Sorock GS, Mittleman MA. Characteristics of past unreported sharps-related injuries in healthcare workers. Society for Healthcare Epidemiology of America Annual Meeting, Philadelphia, PA, April 17-20, 2004.

15. **Fisman DN,** Harris AD, Sorock GS, Mittleman MA. Fatigue increases needlestick risk in medical trainees: results from a case-crossover study. Society for Healthcare Epidemiology of America Annual Meeting, Philadelphia, PA, April 17-20, 2004.

14. **Fisman DN**. Health-related quality of life and symptomatic genital herpes: Comparison of measures. CDC STD Prevention Meeting, Philadelphia PA, March 8-11, 2004.

13. Lynd L, Johnson-Masotti A, Sheehan D, **Fisman DN**. Anonymous HIV testing in Canada: A cost-effective health intervention. CDC STD Prevention Meeting, Philadelphia PA, March 8-11, 2004.

12. Lynd L, Johnson-Masotti A, Sheehan D, **Fisman DN.** Anonymous HIV testing in Canada: A cost-effective health intervention. CDC STD Prevention Meeting, Philadelphia PA, March 8-11, 2004.

11. **Fisman DN**, Goldie SJ, Hook EW, Lipsitch M. Dynamic projection of the effectiveness and cost-effectiveness of an HSV-2 vaccine for young women. International Society for STD Research 2003 Congress. Ottawa, Ontario, Canada. July 27-30, 2003.

10. Jang D, Chong S, Howard M, Smeija M, **Fisman D**, Chernesky M. Diagnosis of Chlamydia trachomatis (CT) infections in men and women by a new VIDAS Probe CT amplification assay performed on swabs and urines. International Society for STD Research 2003 Congress. Ottawa, Ontario, Canada. July 27-30, 2003.

9. **Fisman DN**, Lowry L. Cost-effectiveness of safer sharp medical devices for prevention of HIV infection in healthcare workers. 40th Annual Meeting of Infectious Diseases Society of America, Chicago, Ill October 24-27, 2002.

8. **Fisman DN**. Cost-effectiveness of post-exposure antibiotic prophylaxis in household contacts of individuals with severe invasive group A streptococcal disease. 40th Annual Meeting of Infectious Diseases Society of America, Chicago, Ill October 24-27, 2002.

7. **Fisman, DN**. Cost-effectiveness of safer sharp medical devices for prevention of bloodborne infection in healthcare workers. 24th Annual Meeting of the Society for Medical Decision Making Annual Meeting, Baltimore, MD, October 20–23, 2002

6. **Fisman DN**, Leder K. Age and efficacy of recombinant hepatitis B vaccination: a meta-analysis. 12th Annual Meeting, Society for Hospital Epidemiology of America, Salt Lake City, UT April 6-9, 2002.

5. **Fisman DN**, Harris AD, Sorock GS, Mittleman MA. Transient risk-factors for sharps-related injuries in healthcare workers. Society for Hospital Epidemiology of America Annual Meeting, Toronto, Ontario April 2001.

4. **Fisman DN**, Harris AD, Sorock GS, Mittleman MA. A pilot case-crossover study of sharps-related injuries in healthcare workers. National Occupational Injury Research Symposium, Pittsburgh, PA October 17 – 19, 2000.

3. **Fisman DN**, Freeman J, Lipsitch M, Goldie SJ. The future economic costs of the herpes simplex virus type 2 epidemic in the United States. Infectious Disease Society of America 38th Annual Meeting, New Orleans, LA September 7 – 10, 2000.

2. **Fisman DN**, Goldie SJ. Estimating the costs and benefits of screening monogamous, heterosexual couples for asymptomatic infection with herpes simplex virus type 2. Infectious Disease Society of America 38th Annual Meeting, New Orleans, LA September 7 – 10, 2000.

1. **Fisman DN**, Barlam TF, Dorman S, Holland S, O'Donnell MA. Risk factors for BCGosis in bladder cancer patients. Infectious Disease Society of America 37th Annual Meeting, Philadelphia, PA Fall, 1999.

G. TEACHING AND STUDENT SUPERVISION

Full graduate courses developed, in development, or substantially revised

University of Toronto

2020- **Decision Making in Communicable Disease Control (course code pending, course in development)**. This course, in development, is an advanced companion course to CHL5425, below. Students integrate skills in construction and parameterization of communicable disease models with principles of medical decision making and cost-effectiveness analysis through a series of case-based lectures and exercises. By the end of the course students should be capable of using dynamic infectious disease models as a platform for evaluating cost-effectiveness of communicable disease control strategies, in a manner that allows them to account for both direct costs of programs (e.g., vaccination costs, negative costs of cases averted directly through vaccination) and indirect costs (e.g., those associated with development of herd immunity, strain replacement, and changing average age at infection).

2019- **Health Trends and Surveillance (CHL5405):** I began teaching this course due to a colleague being on sabbatical in 2019 and have extensively modified the course so that it is now skills based, and covers topics including geographic information systems, time series analysis, analysis of complex survey data, and ethics of public health surveillance, not covered elsewhere in the MPH Epidemiology curriculum.

2016- **Epidemiologic Methods for Communicable Disease Control (CHL5432)**. With Prof. Paul Arora now teaching CHL5412, I have had the opportunity to focus on a skills-based course for intermediate level learners in communicable disease epidemiology. The course is built around cases that refer to specific infectious disease entities and challenges, including outbreaks of emerging infectious diseases. Topics covered include statistical methods for communicable diseases (Poisson regression, case-control methods and logistic regression, distributed lag models), social network analysis for sexually transmitted infections, an introduction to forecasting using both statistical and mathematical models, and parameterization and construction of epidemic models, ranging from single equation descriptive models to compartmental ODE models. Integration of molecular epidemiology and phylogenetics with standard epidemiological methods is also discussed.

2011-2015 **Epidemiology of Communicable Diseases (CHL5412H)**. This course represents an amalgam of two courses in communicable diseases previously taught at the Dalla Lana School of Public Health. Course co-director Dr. Amy Greer and I have totally reorganized and restructured the course, which now focuses on building quantitative and data management and analysis skills needed by frontline public health professionals and infectious disease epidemiologists. In the current year (2013) the course was co-taught with Effie Gournis of Toronto Public Health.

2010 -2015 **Epidemiology I: Introduction to Epidemiology (CHL5401H)**. Although an introductory epidemiology course with this number had been in existence previously, when I inherited this course in 2010 I revised and reorganized the course in its entirety.

The course now puts a major emphasis on the development of quantitative skills necessary for front-line public health practice. It is a core course for the MPH with Epidemiology concentration at Dalla Lana School of Public Health.

2010- **Mathematical Epidemiology of Infectious Diseases (CHL5425H)**. This is an intermediate level course on dynamic modeling of infectious diseases. I developed this new course in its entirety and am the sole instructor. This 36 hour course provides students with extensive instruction and hands-on experience with mathematical modeling as a tool for the study and control of communicable diseases.

2009 **Spatial Epidemiology and Infectious Disease Modeling (CHL 7001)**: Introduction to mathematical modeling and geospatial analysis in infectious diseases. This was a 10-week seminar course on the use of mathematical modeling, GIS, and spatial analysis for evaluation of disease epidemiology and disease control programs. This course was developed and taught by Dr. Gesink and myself.

Princeton University

2006 **Epidemiology (Public Affairs 598)**. This was a 12 week introductory course on epidemiologic measures and principles for students in the Wilson School Master of Public Affairs program. While PA598 had been taught previously, I completely redeveloped the course at Princeton during my year as a Visiting Assistant Professor at Princeton University. The course was very successful and the course version developed by myself is still in use at Princeton.

Drexel University

2004 **Infectious Diseases Epidemiology** (9 hour lecture/workshop block), Drexel University School of Public Health Epidemiology Concentration Seminar. I developed a series of lectures and exercises that introduced MPH students to core concepts in infectious disease epidemiology and public health communicable disease control.

2004 **Introduction to Epidemiology (Block III)**. This was an entirely new introductory epidemiology curriculum, developed by myself at Drexel University. The course included a series of lectures, graded and ungraded problem sets, computer exercises, and "journal clubs" for critical appraisal of the public health literature. The course also included an "evidence-based public health project" that introduced students to the concept of evidence-based clinical practice in public health.

Graduate courses taught*

**University of Toronto unless otherwise stated*.

2019- Health Trends and Surveillance (CHL5405). Sole instructor, 36 lecture/lab hours.

2019 Epidemiology II (CHL5402), with Prof. Arjumand Siddiqi (36 lecture/lab hours.)

2017-	Communicable Diseases Epidemiology Methods (alternate years) (CHL5432) 36 lecture/lab hours.
2015	University of Guelph, Infectious Disease Modeling (POPM 6950-02). 15 hours.
2015	McGill University, Infectious Disease Epidemiology (EPIB-615), 10 hours.
2013	Lecturer and Course Co-director, Communicable Disease Epidemiology, Prevention and Control (CHL5412H). 36 lecture/lab hours.
2012	Lecturer, Introduction to Public Health (CHL5004). 3 lecture/lab hours. Introduction to infectious disease epidemiology and outbreak investigation.
	Lecturer and Course Director, Introduction to Epidemiology and Public Health (CHL5401H). 36 lecture/lab hours.
	Lecturer and Course Co-director, Communicable Disease Epidemiology, Prevention and Control (CHL5412H). 36 lecture/lab hours.
2011	Lecturer, Introduction to Public Health (CHL5004). 3 lecture/lab hours. Introduction to infectious disease epidemiology and outbreak investigation.
	Lecturer and Course Co-director (with Dr. Amy Greer), Introduction to Communicable Disease Epidemiology (CHL5412H). 36 lecture/lab hours.
	Lecturer and Course Director, Introduction to Epidemiology/Epidemiology I (CHL5401H). 39 lecture/lab hours.
	Guest Lecturer, "Communicable disease surveillance and outbreak investigation". Health Trends and Surveillance (CHL5405H) (Profs. Lilian Yuan and Eric Holowaty, 3 lecture hours).
2010-	Lecturer and Course Director, Mathematical Epidemiology of Infectious Diseases (CHL5425H). 36 lecture/lab hours.
	Lecturer and Course Director, Introduction to Epidemiology/Epidemiology I (CHL5401H). 36 lecture/lab hours.
	Lecturer, CHL 5415F (Practice of Communicable Disease Epidemiology, Prevention and Control, Prof. Elizabeth Rea). Taught 3 two hour blocks (Vaccines I, Vaccines II, and Zoonotic Disease).
	Lecturer, CHL5416H (Environmental Epidemiology, Prof. Don Cole). 1 hour lecture (Global Climate Change and Infectious Diseases). November 30, 2009.
	Guest Lecturer, CHL5412H (Communicable Disease Epidemiology, Prevention and Control: Principles, Prof. Robert Remis). 3 hour lecture (Introduction to Mathematical Modeling). November 16, 2009.
	Co-instructor (with Dr. Reshma Amin): "Introduction to decision analysis", lecture/seminar (3 hours) , HAD 5301H. Department of Health Policy, Evaluation and Management. August 6, 2010.
2009	Co-instructor (with Dr. Andreas Laupacis): "Introduction to decision analysis", lecture/seminar (3 hours) , HAD 5301H. Department of Health Policy, Evaluation and Management. August 5, 2009.
	Co-instructor (with Dr. Matthew Stanbrook): "Introduction to test theory: diagnostic tests", lecture/seminar (3 hours), HAD 5301H. Department of Health Policy, Evaluation and Management. July 23, 2009.

Lecturer, Public Health Sciences CHL 5415F (Practice of Communicable Disease Epidemiology, Prevention and Control). Taught 3 two hour blocks (Vaccines I, Vaccines II, and Zoonotic Disease).

Tutor, HAD 5304H (Clinical Decision-Making and Cost-Effectiveness), Prof. Ahmed Bayoumi. Students: Drs. Kaede Ota and Darrell Tan, Pre-exposure antiretroviral prophylaxis for individuals at high risk of HIV infection.

Co-instructor (with Dr. Lawrence Paszat): "Non-experimental methods in epidemiology", lecture/seminar (3 hours), HAD 5301H. Department of Health Policy, Evaluation, and Management. July 28, 2008.

Co-instructor (with Dr. Gary Naglie): "Introduction to decision analysis", lecture/seminar (3 hours) , HAD 5301H. Department of Health Policy, Evaluation and Management. August 1, 2008.

Tutor, HAD 5304H (Clinical Decision-Making and Cost-Effectiveness), Prof. Ahmed Bayoumi. Student: Dr. Henry Ahn, Operative vs. conservative management of scoliosis in adolescent girls.

Lecturer, Public Health Sciences CHL 5415F (Practice of Communicable Disease Epidemiology, Prevention and Control). Taught 3 two hour blocks (Vaccines I, Vaccines II, and Zoonotic Disease).

2007 Lecturer, Health Policy, Management and Evaluation HAD 5301 H (Introduction to Clinical Epidemiology and Health Care Research). Taught two 3-hour blocks (Bias and Confounding, and Disease Frequency).

2006 Tutor, HAD 5304H (Clinical Decision-Making and Cost-Effectiveness), Prof. Ahmed Bayoumi. Student: Dr. Eyal Cohen, Cost-effectiveness of Strategies for the Management of Pediatric Empyema.

"Introduction to Test Theory and Screening". MI580 Principles of Epidemiology. January 26, 2006. Thomas Jefferson University, Philadelphia.

2005 "Introduction to Test Theory and Screening". MI580 Principles of Epidemiology. March 10 and May 25, 2005. Thomas Jefferson University, Philadelphia, PA.

Needlestick injuries and case-crossover study design". Infectious Disease Epidemiology Seminar (EP656), Center for Clinical Epidemiology and Biostatistics. March 15, 2005. University of Pennsylvania, Philadelphia, PA.

"Case-crossover study design". Advanced Epidemiology Methods Seminar (EP640), Center for Clinical Epidemiology and Biostatistics. March 2, 2005. University of Pennsylvania, Philadelphia, PA.

"Seroepidemiology". Infectious Disease Epidemiology Seminar (EP656), Center for Clinical Epidemiology and Biostatistics. March 2, 2005. University of Pennsylvania, Philadelphia, PA.

"Tick-Borne Infectious Diseases: A Review", Drexel University Infectious Disease Fellows Lecture. Hahnemann Hospital, Philadelphia, May 26, 2005.

Panelist, "Typhoid Mary: Villain or Victim?" (with Drs. Janet Fleetwood, Ed Mormon, and Steven Peitzman). Drexel University College of Medicine Medical Humanities Grand Rounds, May 24, 2005.

2004	Introduction to Infectious Diseases Epidemiology (Block I). Drexel University School of Public Health MD/MPH Program.
	Facilitator, Lab Instructor and Lecturer, Epidemiology and Biostatistics I (6 week introductory epidemiology and biostatistics course: 8-10 teaching hours/week), Drexel University School of Public Health.
	Facilitator, Lab Instructor/Leader and Lecturer, Epidemiology and Biostatistics II (7 week introductory epidemiology and biostatistics course: 8-10 teaching hours/week), Drexel University School of Public Health.
2003	Guest Lecturer, Clinical Health Sciences-Health Research Methods 789 (Health Economics for Health Care Managers), March 26, 2003. McMaster University, Hamilton, Ontario, Canada.
	Unit 6 Undergraduate Medicine (Obstetrics & Gynecology Clinical Clerkship)"Inroduction to Sexually Transmitted Diseases", McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada. Lecture given every 6 weeks.
2002	Department of Clinical Epidemiology and Biostatistics, Continuing Education Sessions. "The Burden of Genital Herpes in the United States: Estimation and Projection Using a Difference-Equation Model." McMaster University, Hamilton, Ontario, Canada. March 21, 2002.
	Tutor: Clinical Health Sciences-Health Research Methods 721 Period: October – November. McMaster University, Hamilton, Ontario, Canada.
	Preceptor: Unit 1 Undergraduate Medicine, Microbiology and Infectious Diseases. Period: September – October. McMaster University, Hamilton, Ontario, Canada.
	Tutor: Clinical Health Sciences-Health Research Methods 721. Period: July – August. McMaster University, Hamilton, Ontario, Canada.
	Unit 6 Undergraduate Medicine (Obstetrics & Gynecology Clinical Clerkship)"Inroduction to Sexually Transmitted Diseases", McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada. Lecture given every 6 weeks.
2001	Unit 6 Undergraduate Medicine (Obstetrics & Gynecology Clinical Clerkship)"Inroduction to Sexually Transmitted Diseases", McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada. Lecture given every 6 weeks.
2000	Teaching Assistant, "Mathematical Modeling of Infectious Diseases (EPI 260d)" Professor Marc Lipsitch, Harvard School of Public Health, Boston, MA.
	Teaching Assistant, "Decision Analysis in Clinical Research", Professor M.C. Weinstein, Summer Clinical Effectiveness Course, Harvard School of Public Health, Boston, MA.
1999	Teaching Assistant, "Decision Analysis for Health and Medical Practices" Professor S.J. Goldie, Harvard School of Public Health, Boston, MA.
	Teaching Assistant, "Decision Analysis in Clinical Research". Professor M.C. Weinstein, Summer Clinical Effectiveness Course. Harvard School of Public Health, Boston, MA

Professional, continuing education and faculty development training and workshops

[see also invited presentations]

2012 Infectious disease modeling course for public health epidemiologists. With Ashleigh Tuite. DLSPH, Toronto, Ontario, Canada, May 28 and June 4, 2012; Hamilton, Ontario, Canada, August 23, 2012; and Ottawa, Ontario, Canada, October 18, 2012; and Hamilton, Ontario, Canada,

2011 Invited participant and facilitator, Institute on Science for Global Policy, Emerging and Persistent Infectious Diseases: Focus on Mitigation. Edinburgh, Scotland, October 22-26, 2011.

Invited participant, Institute on Science for Global Policy, Emerging and Persistent Infectious Diseases: Focus on Prevention. La Jolla, California, June 5-8, 2011.

Infectious Disease Modeling: Beyond the Basics (Short Course). With Dr. Amy Greer and Ashleigh Tuite. North American Congress of Epidemiology, Montreal, Quebec. June 21-24, 2011.

2010 Introduction to Infectious Disease Modeling (with Dr. Amy Greer), DLSPH Summer Institute in Biostatistics, Toronto, Ontario, Canada June 9-11, 2010.

Health Policy, Management and Evaluation. 2010 Clinical Epidemiology Institute, planning committee member and faculty (5 x 1.5 hour critical appraisal sessions and 1 hour lecture on "Practical Prognostication: Introduction to Clinical Prediction Rules".)

Introduction to Infectious Disease Modeling (Short Course). With Dr. Amy Greer, Vicky Ng, and Ashleigh Tuite. 31st Annual Meeting of the Society for Medical Decision Making, Toronto, Ontario, Canada, October 24, 2010.

2009 "Epidemiology on the Fly: Infectious Disease Epidemiology and the Public Health Response to Novel Influenza A (H1N1)". Dalla Lana School of Public Health Research Seminar Series. July 3, 2009

"Climate Change and Infectious Diseases in North America: Bugs to Watch." Public Health Research Seminar Series, March 5, 2009.

"A wake up call? Links between fatigue, healthcare worker injury, and medical errors." Toronto General Hospital Clinical Epidemiology Rounds, March 30, 2009.

"Climate change and infectious diseases in North America: Bugs to watch." Faculty of Medicine Public Health Interest Group, March 10, 2009.

University of Toronto Division of Infectious Diseases: "Demoting the 'Captain of the Men of Death': Recent work on optimizing outcomes in community-acquired pneumonia". Departmental Rounds, March 3, 2009.

Health Policy, Management and Evaluation. 2009 Clinical Epidemiology Institute, planning committee member and faculty (5 x 1.5 hour critical appraisal sessions and 1 hour lecture on "Practical Prognostication: Introduction to Clinical Prediction Rules".)

"Systematic Review and Meta-Analysis" (2 hour lecture), December 1, 2009. Respiratory GREAT Network Training Program. This program, created by Dr. Teresa To, provides training in clinical epidemiology and biostatistics to international pediatric respirology trainees.

Introduction to Conjoint Analysis in Health Care (Short Course). With Vicky Ng. 31st Annual Meeting of the Society for Medical Decision Making, Hollywood, CA. October 18, 2009.

Advanced Topics in Infectious Diseases Modeling (Short Course). With Amy L. Greer. 31st Annual Meeting of the Society for Medical Decision Making, Hollywood, CA. October 18, 2009.

Organizer and Co-instructor (with Ms. Victoria Ng and Ms. Melanie Zahab): "Putting Public Health on the Map", an interactive workshop for public health epidemiologists. Canadian Society for Epidemiology and Biostatistics Annual Meeting, Ottawa, Ontario, Canada, May 25, 2009.

2008 "Climate Change and Infectious Diseases in Canada: A Challenge to Public Health and Healthcare" University of Toronto Environment and Health Seminar, November 20, 2008.

University of Toronto Community Medicine Program: "Introduction to Mathematical Modeling of Infectious Diseases", Biostatistical Methodology Unit short course on infectious diseases modeling. Presented basic elements of communicable disease models (including herd immunity and critical fraction, seasonality, model fitting, and modeling of antibiotic resistance) to 15 community medicine residents from University of Toronto and McMaster University. Two 3-hour sessions, October 10 and October 17, Hamilton and Toronto, Ontario.

Health Policy, Management and Evaluation. 2008 Clinical Epidemiology Institute, planning committee member and faculty (5 x 1.5 hour critical appraisal sessions and 1 hour lecture on "Practical Prognostication: Introduction to Clinical Prediction Rules".)

"Herd Immunity". Ontario Public Health Laboratory "Lab Rounds". March 6, 2008.

"Systematic Review and Meta-Analysis" (2 hour lecture), November 18, 2008. Respiratory GREAT Network Training Program. This program, created by Dr. Teresa To, provides training in clinical epidemiology and biostatistics to international pediatric respirology trainees.

"Introduction to Mathematical Modeling of Infectious Diseases", Biostatistical Methodology Unit short course on infectious diseases modeling. Presented basic elements of communicable disease models (including herd immunity and critical fraction, seasonality, model fitting, and modeling of antibiotic resistance) to 25 students from research and clinical infectious disease backgrounds. Two 3-hour sessions, July 7 and July 21, 2008.

"Making Best Bets: Mathematical Modeling as a Tool for Vaccine Policy". Pediatric Medicine Grand Rounds, January 16, 2008.

Judge, SickKids Student Summer Research Experience Research Day, July 24, 2008.

Speaker, Kids Science "Science Extravaganza" Program (science outreach to high-school students from high-risk backgrounds), Dr. Lisa Robinson, Director, May 8, 2008.

2007 Health Policy, Management and Evaluation. 2007 Clinical Epidemiology Institute, planning committee member and faculty (5 x 1.5 hour critical appraisal sessions).

University of Toronto Division of Infectious Diseases: Haygarth and Snow: Insights into "Emerging Infections" from the Pre-Microbiologic Era. Infectious Disease Fellows Retreat, August 23, 2007.

Judge, Health Policy, Management and Evaluation Student Research Day, May 2, 2007.

Judge, Department of Pediatrics Research Day, May 23 2007.

University of Toronto Division of Infectious Diseases: "Seasonality, Environment and Infectious Diseases". Departmental Rounds, April 3, 2007.

Introduction to Public Health Surveillance and Microbiology, in partnership with the Ontario Science School (tour and lab session for gifted 12th grade science students). Ontario Public Health Laboratory, May 30, 2007.

"Practical prognostication: a hands-on guide to clinical prediction rules". Pediatric Outcomes Research Team Rounds, December 13, 2007.

"Climate Change, Environment, and Infectious Diseases". (with Dr. Amy Greer, Vicky Ng-Brett, and Laura Kinlin). Child Health Evaluative Sciences Seminar Series, October 29, 2007

"Whooping it Up: The Apparent Resurgence of Pertussis in the Greater Toronto Area". Child Health Evaluative Sciences Seminar Series, September 10, 2007.

Coordinator, Biostatistical Methodology Unit Journal Club.

Judge, SickKids Student Summer Research Experience Research Day, July 18, 2007.

"Under Surveillance: How I Got Into Infectious Disease Epidemiology". Child Health Evaluative Sciences Outcomes Pillar meeting, May 15, 2007.

Sticky Situations: Needlesticks and their Implications for Patient Safety". SickKids Patient Safety Rounds, February 28, 2007.

Introduction to Infectious Disease Modeling (Short Course). With John Edmunds and Beate Sander, 29th Annual Meeting of the Society for Medical Decision Making, Pittsburgh, PA. October 20, 2007.

2006 "Seasonality of Infectious Diseases." Ontario Public Health Laboratory Seminar Series. December 14, 2006.

"There's a Bug in this Model: Transmission Modeling as a Tool for Epidemiology and Health Policy", Child Health Evaluative Sciences Seminar Series, Research Institute of the Hospital for Sick Children, December 4, 2006.

"There's a Bug in this Model: Transmission Modeling as a Tool for Epidemiology and Health Policy", Infectious Disease Division Research Rounds, November 22, 2006.

"Sneezonality: What we know (and don't) about seasonality of respiratory infections." Child Health Evaluative Sciences Seminar Series, Research Institute of the Hospital for Sick Children, October 23, 2006.

"Weather, Seasonality, and Communicable Disease Occurrence". Woodrow Wilson School (Princeton University) Science, Technology and the Environment Program (STEP) Seminar, February 20, 2006.

"SARS, Emerging Infectious Diseases, and the Basic Reproductive Number". Epidemiology of Infectious Diseases. February 13, 2006. Univ. Medicine and Dentistry of New Jersey School of Public Health, Piscataway, New Jersey.

2005 "The Economics of STD Control: Why Transmissibility Matters". Center for Health and Wellbeing Seminar Series, November 28, 2005.

Public Health Law and Infectious Diseases" (with Drs. John Culhane and Andy Newman). Current Concepts in Law and Medicine. Widener University Law School, Wilmington, Delaware

"SARS, Emerging Infectious Diseases, and the Basic Reproductive Number". Epidemiology of Infectious Diseases. April 25, 2005. Univ. Medicine and Dentistry of New Jersey School of Public Health, Piscataway, New Jersey.

2003 "SARS: Lessons learned (and already forgotten?)". Hahnemann Hospital SARS Planning Committee, Philadelphia, PA, December 17, 2003.

2004 Drexel University Math/Computer Science Seminar, April 12, 2004.
"Dynamic Projection of Effectiveness and Cost-Effectiveness of HSV-2 Vaccines for Young Women: How Good is Good Enough?"

"One in Five: Adventures in Genital Herpes". Drexel University School of Public Health Grand Rounds. February 19, 2004.

"SARS" Drexel University College of Medicine, Department of Medicine Hospital Infections Seminar. Hahnemann Hospital, Philadelphia, April 21, 2004.

"Directly Observed Therapy for HIV: A Useful Paradigm?" Infectious Disease Fellows Lecture Series. Hahnemann Hospital, Philadelphia, March 17, 2004.

Introduction to Infectious Disease Modeling (Short Course). With John Edmund 26th Annual Meeting of the Society for Medical Decision Making, Atlanta, GA. October 17, 2004.

2003 "Bring an umbrella and some penicillin: weather and invasive bacterial disease." Drexel University School of Public Health Research Friday Lunch Forum. October 31, 2003.

Hamilton Emergency Services Network. "Smallpox preparedness". Shalom Village, Hamilton, Ontario, Canada, March 28, 2003.

"Dynamic projection of the effectiveness and cost-effectiveness of an HSV-2 vaccine for young women". Hamilton Public Health Research, Education and Development (PHRED) "Share Symposium", Hamilton, Ontario, Canada, March 25, 2003.

McMaster University Regional Infectious Diseases Rounds, Division of Infectious Diseases. "Smallpox vaccination: risk vs. risk". March 13, 2003.

Hamilton Regional Emergency Medicine Rounds. "Smallpox vaccination: risk vs. risk". March 12, 2003.

Hamilton Regional Microbiology Research Day. "Dynamic projection of the effectiveness and cost-effectiveness of an HSV-2 vaccine for young women", St. Joseph's Healthcare Centre, February 27, 2003.

Department of Family Medicine, St. Joseph's Healthcare Centre, Hamilton, Ontario, Canada. "Public Health Update". March 14, 2003.

Center for Evaluation of Medicines Rounds. St. Joseph's Healthcare Centre, Hamilton, Ontario, Canada. "Invasive group A streptococcal infection: applying pharmacoeconomics to the 'flesh-eating disease'". February 11, 2003.

International Herpes Management Forum, "Strategies for Interrupting the Transmission of HSV" Workshop Participant, Seattle, Washington, May 4-6, 2003.

2002 "SARS in Toronto: the good, the bad, and the ugly." Drexel University College of Medicine Division of Infectious Diseases. Hahnemann Hospital, Philadelphia, November 6, 2003.

1-Day Workshop in Communicable Diseases for Emergency Service Workers. Hamilton,Ontario. November 19, 2002.

"Bloodborne Infectious Diseases: An Overview". City of Hamilton Police, Fire and Ambulance Designated Medical Officers, Hamilton, Ontario. February 19, 2002.

Department of Clinical Epidemiology and Biostatistics, Departmental Rounds, McMaster University, Hamilton, Ontario, Canada. "Case-crossover study of needlestick injury". March 14, 2002.

Regional Infectious Diseases Rounds, Division of Infectious Diseases. "Sharps-related injuries: identifying precipitants and measuring the costs of prevention". September 12, 2002.

Department of Family Medicine Rounds, McMaster University, Hamilton, Ontario, Canada. "Update in Sexually Transmitted Diseases, Part I". November 27, 2002

2001 "Bioterrorism". City of Hamilton Police, Fire and Ambulance Designated Medical Officers, Hamilton, Ontario. November 27, 2001.

"Bioterrorism". City of Hamilton Department of Social and Public Health Services, Hamilton, Ontario. November 9, 2001.

"Update in STDs". Infectious Diseases Fellows Academic ½ Day, Faculty of Health Sciences, McMaster University, Hamilton, Ontario. November 21, 2001.

"Bioterrorism". Infectious Diseases Fellows Academic ½ Day, Faculty of Health Sciences, McMaster University, Hamilton, Ontario. November 7, 2001.

Regional Infectious Diseases Rounds, McMaster University Division of Infectious Diseases. "Bioterrorism", October, 2001.

Clinical Preceptor, Hamilton Sexually Transmitted Diseases Clinic.

Economic Issues in Infectious Diseases Seminar, Harvard Center for Risk Analysis. "Prickly precipitants: a case-crossover study of sharps-related injuries in healthcare workers". Spring 2001, Harvard School of Public Health, Boston, MA.

Department of Medicine Special Rounds, St. Joseph's Healthcare Centre, Hamilton, Ontario, Canada.. "Bioterrorism: thinking about the unthinkable". November 13, 2001.

Seminar in Clinical Effectiveness. "Clinical and cost-effectiveness of 2 Clinical and cost-effectiveness of 2 management strategies for infected total hip arthroplasty in the elderly." Spring 2000. Harvard School of Public Health, Boston, MA.

Coordinator, Economic Issues in Infectious Diseases Seminar, Harvard Center for Risk Analysis

Seminar in Clinical Effectiveness. "Survival after percutaneous endoscopic gastrostomy". Spring 1999.

2000 Economic Issues in Infectious Diseases Seminar, Harvard Center for Risk Analysis. Estimating the costs and benefits of screening monogamous, heterosexual couples for asymptomatic infection with herpes simplex virus type 2. April 2000.

Centre for Outcomes and Policy Research Seminar. "Prickly precipitants: a case-crossover study of sharps-related injuries in healthcare workers." Dana-Farber Cancer Center, Boston, MA, USA.

Harvard Medical School Division of Infectious Diseases, City-wide Conference. "Clinical and cost-effectiveness of 2 Clinical and cost-effectiveness of 2 management strategies for infected total hip arthroplasty in the elderly.

Centre for Outcomes and Policy Research Seminar. The demise of genital herpes vaccines: using modeling to define 'Plan B'. Fall 2000. Dana-Farber Cancer Center, Boston, MA, USA.

1999 Visiting Physician ("Teaching Attending"), Department of Medicine, Beth Israel Deaconess Medical Centre, Boston, MA, April 1999.

Caregroup Center for Quality and Value, Beth Israel Deaconess Medical Centre, Boston, MA. "Management of the infected hip prosthesis".

Department of Orthopedics Departmental Conference, Beth Israel Deaconess Medical Centre, Boston, MA. Management of the infected hip prosthesis".

Department of Medicine, Beth Israel Deaconess Medical Centre, Boston, MA, Resident Lecture Series. "Fever of unknown origin"

Department of Obstetrics and Gynecology, Beth Israel Deaconess Medical Centre, Boston, MA, Resident Lecture Series. "Sexually transmitted diseases: an overview"

Department of Medicine, Beth Israel Deaconess Medical Centre, Boston, MA, Resident Journal Club. "An introduction to decision analysis".

Harvard Medical School Division of Infectious Diseases, City-wide Conference. "Infectious disease and hemophagocytosis."

1998 Harvard Medical School Division of Infectious Diseases, City-wide Conference. "Epiglottitis in the immunocompromised host".

1996 Department of Medicine Research Day, Royal Victoria Hospital, Montreal, Quebec. "Intrapleural placement of a nasogastric tube." Spring 1996.

Journal Clubs and Seminar Series

2011- Founder and Coordinator, FitzGerald Seminar Series in Communicable Disease Epidemiology, Dalla Lana School of Public Health. This series provides continuing education to public health and medical professionals on a variety of topics in communicable disease control and vaccinology. The seminar is webcast to participants across Canada. Past seminars are archived at: . http://www.dlsph.utoronto.ca/page/fitzgerald-seminars.

2008- Founder and Coordinator, Infectious Disease Epidemiology Afficionados (IDEA) Seminar Series, Fields Institute for Mathematics and Dalla Lana School of Public Health

2008-2010 Founder and Coordinator, Infectious Disease Epidemiology Afficionados (IDEA) Journal Club, Hospital for Sick Children.

2007-2008 Founder and Coordinator, Hospital for Sick Children Biostatistics Methodology Unit Journal Club.

2000-2001 Founder and Coordinator, Economic Issues in Infectious Diseases Seminar Series, Harvard Centre for Risk Analysis

Supervision of Trainees

1. Supervisor /Co-Supervisor

Post-doctoral Fellows

2023-2025 Tegan Mosugu, PhD. University of Toronto Institute for Pandemics, *Indoor air, equity and infectious diseases.*

2010-2012 Amy Hurford, PhD. Fields Institute for Research in Mathematical Sciences, University of Toronto. *Mathematical modeling of antimicrobial resistance in healthcare settings.* Co-supervisor with Dr. Jianhong Wu (York University).

2009-2011 Sharmistha Mishra, University of Toronto. Research mentor/co-supervisor (with Dr. M.C. Boily, Imperial College London)*, Division of Infectious Diseases, University of Toronto. Dr. Mishra obtained both a Commonwealth Scholarship and a Canadian Institutes for Health Research Fellowship in Public Health Sciences (2009-2011) ($60,000 per year).

2007-2009 Amy L. Greer, PhD. Hospital for Sick Children. Dr. Greer is a disease ecologist by training, and has joined our group to expand her expertise and understanding of *infectious diseases of humans*. Dr. Greer is a recipient of a SickKids Research Training Centre Travel Award (2008) ($960) and an Ontario Ministry of Innovation Post-doctoral Fellowship Award (2009) ($26,000).

Post-graduate Medical Trainees

2005 Wendy Bamberg, Drexel University School of Public Health, Philadelphia PA. *Risk factors for infection after cardiac surgery.* Infectious Disease Fellowship Research Supervisor.

2002-2003 Cheryl Main, McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada. *Community MRSA outbreak in a Canadian city jail.* Infectious Disease/Microbiology Fellowship Research Supervisor

Doctoral Students

2019-present Alison Simmons, *Epidemiology of invasive pneumococcal disease.* Co-supervisor with Prof. Ashleigh Tuite. Connaught International Scholarship to University of Toronto. https://www.alisonesimmons.com/

2020-present Afia Amoako, *The unequal landscape of COVID-19*. EPIC Doctoral Fellow, University of Toronto

2017-2022 Isha Berry, *Epidemiology of avian influenza at the human-animal interface in Bangladesh.* https://www.csih.org/fr/isha-berry. Vanier Scholarship, National Geographic Doctoral Award.

2017-present Jean-Paul Soucy, *Novel data analytic methods for infectious disease epidemiology.* Co-supervisor with Dr. Kevin Brown. Vanier Scholarship. https://jeanpaulsoucy.com/

2016-2020 Tiffany Fitzpatrick, *Epidemiology of pertussis in Ontario.* Co-supervisor with Dr. Astrid Gutmann. Canadian Immunization Research Network Award (to Dr. Fisman). Now Banting Post-doctoral fellow, Yale University (Prof. Alison Galvani).

2015-2019 Derek MacFadden, PhD Student (co-Supervisor), Harvard University School of Public Health. Co-supervisor with Dr. Bill Hannage. *Bioinformatic and geographic models for antimicrobial resistance.* Clinician-investigator, University of Ottawa.

2014-2016 Eva Wong, PhD Student (Supervisor), Dalla Lana School of Public Health, University of Toronto. *Climate change and infectious disease risk in Canada.* (Ms. Wong left the program without completing her degree).

2012-2015 Ashleigh Tuite, PhD Student (Supervisor), Institute of Medical Sciences, University of Toronto. *Mathematical modeling of syphilis control and "rebound".* Assistant Professor, Dalla Lana School of Public Health.

2012-2017 Ashleigh McGirr, PhD Student (Supervisor), Dalla Lana School of Public Health, University of Toronto. *Mathematical modeling of pertussis control strategies.* Pharmacoeconomics lead, GSK Vaccines.

2010-2014 Kevin Brown, PhD Student (Supervisor), Dalla Lana School of Public Health, University of Toronto. *Spatial and temporal patterns in Clostridium difficile outbreaks.* Committee: Allison McGeer, Nick Daneman, Rahim Moineddin. Kevin Brown received a CIHR Banting and Best Doctoral Award in 2010 ($105,000). Scientist, Public Health Ontario and Assistant Professor, Dalla Lana School of Public Health.

Medical Students

2010 Tanya Hauck, Faculty of Medicine, University of Toronto*. Pertussis epidemiology in Ontario*. Comprehensive Research Experience for Medical Students (CREMS) Supervisor.

Master's Students

2021 Kiera Murison MPH Student, Dalla Lana School of Public Health, Epidemiology. *SARS-CoV-2 in pregnancy.* Practicum Supervisor.

2021 Alicia Grima, MPH Student, Dalla Lana School of Public Health, Epidemiology. *Protective effects of SARS-CoV-2 vaccines.* Practicum Supervisor.

2019 Angie Salomon, MPH student, Dalla Lana Public Health Sciences, Epidemiology. *Growth characteristics of Western Hemisphere Chikungunya Epidemic.* Practicum Supervisor.

2015 Tahmina Nasserie, MPH student, Dalla Lana Public Health Sciences, Epidemiology. *Growth characteristics of Western Hemisphere Chikungunya Epidemic.* Practicum Supervisor.

2013 Sandy Bae, MPH student, Dalla Lana Public Health Sciences, Epidemiology. *Impact of El Nino Southern Oscillation on Infectious Disease Hospitalization Risk in the United States, and Implications for Climate Change.* Practicum Supervisor.

2011-2012 Gregory Kujbida, MPH student, Dalla Lana Public Health Sciences, Epidemiology. *Incorporating fine scale water quality and case data for modeling cholera in Haiti.* Practicum Supervisor.

2010-2011 Ruth Campbell, MSc (Supervisor), Health Policy, Management and Evaluation. *The experiences of immigrants seeking healthcare in Toronto*. Committee: Brian Hodges, Angela Robertson.

2010-2011 Christina Chan, MPH student, Dalla Lana Public Health Sciences, Epidemiology.

Latent class analysis for STD test methods. Practicum Supervisor.

2009 Laura Kinlin, MPH, University of Toronto*. Sharps injuries in healthcare workers; Prediction of pneumonia after cardiac surgery*. SickKids Summer Student Research Experience.

2009 Ashleigh Tuite, MSc. University of Toronto. *Influenza modeling*. SickKids Summer Student Research Experience.

2008-2009 Laura Kinlin, MPH, University of Toronto*. Epidemiology of invasive meningococcal disease in Ontario, Sydney, Australia, and London, England.* Public Health Sciences Master's Student Practicum Supervisor

2008-2009 Ashleigh Tuite, MSc, University of Toronto. *Biases in discordant couples study designs.* Public Health Sciences Master's Student Practicum Supervisor

2005 Rory Gagan, Drexel University School of Public Health, Philadelphia PA. *Hospital-acquired pneumonia after coronary artery bypass grafting: attributable mortality and length of stay*. MPH Thesis Supervisor

2005 Kimberly Spaude, Drexel University School of Public Health, Philadelphia PA. *Prior influenza vaccination and mortality among individuals hospitalized with community-acquired pneumonia.* MPH Thesis Supervisor

2005 Oumar H. Gaye, Drexel University School of Public Health, Philadelphia PA. *Epidemiology of Lyme disease in Philadelphia.* MPH Thesis Supervisor

2005 Joseph Noorigian, Drexel University School of Public Health, Philadelphia PA. *Risk factors for falling in Parkinson's disease*. MPH Thesis Supervisor

2004 John F. Ambrose, Drexel University School of Public Health, Philadelphia PA. *Risk factors for sternal wound infection after coronary artery bypass grafting.* MPH Thesis Supervisor

Undergraduate Students

2022 Amy Peng, BSc, Western University. *Relative performance of Canada and four high-income peers during the SARS-CoV-2 pandemic.*

2009 Caitlin McCabe, BSc. University of Toronto. *Directly observed therapy in women with HIV infection.* SickKids Summer Student Research Experience.

2007 Laura Kinlin, MPH. University of Toronto. *Environmental influences on invasive meningococcal disease in Philadelphia.* SickKids Summer Student Research Experience.

2008 Alexander White. *Environmental influences on campylobacteriosis in Philadelphia.* SickKids Summer Student Research Experience

2008 Caitlin McCabe, BSc. University of Toronto. *Compliance with antibiotic treatment guidelines and mortality in community acquired pneumonia.* SickKids Summer Student Research Experience.

2008 Stephanie Ross, BSc, University of Toronto. *Systematic review and meta-analysis of relative risk of cervical cancer in Indigenous women in Australia, Canada, New Zealand and the United states.* SickKids Summer Student Research Experience.

2008 Jennifer Ku, Applied Health Sciences Co-op Program, University of Waterloo. *Systematic review of factors influencing vaccine acceptance by healthcare workers*. Co-op Placement Supervisor.

2007 Alexander White. *Environmental influences on invasive pneumococcal disease in Philadelphia*. SickKids Summer Student Research Experience.

2001 Louisa Lowry, McMaster University Faculty of Health Sciences, Hamilton, Ontario, Canada . *Cost-effectiveness of safer sharps devices*. Supervisor: Student Research, Health Sciences 4L02 (Research Practicum).

Other Supervisory Activities

2009-2010 Ashleigh Tuite, MHSc. University of Toronto. *Modeling of vaccination strategies for pandemic H1N1 influenza*. MITACS Accelerate program (in conjunction with Ontario Agency for Health Protection and Promotion).

2009-2010 Beate Sander, University of Toronto. *Health economic aspects of pandemic mitigation.* Strategic Training Initiative in Health Research—Health Policy, Dalla Lana School of Public Health and MITACS Accelerate program (in conjunction with Ontario Agency for Health Protection and Promotion).

Note: The MITACS Accelerate program is an internship experience that fosters the integration of students in applied mathematics and other quantitative backgrounds into the corporate or public sector workplace.

2009-2010 Yanyu Xiao, University of Western Ontario. Mathematical modeling of H1N1 influenza in remote First Nations. CIHR Pan-Canadian Decision-Making Support Network for Pandemic Preparedness (CanPan) internship program.

2009-2010 Venkata Duvvuri, York Unversity. Conserved epitopes and cellular immunity as determinants of the epidemiology of the 2009 influenza pandemic. CIHR Pan-Canadian Decision-Making Support Network for Pandemic Preparedness (CanPan) internship program.

Note: The CanPan program was a national training effort aimed at fostering mathematical modeling expertise as part of Canada's response to the 2009 influenza pandemic. It was supported by the Canadian Institutes for Health Research. Additional information is available at https://canpan.ca/.

2. Committee member

PhD students

2008-2011 David Vickers, PhD candidate in interdisciplinary studies, University of Saskatchewan. *Epidemiology and immunology of Chlamydia trachomatis*. (Chair Dr. Nathaniel Osgood). Doctoral Thesis Committee.

2008- Paul Arora, PhD candidate in Epidemiology, University of Toronto. *Sexually transmitted disease risk in India.* (Chair Dr. Prabhat Jha). Doctoral Thesis Committee.

2008- Andrea Stachon MD, PhD candidate, University of Toronto Institute of Medical Sciences, Department of Psychiatry. *Gene expression and psychosis risk in 22q11 microdeletion syndrome*. (Chair Dr. Kathy Siminovich). Doctoral Thesis Committee Member

Master's students

2007-2010 Kaede Ota, MD, Health Policy, Management and Evaluation, University of Toronto. *Epidemiology of Antimicrobial Resistant Gonorrhea in Greater Toronto*. (Chair Dr. Sharon Walmsley). Master's Thesis Committee Member.

2008-2010 Elizabeth Brown, Laboratory Medicine and Pathobiology, University of Toronto. *Characterization of the epidemiology and microbiology of blastomycosis in Ontario.* (Chair Dr. Susan Richardson). Master's Thesis Committee Member.

H. UNIVERSITY SERVICE

Faculty Responsibilities

2016- Promotion and Tenure Committee, Dalla Lana School of Public Health

2013-2016 School of Public Health Faculty Representative
Clinician-Scientist Training Program Review
Faculty of Medicine, University of Toronto

2012- Faculty Council
Dalla Lana School of Public Health
University of Toronto

2011 Strategic Planning Steering Committee
Dalla Lana School of Public Health
University of Toronto

2011 Directorial Search Committee
Dalla Lana School of Public Health
University of Toronto

2004-2005 Continuing Medical Education Committee
Drexel University School of Public Health (Dr. Arthur Frank, Chair)

2002 - 2003 Local Planning Committee
Regional Training Centre in Health Services Research
McMaster University

1994 - 1996 Sciences Library Committee
McGill University

Departmental Responsibilities

2017- Head, Divison of Epidemiology, Dalla Lana School of Public Health, University of Toronto

2017- Member, Senior Administration Committee, Dalla Lana School of Public Health, University of Toronto

2017- Member, Dalla Lana School of Public Health Curriculum Committee

2014-15 Faculty Advisor (with Prof. Ross Upshur), Student Ebola Working Group

2011- Chair, Scientific Advisory Group for the FitzGerald Seminar Series on Communicable Diseases Control and Prevention

2011-2012 Member, Curriculum Review Committee

	Epidemiology Division, Dalla Lana School of Public Health University of Toronto
2010-present	Chair, Curriculum Review Committee Epidemiology Division, Dalla Lana School of Public Health University of Toronto
2009-	Admissions Committee Dalla Lana School of Public Health University of Toronto
2007-2012	Clinical Epidemiology Institute Planning Committee Department of Health Policy, Management and Evaluation University of Toronto
2004-5	Department of Epidemiology and Biostatistics Chair Search Committee Drexel University School of Public Health (Dr. Arthur Frank, Chair)
2003-2005	Admissions Committee Drexel University School of Public Health (Dr. Todi Villanueva, Chair)
2001	Pre-Medical Committee Member and Non-resident Tutor, Cabot House, Harvard College

This is Exhibit "B" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Global impact of the first year of COVID-19 vaccination: a mathematical modelling study

Oliver J Watson, Gregory Barnsley*, Jaspreet Toor, Alexandra B Hogan, Peter Winskill, Azra C Ghani*

Summary

Background The first COVID-19 vaccine outside a clinical trial setting was administered on Dec 8, 2020. To ensure global vaccine equity, vaccine targets were set by the COVID-19 Vaccines Global Access (COVAX) Facility and WHO. However, due to vaccine shortfalls, these targets were not achieved by the end of 2021. We aimed to quantify the global impact of the first year of COVID-19 vaccination programmes.

Methods A mathematical model of COVID-19 transmission and vaccination was separately fit to reported COVID-19 mortality and all-cause excess mortality in 185 countries and territories. The impact of COVID-19 vaccination programmes was determined by estimating the additional lives lost if no vaccines had been distributed. We also estimated the additional deaths that would have been averted had the vaccination coverage targets of 20% set by COVAX and 40% set by WHO been achieved by the end of 2021.

Findings Based on official reported COVID-19 deaths, we estimated that vaccinations prevented 14·4 million (95% credible interval [CrI] 13·7–15·9) deaths from COVID-19 in 185 countries and territories between Dec 8, 2020, and Dec 8, 2021. This estimate rose to 19·8 million (95% CrI 19·1–20·4) deaths from COVID-19 averted when we used excess deaths as an estimate of the true extent of the pandemic, representing a global reduction of 63% in total deaths (19·8 million of 31·4 million) during the first year of COVID-19 vaccination. In COVAX Advance Market Commitment countries, we estimated that 41% of excess mortality (7·4 million [95% CrI 6·8–7·7] of 17·9 million deaths) was averted. In low-income countries, we estimated that an additional 45% (95% CrI 42–49) of deaths could have been averted had the 20% vaccination coverage target set by COVAX been met by each country, and that an additional 111% (105–118) of deaths could have been averted had the 40% target set by WHO been met by each country by the end of 2021.

Interpretation COVID-19 vaccination has substantially altered the course of the pandemic, saving tens of millions of lives globally. However, inadequate access to vaccines in low-income countries has limited the impact in these settings, reinforcing the need for global vaccine equity and coverage.

Funding Schmidt Science Fellowship in partnership with the Rhodes Trust; WHO; UK Medical Research Council; Gavi, the Vaccine Alliance; Bill & Melinda Gates Foundation; National Institute for Health Research; and Community Jameel.

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See Comment page 1254

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Introduction

The first COVID-19 vaccine was delivered outside of a clinical trial setting on Dec 8, 2020.[1] By Dec 8, 2021, 55·9% of the global population was estimated to have received at least one dose of a COVID-19 vaccine, 45·5% estimated to have received two doses, and 4·3% estimated to have received a booster dose.[2] Despite the incredible speed with which COVID-19 vaccines were developed in 2020 and subsequently distributed during 2021, more than 3·5 million deaths due to COVID-19 have been reported globally since the first vaccine was administered.[2]

Understanding the global impact of vaccination on the course of the COVID-19 pandemic is challenging given the heterogeneous access to vaccines coupled with different levels of transmission and ongoing non-pharmaceutical interventions across countries. In the early months of 2021, the impact of vaccination would have been minimal because of the delay in developing the infrastructure for a widespread vaccination campaign, the need for a delayed two-dose regimen in some jurisdictions to ensure maximum protection,[3] and the delay in the development of antibodies following vaccination. Additionally, as vaccine supply was constrained, most countries opted to prioritise vaccination in high-risk populations, including health-care workers and older people. Such strategies would have generated direct protection but would have had comparatively less impact on SARS-CoV-2 transmission. However, from mid-2021 onwards those countries with access to plentiful vaccine supply opted for mass vaccination of the adult population, later including children and subsequent boosting to maintain high levels of protection given the waning in vaccine efficacy and the emergence of new variants of

Research in context

Evidence before this study

We searched PubMed up to April 26, 2022, without any date limits or language restrictions, using the search terms "vaccin* AND impact AND (death* OR live*) AND (estimat* OR evaluat*) AND (COVID-19 OR SARS-CoV-2)". We found eight published studies that estimated the impact of COVID-19 vaccination, including deaths averted from vaccination. None of the studies considered the global impact of COVID-19 vaccination, focusing instead on specific regions (Italy, California, North Carolina, Stockholm, subsets of states in the USA, New York City, and the WHO European Region). Furthermore, the study focusing on the WHO European region only quantified the direct impact of vaccination and did not estimate the indirect effects (ie, decreasing infection risk of both vaccinated and unvaccinated susceptible individuals).

Added value of this study

This mathematical modelling study advances previous work both in terms of scale (number of regions modelled) and in terms of quantifying both the direct and indirect impact of COVID-19 vaccination globally. We estimated the impact of vaccination up to Dec 8, 2021, by fitting COVID-19 transmission models to both reported deaths and excess mortality during the pandemic as a proxy for deaths due to COVID-19. This study is, to the best of our knowledge, the first to use excess mortality estimates in this way, allowing for the impact of COVID-19 vaccination to be estimated more accurately in countries with weaker surveillance systems.

Implications of all the available evidence

The results highlight the substantial impact that vaccination has had on the trajectory of the COVID-19 pandemic. They also illustrate the broader impact of COVID-19 vaccination in terms of allowing countries with high vaccine coverage to relax interventions. Furthermore, the findings highlight the importance of equitable access to vaccines, particularly in low-income regions, where substantially more lives could have been saved if the vaccination targets set out by the COVID-19 Vaccines Global Access (COVAX) Facility (20% coverage in COVAX Advance Market Commitment countries by the end of 2021) and WHO (40% coverage in each country by the end of 2021) had been reached.

concern. This approach has resulted in vast inequalities in global vaccine distribution.[4]

To reduce inequality, a fair allocation mechanism for COVID-19 vaccines was developed through the COVID-19 Vaccines Global Access (COVAX) facility, with a key target of achieving 20% vaccine coverage for the countries covered by its Advance Market Commitment (AMC) through COVAX-secured doses by the end of 2021.[5] WHO expanded this target by setting a global strategy to achieve 70% coverage in all countries by mid-2022, with an interim target of 40% coverage by the end of 2021.[7] However, as a result of numerous challenges, particularly the constrained vaccine supply to COVAX (exacerbated by some countries obtaining a greater proportion of the global vaccine supply, pharmaceutical companies not meeting their contractual obligations to COVAX, and unpredictable delays in supply including vaccines with brief expiry windows), these targets were not reached in many lower-middle-income countries and low-income countries.[6] Vaccine uptake has also been suboptimal in many countries because of vaccine hesitancy.[7] This considerable heterogeneity in vaccination coverage has resulted in continued reliance on non-pharmaceutical interventions for pandemic management in some countries[8] but concomitantly enabled other nations to relax interventions as a route out of the pandemic.[9]

Quantifying the impact of vaccination is further challenged by the incomplete picture of the COVID-19 pandemic that is obtained from reported deaths. In many countries, vital registration systems are incomplete and therefore only a fraction of deaths are routinely reported. However, even in countries with complete vital registration systems, it is difficult to accurately define the cause of death in individuals who present with multiple morbidities. Excess all-cause mortality (the difference between the observed and expected number of deaths in non-pandemic years) has therefore been used to quantify the impact of the COVID-19 pandemic.[10] Although the exact contribution of COVID-19 to excess mortality is unknown, the strong temporal correlation observed globally between reported COVID-19 mortality and excess mortality provides evidence that excess mortality is an informative indicator of pandemic-related mortality.[11] Robust vital registration systems do not exist in many parts of the world, with WHO estimating that 40% of global deaths that occurred in 2020 were unregistered,[12] and therefore data on excess mortality are not available for every country. Model-based estimates have therefore been developed to obtain a more complete estimate of the pandemic to date. One set of estimates produced by *The Economist* uses a range of socioeconomic and epidemiological data to infer excess mortality.[13] Although the precise estimates differ between research groups[14] and WHO,[15] they all suggest a substantially larger number of COVID-19 deaths than have been reported to date.

We aimed to quantify the global impact of the first year of COVID-19 vaccination and estimate the number of deaths from COVID-19 averted in 185 countries and territories, both from the direct protection of vaccinated individuals and from the indirect protection of all individuals living in vaccinated environments due to the reduction in risk of infection. Additionally, we aimed to quantify the impact that a more equitable global vaccination campaign, meeting the vaccination targets set by COVAX of 20% vaccination coverage of the eligible population by the end of 2021, could have had in COVAX

AMC countries. We also aimed to quantify the impact of achieving the WHO target of 40% coverage by the end of 2021 in all countries.

Methods

Transmission model fitting

For this mathematical modelling study, we used a previously published COVID-19 transmission model[16,17] and fitting framework[18] to obtain profiles of the COVID-19 pandemic in each country and thus estimate the counterfactual scenario in which vaccines are not delivered. Briefly, the model is a population-based, age-structured susceptible-exposed-infectious-recovered-susceptible (SEIRS) model, which explicitly captures disease severity, passage through different indicated health-care levels, and the roll-out of vaccination. We incorporated country-level data on demography, age-based mixing patterns, and health-care capacity. We fit the model to officially reported COVID-19 deaths in each country, resulting in an inferred time-varying level of transmission, R_t, denoting the mean number of secondary infections in the absence of both infection-induced and vaccine-derived immunity. By fitting directly to mortality, we indirectly captured the impact that non-pharmaceutical interventions have had over the course of the COVID-19 pandemic.

Vaccination rates for first and second doses in each country were taken from Our World in Data[2] and the WHO dashboard. We assumed a vaccination strategy that first targets those most at risk (including health-care workers) and then iteratively distributes vaccines in descending age order. Vaccination was assumed to confer protection against SARS-CoV-2 infection and the development of severe disease requiring hospital admission,[3] and to reduce transmission from vaccine breakthrough infections (ie, we assumed vaccinated individuals who develop infection would be less infectious than unvaccinated individuals).[19] We inferred vaccine efficacy for each country on the basis of vaccine types known to be predominantly used in each country. We explicitly modelled the emergence of the delta (B.1.617.2) variant and its impact on vaccine efficacy, hospital admissions, and immune escape.[20,21] Any epidemiological differences associated with previous variants were assumed to be reflected by their effects on mortality,[22] which were subsequently captured by the estimated R_t trend. We fit the model to COVID-19 mortality in a Bayesian framework using a Metropolis-Hastings Markov Chain Monte Carlo-based sampling scheme. We used the resulting fit to estimate the time-varying reproductive number, R_t, and its associated uncertainty.

Complete details of the model, vaccination, variants, and model fitting are given in the appendix (pp 2–10). No ethical concerns were noted for this study, with all mortality data used based on nationally aggregated statistics; all datasets used were publicly available.

Excess mortality and COVID-19 mortality data

Because of the heterogeneity in death registration and certification worldwide, we also fit the model to all-cause excess mortality. For countries and time periods for which excess mortality had not been reported, we used model-based estimates of all-cause excess mortality, first produced by *The Economist*.[13] More details of the methodology are given in the appendix (p 2). Given the wide uncertainty in these model-based estimates of excess mortality in many parts of the world, we also presented the deaths averted as estimated by fitting to official reported COVID-19 deaths from the Johns Hopkins University COVID-19 Data Repository (appendix p 2). Importantly, these estimates based on official reported COVID-19 deaths represent the lower bound of deaths averted at the global level due to the considerable levels of under-reporting of COVID-19 mortality documented worldwide.[23]

For the **Johns Hopkins University COVID-19 Data Repository** see https://coronavirus.jhu.edu/map.html

Estimating deaths averted due to vaccination

The first vaccination outside a clinical trial setting was given on Dec 8, 2020. We introduced vaccination from this point onwards in the model and explored the impact of the first year of vaccination up to Dec 8, 2021. To quantify the impact of vaccination and its associated uncertainty, we took 100 draws from the estimated distribution of R_t and vaccine efficacy estimates for each country and simulated a counterfactual scenario in which no vaccines are available and the epidemic in each country follows the same R_t trend since the start of the pandemic; a counterfactual in which vaccines are delivered but there are no indirect effects (ie, they do not reduce SARS-CoV-2 transmission); and the observed scenario in which vaccines were delivered at the rates reported. The third scenario generated an estimate of the trajectory of the epidemic for our fitted model and hence closely matched reported COVID-19 or excess deaths or estimated excess deaths in each country. We calculated the deaths averted as a result of vaccination by subtracting the estimated COVID-19 deaths from the simulation with vaccines included (the observed scenario) from the estimated COVID-19 deaths under the first counterfactual scenario. This process is illustrated in the appendix (p 18), which shows the estimated deaths averted for the USA. Because of the difficulty in predicting how governments and populations would have responded, and how viral evolution would have progressed if vaccines had not been available, we made no attempt to adjust the R_t trends for further non-pharmaceutical interventions, changes in mobility, or development of variants that probably would have occurred differently in the absence of vaccination. To explore the impact of key model parameters on estimates of deaths averted, we did additional sensitivity analyses. These included characterising the effects of the assumed relationship between the infection fatality ratio (IFR) and age (appendix p 10), as well as the assumed degree of immune evasion exhibited by the delta variant (appendix p 7).

For the **WHO dashboard** see https://covid19.who.int/

See **Online** for appendix

	Total COVID-19 deaths	Vaccination coverage (%)	Estimated deaths averted by vaccinations		
			Total	Per 10 000 people	Per 10 000 vaccines
Worldwide	5 469 000 (5 339 000–5 613 000)	38·30%	14 400 000 (13 650 000–15 900 000)	22·81 (21·63–25·18)	25·99 (24·64–28·69)
World Bank income group					
High-income countries	1 956 000 (1 892 000–2 032 000)	68·80%	6 353 000 (6 105 000–6 604 000)	52·6 (50·54–54·67)	36·67 (35·23–38·11)
Upper-middle-income countries	2 287 000 (2 220 000–2 355 000)	50·10%	2 914 000 (2 785 000–3 047 000)	25·6 (24·47–26·77)	23·36 (22·33–24·43)
Lower-middle-income countries	1 188 000 (1 099 000–1 302 000)	29·80%	5 083 000 (4 379 000–6 628 000)	15·27 (13·16–19·91)	20·39 (17·57–26·59)
Low-income countries	36 520 (33 390–40 410)	3·57%	20 380 (17 680–23 870)	0·3188 (0·2766–0·3733)	2·965 (2·572–3·472)
WHO region					
African region	153 800 (145 100–164 700)	5·48%	97 190 (88 420–107 400)	0·8677 (0·7894–0·9589)	5·958 (5·420–6·584)
Region of the Americas	2 492 000 (2 418 000–2 576 000)	58·30%	3 813 000 (3 624 000–3 987 000)	37·46 (35·6–39·17)	29·28 (27·83–30·62)
Eastern Mediterranean region	318 700 (307 200–331 500)	28·10%	639 200 (581 600–707 700)	8·746 (7·958–9·684)	13·50 (12·28–14·95)
European region	1 628 000 (1 589 000–1 673 000)	56·50%	4 334 000 (4 214 000–4 487 000)	46·77 (45·48–48·42)	39·52 (38·43–40·92)
South-East Asian region	713 800 (635 900–807 000)	35·40%	3 913 000 (3 234 000–5 491 000)	19·61 (16·21–27·52)	21·63 (17·88–30·36)
Western Pacific region	149 000 (120 100–234 400)	62·40%	1 574 000 (1 267 000–1 839 000)	30·14 (24·26–35·21)	22·58 (18·18–26·38)

Deaths averted are presented as medians with 95% credible intervals, with values also presented per 10 000 total population and per 10 000 vaccinations (first or second dose). Vaccination coverage is the proportion of the population with a full dose in the modelled countries by Dec 8, 2021. Total deaths are all modelled deaths in the presence of vaccinations when fitted to reported deaths from the start of the pandemic up to Dec 8, 2021.

Table 1: **Estimated deaths averted in the first year of COVID-19 vaccinations worldwide based on fits to officially reported COVID-19 deaths**

We also explored the impact of increasing vaccine distribution to meet WHO and COVAX targets. We modelled two scenarios in which the targets set by WHO to fully vaccinate 40% of the eligible population in each country and administrative region, and by COVAX to fully vaccinate 20% of the eligible population in AMC countries, by the end of 2021 had been reached. To do so, for countries in which these targets had not been met, we scaled the roll-out of vaccines across the year by a constant factor such that exactly the targeted amount of the population had received their second vaccine dose by our end date (Dec 8, 2021).

Statistical analysis

All analyses were done with R software (version 4.1.3), with all data, code, packages, and versions used available online at GitHub. This analysis covered 185 countries and territories with a population greater than 90 000 as reported in *World Population Prospects 2019*,[24] and that reported at least one death due to COVID-19 or 1 week of positive estimated excess mortality. We excluded China from our estimates because of its unique position as the origin of the detected epidemic and its large influence on estimates of deaths averted stemming from its population size.

Role of the funding source

The sponsors of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

Based on our model fit to officially reported COVID-19 deaths, we estimated that 18·1 million (95% credible interval [CrI] 17·4–19·7) deaths due to COVID-19 would have occurred without vaccinations worldwide during the first year of the COVID-19 vaccination programme (Dec 8, 2020, to Dec 8, 2021). Of these, we estimated that vaccination prevented 14·4 million (95% CrI 13·7–15·9) deaths due to COVID-19, representing a global reduction of 79% of deaths (14·4 million of 18·1 million) during the first year of COVID-19 vaccination (table 1). These estimates of vaccine impact do not account for the potential under-ascertainment of deaths related to COVID-19.

Using our model fit to predicted and reported excess mortality (appendix p 23), we estimated that 31·4 million (95% CrI 30·6–32·1) deaths due to COVID-19 would have occurred without vaccinations during the first year of COVID-19 vaccination, with 19·8 million (95% CrI 19·1–20·4) deaths averted, corresponding to 63% (19·8 million of 31·4 million) of total deaths (table 2). The difference between vaccine impact estimates based on excess mortality and official deaths due to COVID-19 was greatest in low-income regions, with approximately ten times more deaths estimated to have been averted in low-income countries when relying on excess mortality estimates (appendix pp 13, 19).

Using our model fit to excess mortality, we estimated that most deaths averted were due to the high levels of individual-level direct protection conferred by vaccination, with 79% (15·5 million of 19·8 million) of deaths averted through direct protection (figure 1A). Vaccine impact was also conferred through reducing the levels of burden placed on health-care systems, reducing the number of days that health-care capacity would have been exceeded and therefore contributing to an overall lower fatality rate from infection (appendix p 20). Throughout 2021, vaccine impact changed over time and space. Vaccine impact was

	Total excess deaths	Estimated deaths averted by vaccinations		
		Total	Per 10 000 people	Per 10 000 vaccines
Worldwide	17 990 000 (17 610 000–18 530 000)	19 810 000 (19 130 000–20 380 000)	31·21 (30·14–32·1)	35·68 (34·47–36·71)
World Bank income group				
High-income countries	2 503 000 (2 412 000–2 609 000)	8 004 000 (7 644 000–8 438 000)	66·18 (63·20–69·77)	46·14 (44·07–48·64)
Upper-middle-income countries	4 717 000 (4 611 000–4 827 000)	4 230 000 (4 051 000–4 384 000)	36·97 (35·40–38·31)	33·71 (32·28–34·94)
Lower-middle-income countries	9 688 000 (9 329 000–10 170 000)	7 401 000 (6 841 000–7 655 000)	22·23 (20·55–23·00)	29·69 (27·44–30·71)
Low-income countries	1 087 000 (1 068 000–1 106 000)	180 300 (171 400–188 900)	2·711 (2·576–2·840)	26·23 (24·93–27·48)
WHO region				
African region	1 614 000 (1 580 000–1 652 000)	466 400 (446 300–487 000)	4·164 (3·985–4·348)	28·59 (27·36–29·85)
Region of the Americas	3 354 000 (3 260 000–3 456 000)	4 469 000 (4 233 000–4 728 000)	43·89 (41·57–46·43)	34·31 (32·50–36·29)
Eastern Mediterranean region	2 310 000 (2 248 000–2 376 000)	992 800 (938 800–1 066 000)	13·58 (12·85–14·59)	20·97 (19·83–22·52)
European region	3 448 000 (3 347 000–3 568 000)	5 811 000 (5 551 000–6 187 000)	62·30 (59·51–66·33)	52·63 (50·28–56·04)
South-East Asian region	6 741 000 (6 398 000–7 247 000)	5 658 000 (5 114 000–5 858 000)	27·99 (25·3–28·98)	31·29 (28·28–32·39)
Western Pacific region	518 700 (489 200–547 800)	2 429 000 (2 266 000–2 617 000)	46·31 (43·21–49·91)	34·74 (32·42–37·44)

Deaths averted are presented as medians with 95% credible intervals, with values also presented per 10 000 total population and per 10 000 vaccinations (first or second dose). Total deaths are all modelled deaths in the presence of vaccinations when fitted to excess mortality from the start of the pandemic up to Dec 8, 2021.

Table 2: **Estimated deaths averted in the first year of COVID-19 vaccinations worldwide based on fits to excess mortality**

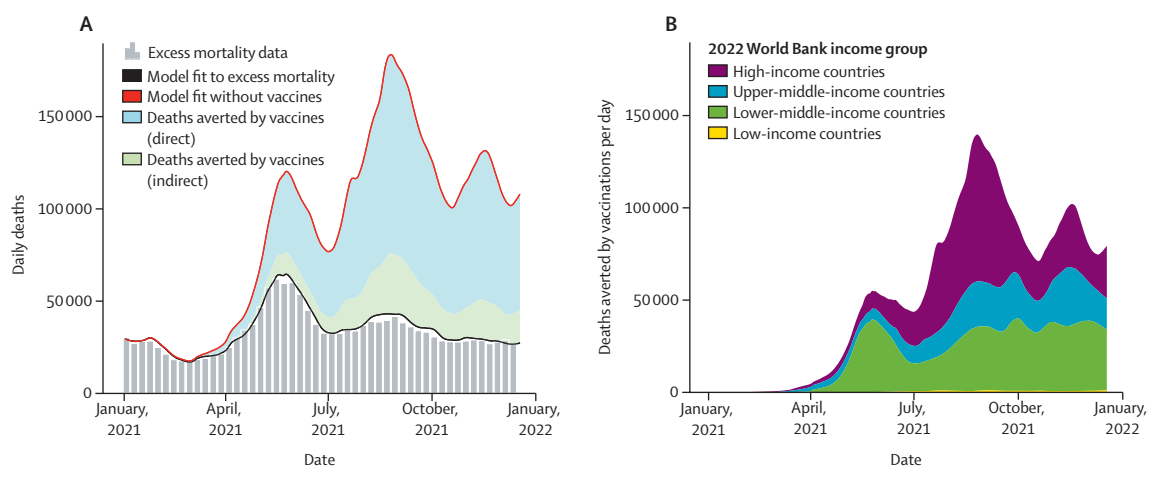

Figure 1: **Global COVID-19 deaths averted due to vaccination based on excess mortality**
(A) Median number of daily COVID-19 deaths based on excess mortality estimates (grey vertical bars) in the first year of vaccination. The baseline estimate of daily COVID-19 deaths from the model fit to excess mortality is plotted with the solid black line and the counterfactual scenario without vaccines is plotted with a red line. The gap between the red and black line indicates the deaths averted due to vaccination, with the proportion of total deaths averted by direct protection conferred by vaccination shown in blue and indirect protection shown in green. (B) Median number of daily deaths averted per day as per 2022 World Bank income group.

initially concentrated in lower-middle-income countries (figure 1B), resulting from the significant epidemic wave in India as the delta variant emerged. This was subsequently followed by vaccine impact being concentrated in high-income countries that were then either able to relax interventions due to high vaccination coverage (eg, the UK), or that did not implement further restrictions despite the spread of the more virulent delta variant in the second half of 2021.

Overall, estimated deaths averted per capita were highest in high-income countries, reflecting the earlier and wider roll-out of vaccination campaigns (table 2, figure 2; appendix p 13). We estimated that substantially more deaths were averted in the WHO European region. This was due to both the greater number of vaccinations administered in these regions and the higher levels of vaccine coverage achieved before the arrival of the delta variant.

The estimated number of deaths averted per vaccine administered was notably higher in high-income countries and upper-middle-income countries, in part due to greater access to the more efficacious mRNA vaccines (table 2;

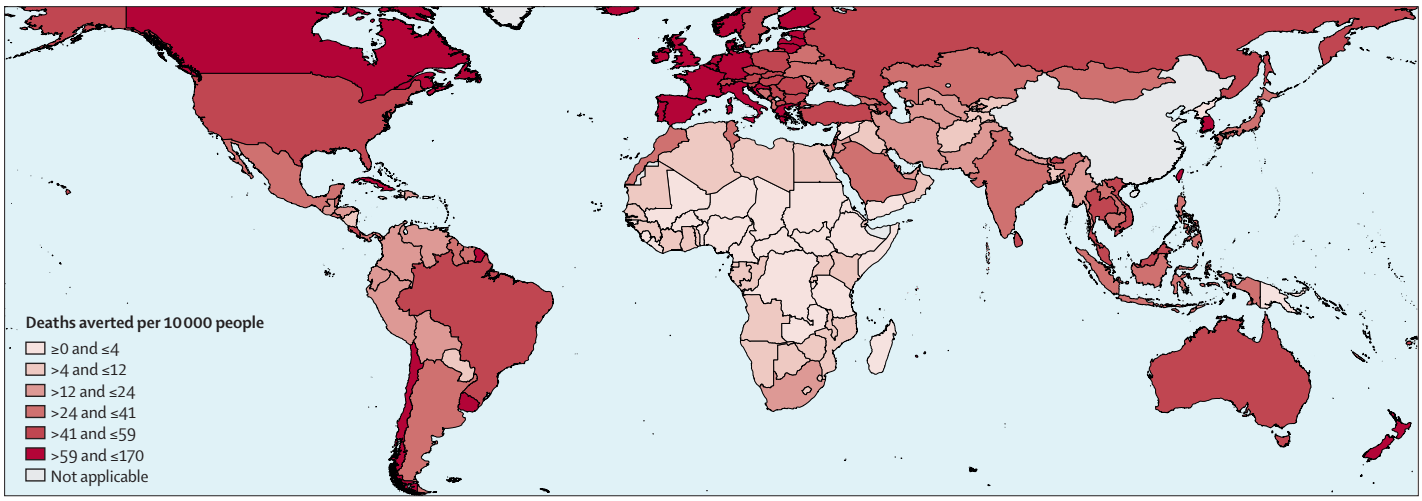

Figure 2: **Median deaths averted by vaccinations per 10 000 people by country in the first year of COVID-19 vaccination**
Estimates of deaths averted were based on model fits to excess mortality and were binned within seven equal quantiles starting at 0 deaths averted. Deaths averted listed as not applicable for China because of its exclusion from our analysis, due to its unique position as the origin of the detected epidemic and large influence on estimates of deaths averted stemming from its population size.

appendix p 14). Across the geographical regions, the estimated number of deaths averted per vaccine administered was estimated to be significantly higher in the European region and significantly lower in the Eastern Mediterranean region, reflecting disparities in access to different vaccine types (appendix p 14) coupled with very high predicted excess mortality in several countries in the Eastern Mediterranean region (table 2). The disparity in the number of deaths averted per vaccine between the European region and the Western Pacific region, despite access to similar vaccine types, reflects the zero-COVID strategy adopted by some countries in the Western Pacific region, such as New Zealand, which resulted in smaller epidemics predicted in the no-vaccine counterfactual (table 2; appendix p 13). Conversely, we estimated the greatest vaccine impact to have occurred in high-income countries that did not pursue a zero-COVID strategy (appendix p 13), reflecting how maximising vaccination coverage was leveraged to re-open the economy, resulting in increased transmission and subsequently higher inferred R_t trends. When viewed across income strata, a linear log–log relationship was observed between per-capita deaths averted and vaccines administered (figure 3), with low-income countries estimated to have a lower vaccine impact resulting from lower vaccine coverage. This relationship was weakest within high-income countries, as all high-income countries had high levels of vaccinations per capita, with the variation in deaths averted explained by other heterogeneities in their epidemics, such as pursuing zero-COVID strategies.

For the 83 COVAX AMC countries modelled, using our model fit to excess mortality, we estimated that 17·9 million (95% CrI 17·2–18·5) deaths due to COVID-19 would have occurred without vaccinations during the first year of COVID-19 vaccination. We estimated that vaccinations averted 7·4 million (95% CrI 6·8–7·7) deaths, 41% (7·4 million of 17·9 million) of the deaths that would have occurred in COVAX AMC countries. Notably, the shortfall of the COVAX target in several regions was estimated to have resulted in an additional 156 900 (95% CrI 147 800–165 400) deaths (table 3). Although these deaths constituted a small proportion of the total deaths averted globally, these avertable deaths were concentrated in 25 low-income countries, which we predict would have averted an additional 81 750 (95% CrI 75 430–88 200) deaths across low-income countries by reaching 20% coverage, representing an additional 45% of deaths averted (table 3).

We found that 96 countries and administrative regions were below the WHO target of 40% vaccination coverage by the end of 2021. Had this target been met, we estimated that 599 300 (95% CrI 577 700–622 400) additional deaths would have been averted (table 3). The majority of these deaths occurred in lower-middle-income countries and the African and Eastern Mediterranean regions, although the largest proportional increase was seen in low-income countries, with the averted deaths making up a 111% increase in estimated deaths averted by vaccinations (table 3).

Our vaccine impact estimates were dependent on the assumed level of immune escape shown by the delta variant and the assumed relationship between age and the IFR. In the scenario in which the epidemic wave caused by the delta variant was comparable to previous waves and neither reached herd immunity nor resulted in health-system capacity being breached, our estimates of vaccine impact were unchanged regardless of the assumed level of immune evasion associated with the delta variant (appendix p 21). However, in scenarios in which the introduction of the delta variant produced a significantly larger wave that resulted in herd immunity being reached in the counterfactual, increased immune escape associated with the delta variant resulted in an

increased number of averted deaths due to the larger effective size of the susceptible population. In sensitivity analyses in which the relationship between age and IFR was changed, we estimated that vaccine impact would be greater in scenarios with higher IFRs, reflecting the higher number of deaths that could be averted by vaccination (appendix p 22).

Discussion

The high individual-level protection against severe disease and mortality due to COVID-19, as well as the population-level benefit afforded by mild protection against SARS-CoV-2 infection (before the emergence of the omicron [B.1.1.529] variant), conferred by vaccination, has fundamentally altered the course of the COVID-19 pandemic. Directly measuring the impact of vaccination programmes on COVID-19 mortality is not possible as the counterfactual (ie, without vaccinations) cannot be observed. Mathematical models are a valuable tool for quantifying the impact of vaccination campaigns on epidemic dynamics.[25] We evaluated the impact of the first year of COVID-19 vaccination, revealing how vaccinations have more than halved the potential global death toll due to COVID-19, with an estimated 19·8 million deaths from COVID-19 averted as a result of vaccination, based on excess mortality estimates of the impact of the pandemic. These reductions were concentrated in high-income countries that relied on their vaccination programmes to relax interventions and allow SARS-CoV-2 transmission to increase as they moved into a new stage of the pandemic.

In low-income countries, particularly countries that did not reach the 20% targets set out by COVAX, vaccine impact was substantially lower, with vaccine impact estimated to have been almost doubled if the targets had been reached. If the 40% target, per country, from WHO had been met, we estimated a further increase in deaths averted, mainly focused in lower-middle-income countries and low-income countries. A limitation of our assessment of the COVAX and WHO targets is the timeframe of our analysis, as these targets were set to be reached by the end of 2021, whereas our modelling endpoint was Dec 8, 2021, to align with 1 year since the start of public vaccination. Hence, some countries might have moved closer to achieving the targets, or achieved them, by the end of the year. However, any recent vaccination drives would have had consequently negligible impact given the delay in developing protection and insufficient impact on COVID-19 dynamics.

Deriving estimates of vaccine impact is heavily dependent on the counterfactual scenario chosen. In our counterfactual, we assumed the same time-varying levels of SARS-CoV-2 transmission as estimated in our model fits. Consequently, the largest impact was observed in countries that delivered the most vaccinations to date and simultaneously relaxed interventions, allowing SARS-CoV-2 transmission to increase. However, several countries with slower vaccination roll-out as well as countries adopting a zero-COVID strategy maintained stronger interventions to suppress transmission and thus observed smaller impacts of their vaccination programmes as a result. As these countries start to reopen, we predict that vaccine impact estimates would increase in line with increasing levels of SARS-CoV-2 transmission.

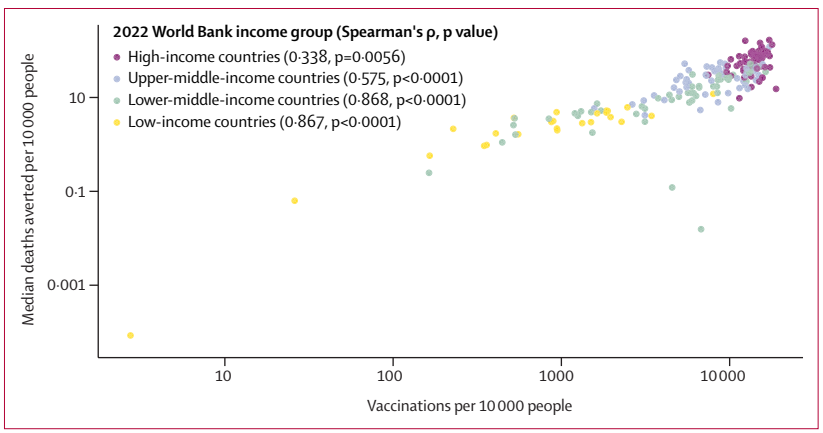

Figure 3: **Median deaths averted by vaccinations per 10 000 against vaccinations per 10 000 for each country**
All measures are on the log-scale. Spearman's rank correlation coefficient (Spearman's ρ) is also given for each income group with a p value based on the Z score against a null hypothesis of no correlation. Countries that did not deliver any vaccinations or had no deaths averted are not included.

Under-ascertainment of COVID-19 mortality is a known issue that has hindered our understanding of the pandemic.[23] In this analysis, we consequently focused on fitting to all-cause excess mortality, which provides a more complete description of the pandemic.[15] However, even when relying on model fits based on reported COVID-19 deaths, we estimated that more than 14 million deaths were averted by COVID-19 vaccination. The discrepancy between vaccine impact estimates based on excess mortality and COVID-19 deaths was concentrated in settings with lower death registration and certification. This substantial discrepancy underpins the crucial need for continued investment in civil registration and vital statistics to prevent biases in mortality reporting further minimising the perceived impact and necessity of vaccination in settings with lower reporting of deaths. In countries with more complete reporting systems, our estimates were broadly comparable to other endeavours focused on officially reported COVID-19 deaths and on understanding the direct impact of vaccination on people older than 60 years in Europe.[26] We identified one study that estimated both the indirect and direct impact of vaccination, which again yielded estimates for vaccine impact in the USA that were similar to our impact estimates based on reported COVID-19 deaths.[27]

In our effort to provide impact estimates globally, we introduced various assumptions into our model. We were hindered by the global disparities in SARS-CoV-2 genomic surveillance and the absence of detailed vaccination data for the majority of countries. Consequently, key model

	COVAX target (20% of eligible population in COVAX Advance Market Commitment countries fully dosed)				WHO target (40% of eligible population fully dosed)			
	Countries failing target	Increased vaccine coverage (%)	Additional deaths averted	Additional deaths averted* (%)	Countries failing target	Increased vaccine coverage (%)	Additional deaths averted	Additional deaths averted* (%)
Worldwide	41	4·15%	156 900 (147 800–165 400)	0·792% (0·744–0·843)	96	27·8%	599 300 (577 700–622 400)	3·03% (2·89–3·17)
World Bank income group								
High-income countries	..	..	..	..	1	0·00191%	20 (20–30)	0·000298% (0·000243–0·000342)
Upper-middle-income countries	..	..	..	..	27	6·1%	51 110 (47 860–66 690)	1·21% (1·12–1·58)
Lower-middle-income countries	16	4·28%	75 540 (68 640–80 380)	1·02% (0·923–1·13)	41	39·7%	347 500 (330 300–363 300)	4·71% (4·43–5·11)
Low-income countries	25	253%	81 750 (75 430–88 200)	45·2% (42·0–49·3)	27	1060%	200 000 (187 900–211 900)	111% (105–118)
WHO region								
African region	31	134%	132 700 (123 800–141 300)	28·4% (26·5–30·4)	44	631%	348 900 (330 200–370 000)	74·9% (70·7–78·8)
Region of the Americas	1	0·248%	1080 (850–1390)	0·0241% (0·0186–0·0308)	14	1·66%	6330 (5870–6840)	0·141% (0·129–0·155)
Eastern Mediterranean region	6	5·56%	20 850 (18 860–22 710)	2·09% (1·86–2·32)	13	61·6%	126 800 (118 900–134 600)	12·7% (11·6–13·7)
European region	..	..	..	..	13	3·05%	41 760 (38 110–46 160)	0·715% (0·644–0·799)
South-East Asian region	1	0·586%	1410 (50–2960)	0·0254% (0·000914–0·0532)	7	17·5%	70 420 (64 300–75 890)	1·25% (1·15–1·39)
African region	2	0·302%	900 (610–1200)	0·0366% (0·0250–0·0492)	5	2·59%	4990 (4390–5730)	0·205% (0·178–0·237)

Data are n (95% credible interval [CrI]) or % (95% CrI). All percentages are reported to 3 significant figures. Increased vaccination coverage is defined as the percentage increase in the proportion of the population with a full dose in all modelled countries when meeting the respective targets. Countries are grouped by 2022 World Bank income group and WHO region. COVAX=COVID-19 Vaccines Global Access.
*In proportion to total deaths averted by vaccines, as shown in table 2.

Table 3: **Estimated increase in deaths averted in the first year of COVID-19 vaccinations worldwide based on fits to excess mortality had all countries met either of the COVAX or WHO vaccination targets**

inputs had to be created from working assumptions on which vaccines were delivered, how they were delivered, and when new variants of concern spread worldwide. We also assumed that the relationship between age and IFR was the same for each country. These assumptions would have affected our estimates of deaths averted, with sensitivity analyses showing that higher overall IFRs will increase the number of deaths that could be averted by vaccination. Our impact estimates were also limited by the inherent uncertainty in model-based estimates of excess mortality.[13] These estimates are likely to have underestimated or overestimated COVID-19 death tolls in many countries. Notably, our model fits were unable to recreate excess mortality death tolls in recent epidemic waves in Iraq and Sudan because of the depletion of the susceptible population. These discrepancies could have been due to multiple reasons, including overestimated excess mortality, proportions of excess mortality not due to COVID-19,[28] higher infection fatality rates by age in low-income settings than those estimated from high-income countries,[29] and lower vaccine effectiveness than assumed in our framework. Last, our impact estimates were dependent on the assumed degree of immune escape that each variant of concern exhibits.[20] If immune escape was higher than we assumed, more of the population would have been susceptible to re-infection and consequently more deaths from COVID-19 could have been averted by vaccination.

More broadly, our estimates should be considered in light of the considerable uncertainty inherent in estimating vaccine impact. Uncertainty in the true death toll of the pandemic, the circulating variants of concern and their immunological phenotypes, and the vaccines themselves administered in many countries vastly complicate efforts to derive accurate estimates of the impact of COVID-19 vaccines. However, the results of this analysis still provide a comprehensive and thorough assessment of the impact of COVID-19 vaccination, revealing the substantial impact that vaccines have had and the millions of lives that are likely to have been saved during the first year of vaccination. Despite this, more lives could have been saved if vaccines had been distributed more rapidly to many parts of the world and if vaccine uptake could have been strengthened worldwide. Reaching vaccination coverage targets and improving vaccine coverage globally is dependent on multiple factors and not solely dependent on improving

vaccine donations.[30] Vaccine intellectual property needs to be shared more quickly in the future, with more open technology and knowledge transfer surrounding vaccine production and allocation. Vaccine distribution and delivery infrastructure also needs to be scaled up worldwide and misinformation combatted to improve vaccine demand. Improvements must be made in all these areas to reach current vaccine targets and help ensure that vaccines are more equitably distributed in the future.

Contributors

OJW and ACG conceived the study with input from GB, ABH, PW, and JT. OJW and GB led the model fitting and counterfactual simulation analyses for the estimation of deaths averted. OJW and GB produced the first draft of the manuscript and have accessed and verified the underlying data. All authors read, contributed to, and approved the final draft. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication.

Declaration of interests

ACG has received personal consultancy fees from HSBC, GlaxoSmithKline, and WHO related to COVID-19 epidemiology and from The Global Fund to Fight AIDS, Tuberculosis and Malaria for work unrelated to COVID-19. ACG is a non-remunerated member of scientific advisory boards for Moderna and the Coalition for Epidemic Preparedness. ABH and PW have received personal consultancy related to COVID-19 work from WHO. All other authors declare no competing interests.

Data sharing

All data, codes, and supplementary tables used and generated by this study are available in a GitHub repository (version 1.0.1) or the Zenodo open repository. All estimates of deaths averted from vaccination are available in the appendix (p 13).

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This is Exhibit "C" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Spike protein produced by vaccine not toxic

By BEATRICE DUPUY June 9, 2021

RELATED TOPICS

Fact-checking

CLAIM: COVID-19 vaccines make people produce a spike protein that is a toxin and can spread to other parts of the body and damage organs.

AP'S ASSESSMENT: False. COVID-19 vaccines instruct the body to produce spike proteins that teach the immune system to combat the spikes on the coronavirus, and experts say these proteins are not toxic.

THE FACTS: A recent radio interview with an Ontario professor is being shared widely online to mislead social media users about COVID-19 vaccines.

In an episode that aired in May, Canadian radio host Alex Pierson interviewed Dr. Byram Bridle, an associate professor in viral immunology at the University of Guelph's Ontario Veterinary College, about whether the vaccine was safe for children.

On the show, Bridle says that he is pro-vaccine, but goes on to discuss a fringe theory that the spike protein that the body produces from the vaccine is toxic and can damage certain organs.

"We made a big mistake. We didn't realize it until now, we thought the spike protein was a great target antigen. We never knew the spike protein itself was a toxin and was a pathogenic protein so by vaccinating people we are inadvertently inoculating them with a toxin," he says.

Though Bridle used the term "we" there is no indication that he was involved in any way in developing COVID-19 vaccines. Other scientists refute Bridle's characterization of the spike protein.

"The spike protein is immunogenic, meaning it causes an immune response, but it is not a toxin,"

said William Matchett, a vaccine researcher at the University of Minnesota Medical School.

All the vaccines that received emergency use authorization in the U.S. do not contain actual spike protein from COVID-19 or the live COVID-19 virus. The spike proteins that coat the coronavirus allow the virus to easily infect the human cell and replicate. However, the vaccine works by teaching the immune system to fight off the spike protein in the body and get rid of it.

Dr. Dan Kaul, an infectious disease expert at the University of Michigan, said that the vaccines have been proven safe and effective through clinical trials and the millions of people who have so far received the vaccines in the U.S.

"In terms of the spike protein itself being pathogenic in some way that's just simply not true," he said in response to Bridle's claims.

The Pfizer and Moderna vaccines rely on messenger RNA, often referred to as mRNA, that delivers a set of instructions to create that spike protein so your body can learn to identify and fight off the virus. The Johnson & Johnson is a vaccine that carries its genetic instructions for the spike protein through a modified adenovirus.

Posts online shared quotes of Bridle's interview to further push the false narrative that COVID-19 vaccines are dangerous and attack the body.

In the interview, Bridle says that the spike proteins generated by the vaccines don't stay in the shoulder muscle, but spread and are "causing so much damage in other parts of the bodies of the vaccinated." But Dr. Adam Ratner, a pediatric infectious disease specialist at NYU Langone Health, said that vaccines are mostly concentrated at the site of injection or the local lymph nodes.

"What was said in the radio show was completely inaccurate," Ratner said. "There is no spike protein in the vaccines first of all. The amounts that are made after the mRNA is injected are very small and it almost exclusively stays locally. It is nowhere near the amount he was talking about."

In the radio interview, Bridle mentions a study of 13 health care workers that he said confirmed that the spike in protein was found in their blood. But experts say they found nothing of concern from that same study, which was conducted by researchers at Brigham and Women's Hospital and

appeared in the journal Clinical Infectious Diseases in May.

Bridle left out key details of the study, which relied on an ultrasensitive detection tool, said Matchett, of University of Minnesota.

"The spike became undetectable by 14 days after the first dose of the vaccine," Matchett said of the study. "After the second dose, they could not detect the spike protein in the blood of any of the participants because the participants had all generated anti-spike antibodies."

Bridle also mentioned a Japanese study to support his claims about the spike protein. But the study, which is written in Japanese, does not look at spike proteins from the vaccine, Matchett said.

Bridle did not respond to requests for comment from The Associated Press. An auto-reply email from his account said that a more comprehensive report on his comments would soon be published.

"My answer to the question posed by the host was objective and founded on multiple reliable scientific sources," he said in the auto-reply.

—

This is part of The Associated Press' ongoing effort to fact-check misinformation that is shared widely online, including work with Facebook to identify and reduce the circulation of false stories on the platform.

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KATHERINE R. COSTIN

REUTERS FACT CHECK

JUNE 15, 2021 / 11:00 AM / UPDATED 2 YEARS AGO

Fact Check-No evidence spike proteins from COVID-19 vaccines are toxic

By Reuters Fact Check

There is no proof that spike proteins created in response to mRNA vaccines are harmful to the body, scientists have told Reuters.

The claim was made by immunologist Byram Bridle (here) in an interview on May 28 (here) with Canadian broadcaster Alex Pierson (here and here).

Bridle asked listeners to brace themselves for "scary" findings that he assured were "completely backed up by peer-reviewed scientific publications". He said: "We made a big mistake... we thought the spike protein was a great target antigen, (but) we never knew the spike protein itself was a toxin and a pathogenic protein."

He speculated that COVID-19 shots could lead to cardiovascular problems and infertility, because "by vaccinating people we are inadvertently inoculating them with a toxin" (timestamp 8.27).

The claim was repeated online (here , here , here), notably in an article by the Hal Turner radio show (here), a radio programme broadcast by the namesake's far-right political commentator.

Reuters presented the statement to experts at the Meedan Digital Health Lab (meedan.com/digital-health-lab), who responded: "So far, there is no scientific evidence

toxic or damaging our organs." (here)

Research shows that spike proteins (here) remain stuck to the cell surface around the injection site and do not travel to other parts of the body via the bloodstream, they added. The 1% of the vaccine that does reach the bloodstream is destroyed by liver enzymes.

Bridle said his findings were corroborated by "cutting-edge science" from Japan's Pharmaceuticals and Medical Devices Agency (PMDA) (www.pmda.go.jp/0017.html), which he allegedly obtained through a freedom of information request (timestamp 4.41).

Turner's website repeated the claim and linked this Japanese document as Bridle's source (here). The article provided no further context, but research conducted by Reuters showed that the chapter was taken from this document (here), which featured the words 'PFIZER CONFIDENTIAL' in the footer.

When Reuters presented the document to Pfizer, however, a spokesperson wrote in an email that the file is a Common Technical Document (CTD) unrelated to Bridle's claim.

Pharmaceutical companies are required to submit CTDs to regulatory authorities in the European Economic Area, Japan and the United States before medicines or vaccines can be approved (here). Pfizer submitted this CTD to be assessed by the PMDA before the shot was certified in February 2021 (here).

"We can confirm the document does not make any reference to spike proteins from the vaccine resulting in dangerous toxins that linger in the body – this claim is incorrect", the spokesperson said.

Rather, the document detailed early pharmacokinetic laboratory studies (here) that assessed how the vaccine moved through the bodies of mice and rats. The study found expected

were found in the vaccine.

Bridle also cited another study that - he claimed - found: “A spike protein in circulation in the blood of 11 of those 13 healthcare workers that had received the vaccine.” He said this was “clear cut evidence” that the vaccine leads to blood clots, bleeding, heart problems and brain damage (timestamp 6.18).

The study co-author, David Walt (here), denied this. “Bridle is taking our results and completely misinterpreting them,” he wrote in an email to Reuters.

Walt said the study (here) found tiny concentrations of the spike protein in the first five days following vaccination, which showed that the body was producing the protein as intended.

Crucially, these spike proteins declined in the subsequent weeks, and no spike proteins were detected after the second injection. This is because the individuals developed antibodies to remove the antigen from the bloodstream, creating an immune response exactly as the vaccine was designed to do.

The tiny quantities measured in the bloodstream of vaccinated people were not toxic, Walt explained. By contrast, people who catch the coronavirus and become infected with COVID-19 can develop high levels of the spike protein that can cause adverse effects.

He added: “The most important message is over 400 million doses of the mRNA vaccine have been administered with negligible serious consequences. It is incredibly safe.”

Scientists online also took to social media to counter Bridle’s claim. This includes pharmacologist Sabina Vohra-Miller (www.vohramillerfoundation.ca/), who produced a Twitter infographic explaining that spike proteins from the vaccine are harmless and do not impact infertility (here).

not accepting media engagements, but rejected a “libellous website” and “public smearing campaign” that resulted from his radio interview.

VERDICT

False. Experts say the tiny level of spike protein measured in the bloodstream of vaccinated people does not cause toxicity; rather, they show the vaccine is getting to work. The studies Bridle cited as proof of toxicity do not support his claims.

This article was produced by the Reuters Fact Check team. Read more about our fact-checking work here .

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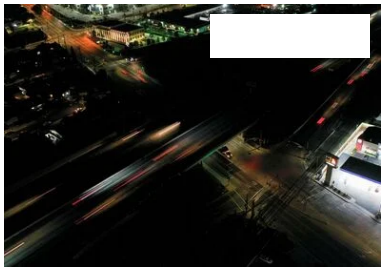

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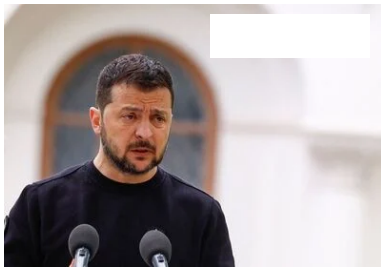

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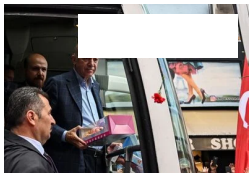

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KATHERINE R. COSTIN

RESEARCH ARTICLE SUMMARY

CORONAVIRUS

Estimated transmissibility and impact of SARS-CoV-2 lineage B.1.1.7 in England

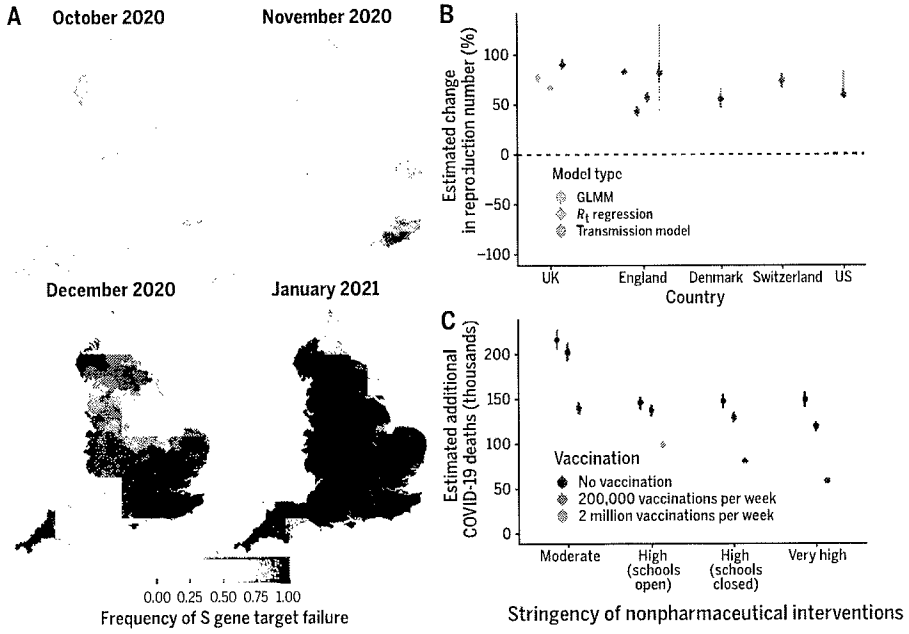
Nicholas G. Davies*, Sam Abbott†, Rosanna C. Barnard†, Christopher I. Jarvis†, Adam J. Kucharski†, James D. Munday†, Carl A. B. Pearson†, Timothy W. Russell†, Damien C. Tully†, Alex D. Washburne†, Tom Wenseleers†, Amy Gimma, William Waites, Kerry L. M. Wong, Kevin van Zandvoort, Justin D. Silverman, CMMID COVID-19 Working Group‡, COVID-19 Genomics UK (COG-UK) Consortium‡, Karla Diaz-Ordaz, Ruth Keogh, Rosalind M. Eggo, Sebastian Funk, Mark Jit, Katherine E. Atkins, W. John Edmunds

INTRODUCTION: Several novel variants of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus that causes COVID-19, emerged in late 2020. One of these, Variant of Concern (VOC) 202012/01 (lineage B.1.1.7), was first detected in southeast England in September 2020 and spread to become the dominant lineage in the United Kingdom in just a few months. B.1.1.7 has since spread to at least 114 countries worldwide.

RATIONALE: The rapid spread of VOC 202012/01 suggests that it transmits more efficiently from person to person than preexisting variants of SARS-CoV-2. This could lead to global surges in COVID-19 hospitalizations and deaths, so there is an urgent need to estimate how much more quickly VOC 202012/01 spreads, whether it is associated with greater or lesser severity of disease, and what control measures might be effective in mitigating its impact. We used social contact and mobility data, as well as demographic indicators linked to SARS-CoV-2 community testing data in England, to assess whether the spread of the new variant may be an artifact of higher baseline transmission rates in certain geographical areas or among specific demographic subpopulations. We then used a series of complementary statistical analyses and mathematical models to estimate the transmissibility of VOC 202012/01 across multiple datasets from the UK, Denmark, Switzerland, and the United States. Finally, we extended a mathematical model that has been extensively used to forecast COVID-19 dynamics in the UK to consider two competing SARS-CoV-2 lineages: VOC 202012/01 and preexisting variants. By fitting this model to a variety of data sources on infections, hospitalizations, and deaths across seven regions of England, we assessed different hypotheses for why the new variant appears to be spreading more quickly, estimated the severity of disease associated with the new variant, and evaluated control measures including vaccination and nonpharmaceutical interventions. Combining multiple lines of evidence allowed us to draw robust inferences.

RESULTS: The rapid spread of VOC 202012/01 is not an artifact of geographical differences in contact behavior and does not substantially differ by age, sex, or socioeconomic stratum. We estimate that the new variant has a 43 to 90% higher reproduction number (range of 95% credible intervals, 38 to 130%) than preexisting variants. Similar increases are observed in Denmark, Switzerland, and the United States. The most parsimonious explanation for this increase in the reproduction number is that people infected with VOC 202012/01 are more infectious than people infected with a preexisting variant, although there is also reasonable support for a longer infectious period and multiple mechanisms may be operating. Our estimates of severity are uncertain and are consistent with anything from a moderate decrease to a moderate increase in severity (e.g., 32% lower to 20% higher odds of death given infection). Nonetheless, our mathematical model, fitted to data up to 24 December 2020, predicted a large surge in COVID-19 cases and deaths in 2021, which has been borne out so far by the observed burden in England up to the end of March 2021. In the absence of stringent nonpharmaceutical interventions and an accelerated vaccine rollout, COVID-19 deaths in the first 6 months of 2021 were projected to exceed those in 2020 in England.

CONCLUSION: More than 98% of positive SARS-CoV-2 infections in England are now due to VOC 202012/01, and the spread of this new variant has led to a surge in COVID-19 cases and deaths. Other countries should prepare for potentially similar outcomes. ▩



Impact of SARS-CoV-2 Variant of Concern 202012/01. (**A**) Spread of VOC 202012/01 (lineage B.1.1.7) in England. (**B**) The estimated relative transmissibility of VOC 202012/01 (mean and 95% confidence interval) is similar across the United Kingdom as a whole, England, Denmark, Switzerland, and the United States. (**C**) Projected COVID-19 deaths (median and 95% confidence interval) in England, 15 December 2020 to 30 June 2021. Vaccine rollout and control measures help to mitigate the burden of VOC 202012/01.

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RESEARCH ARTICLE

CORONAVIRUS

Estimated transmissibility and impact of SARS-CoV-2 lineage B.1.1.7 in England

Nicholas G. Davies[1]*, Sam Abbott[1]†, Rosanna C. Barnard[1]†, Christopher I. Jarvis[1]†, Adam J. Kucharski[1]†, James D. Munday[1]†, Carl A. B. Pearson[1]†, Timothy W. Russell[1]†, Damien C. Tully[1]†, Alex D. Washburne[2]†, Tom Wenseleers[3]†, Amy Gimma[1], William Waites[1], Kerry L. M. Wong[1], Kevin van Zandvoort[1], Justin D. Silverman[4], CMMID COVID-19 Working Group[1]‡, COVID-19 Genomics UK (COG-UK) Consortium‡, Karla Diaz-Ordaz[5], Ruth Keogh[5], Rosalind M. Eggo[1], Sebastian Funk[1], Mark Jit[1], Katherine E. Atkins[1,6], W. John Edmunds[1]

A severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) variant, VOC 202012/01 (lineage B.1.1.7), emerged in southeast England in September 2020 and is rapidly spreading toward fixation. Using a variety of statistical and dynamic modeling approaches, we estimate that this variant has a 43 to 90% (range of 95% credible intervals, 38 to 130%) higher reproduction number than preexisting variants. A fitted two-strain dynamic transmission model shows that VOC 202012/01 will lead to large resurgences of COVID-19 cases. Without stringent control measures, including limited closure of educational institutions and a greatly accelerated vaccine rollout, COVID-19 hospitalizations and deaths across England in the first 6 months of 2021 were projected to exceed those in 2020. VOC 202012/01 has spread globally and exhibits a similar transmission increase (59 to 74%) in Denmark, Switzerland, and the United States.

In December 2020, evidence began to emerge that a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) variant, Variant of Concern 202012/01 (lineage B.1.1.7, henceforth VOC 202012/01), was rapidly outcompeting preexisting variants in southeast England (*1*). The variant increased in incidence during the second national lockdown in November 2020, which was mandated in response to a previous and unrelated surge in COVID-19 cases, and continued to spread after the lockdown despite ongoing restrictions in many of the most affected areas. Concern over this variant led the UK government to enact stronger restrictions in these regions on 20 December 2020 and eventually to impose a third national lockdown on 5 January 2021. As of 29 March 2021, VOC 202012/01 comprises roughly 95% of new SARS-CoV-2 infections in England and has now been identified in at least 114 countries (*2*). Our current understanding of effective pharmaceutical and nonpharmaceutical control of SARS-CoV-2 does not reflect the epidemiological and clinical characteristics of VOC 202012/01. Estimates of the growth rate, disease severity, and impact of this novel variant are crucial for informing rapid policy responses to this potential threat.

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†These authors contributed equally to this work. ‡See supplementary materials for list of consortium members and affiliations.

Characteristics of the new variant

VOC 202012/01 is defined by 17 mutations (14 nonsynonymous point mutations and three deletions), of which eight are in the spike protein, which mediates SARS-CoV-2 attachment and entry into human cells. At least three mutations potentially affect viral function. Mutation N501Y is a key contact residue in the receptor binding domain and enhances virus binding affinity to human angiotensin-converting enzyme 2 (ACE2) (*3*, *4*). Mutation P681H is immediately adjacent to the furin cleavage site in spike, a known region of importance for infection and transmission (*5*, *6*). Deletion ΔH69/ΔV70 in spike has arisen in multiple independent lineages of SARS-CoV-2, is linked to immune escape in immunocompromised patients, and enhances viral infectivity in vitro (*7*, *8*). This deletion is also responsible for certain commercial testing kits failing to detect the spike glycoprotein gene, and genomic data confirm that these S gene target failures in England are now overwhelmingly attributable to the new variant (*1*).

The proportion of COVID-19 cases attributable to VOC 202012/01 increased rapidly in all regions of England, following an initial expansion in the southeast (Fig. 1A), and spread at comparable rates among males and females and across age and socioeconomic strata (Fig. 1B). One potential explanation for the spread of VOC 202012/01 within England is a founder effect; that is, if certain regions had higher levels of transmission as a result of more social interactions, variants that were more prevalent within these regions could become more common overall. Changes in social contact patterns correlate closely with changes in transmission (*9*) (Fig. 1, C and D) and with COVID-19 burden in England (*10*). However, we did not find substantial differences in social interactions between regions of high and low VOC 202012/01 prevalence, as measured by Google mobility (*11*) and social contact survey data (*12*) from September to December 2020 (Fig. 1, E and F). Therefore, the apparent decoupling between contact rates and transmission in late 2020 may suggest altered transmission characteristics for VOC 202012/01.

Measuring the new variant's growth rate

VOC 202012/01 appears unmatched in its ability to outcompete other SARS-CoV-2 lineages in England. Analyzing the COG-UK dataset (*13*), which comprises more than 150,000 sequenced SARS-CoV-2 samples from across the UK, we found that the relative population growth rate of VOC 202012/01 in the first 31 days after its initial phylogenetic observation was higher than that of all 307 other lineages with enough observations to obtain reliable growth-rate estimates (Fig. 2A and fig. S1). Although the relative growth rate of VOC 202012/01 has declined slightly over time, it remains among the highest of any lineage as a function of lineage age (Fig. 2B), and the lineage continues to expand.

To quantify the growth advantage of VOC 202012/01, we performed a series of multinomial and logistic regression analyses on COG-UK data. A time-varying multinomial spline model estimates an increased growth rate for VOC 202012/01 of +0.104 day^{-1} [95% confidence interval (CI), 0.100 to 0.108] relative to the previously dominant lineage, B.1.177 (Table 1, model 1a; Fig. 2C; and figs. S2 and S3). Assuming a generation interval of 5.5 days (*14*), this corresponds to a 77% (95% CI, 73 to 81%) increase in the reproduction number R. The growth advantage of VOC 202012/01 persists under more conservative model assumptions (Table 1, model 1b; fig. S4), is consistent across all regions of the UK (table S1, model 2a; fig. S5), and is similar when measured from S gene target failures among community COVID-19 tests instead of COG-UK sequence data (Table 1, model 2h; fig. S6). Data from other countries yield similar results: We estimate that R for VOC 202012/01 relative to other lineages is 55% (95% CI, 45 to 66%) higher in Denmark, 74% (95% CI, 66 to 82%) higher in Switzerland, and 59% (95% CI, 56 to 63%) higher in the United States, with consistent rates of displacement across regions within each country (Table 1, models 3a to 3c; figs. S6 and S7).

As an alternative approach, we performed a regression analysis of reproduction numbers

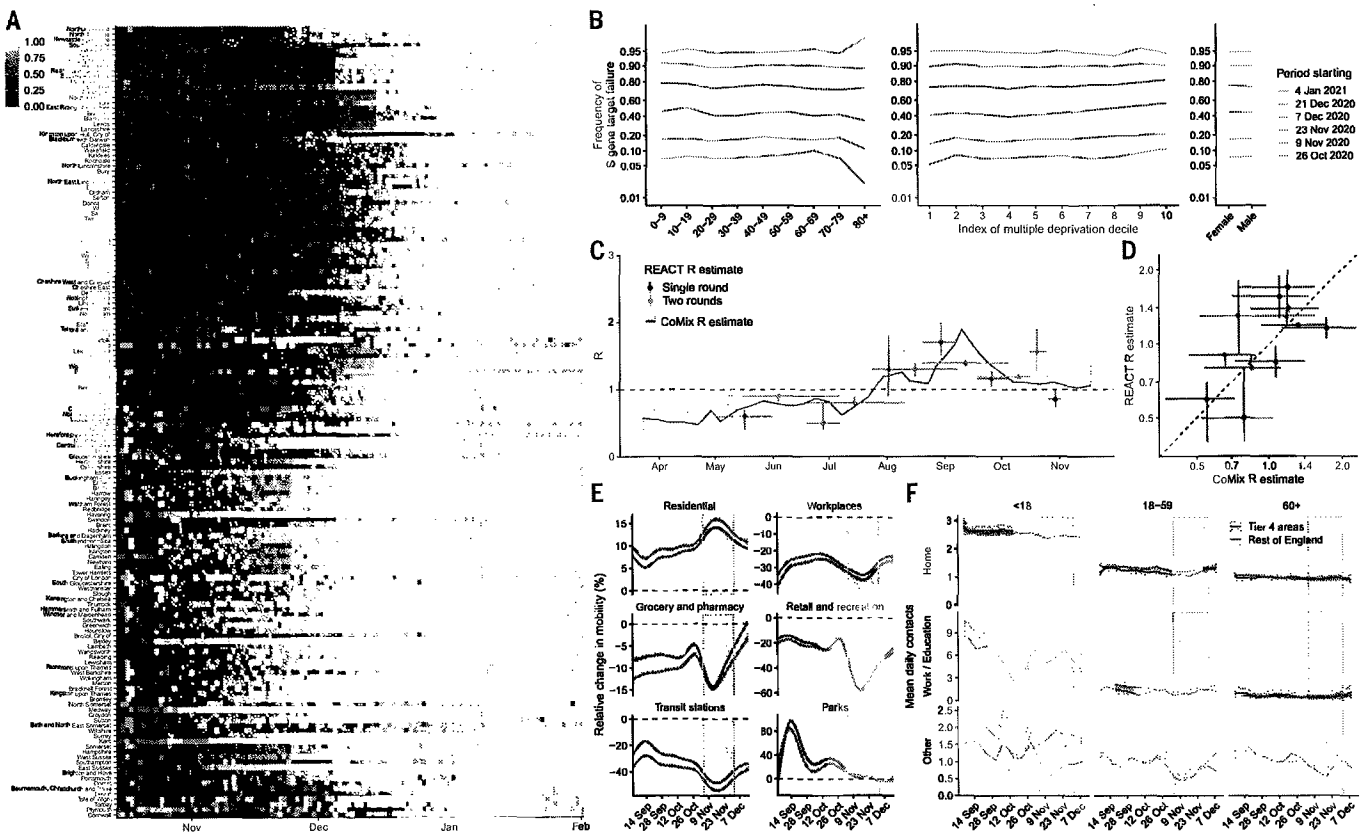

Fig. 1. Rapid spread of VOC 202012/01 in England. (**A**) Proportion of S gene target failure among positive Pillar 2 community SARS-CoV-2 tests in upper-tier local authorities of England from 1 October 2020 to 10 January 2021, sorted by latitude. (**B**) Spread of S gene target failure by age, index of multiple deprivation decile (1 = most deprived), and sex within Greater London. (**C** and **D**) Estimates of R_0 from CoMix social contact survey (*12*) compared to R_t estimates from REACT-1 prevalence survey (*9*) for England, with 90% CIs. R_t estimates based on single and aggregated REACT-1 survey rounds are shown. Horizontal error bars in (**C**) show the date range over which R_t was measured. (**E** and **F**) Percentage change (95% CI) in Google Mobility indices relative to baseline over time (**E**) and setting-specific mean contacts (95% CI) from the CoMix study (*12*) over time and by age for Tier 4 local authorities compared to the rest of England (**F**). Tier 4 local authorities are areas within the South East, East of England, and London regions that were placed under stringent restrictions from 20 December 2020 because of high prevalence of VOC 202012/01 and growing case rates. Gray shaded areas show the second national lockdown in England.

estimated from case data against the frequency of S gene target failure in English upper-tier local authorities (Fig. 2D), using local control policies and mobility data as covariates and including a time-varying spline to capture any unmeasured confounders. This yielded an estimated increase in R for VOC 202012/01 of 43% (95% CI, 38 to 48%), increasing to a 57% (95% CI, 52 to 62%) increase if the spline was not included (Table 1, models 4a and 4b). The various statistical models we fitted yield slightly different estimates for the growth rate of VOC 202012/01, reflecting different assumptions and model structures, but all identify a substantially increased growth rate (table S1).

Mechanistic hypotheses for the rapid spread

To understand possible biological mechanisms for the faster spread of VOC 202012/01 relative to preexisting variants, we extended an age-structured and regionally structured mathematical model of SARS-CoV-2 transmission (*10*, *15*) to consider two co-circulating variants (fig. S8 and tables S2 and S3). The model uses Google mobility data (*11*), validated by social contact surveys (*10*), to capture changes in contact patterns over time for each region of England. We created five versions of the model, each including one alternative parameter capturing a potential mechanism.

The hypotheses we tested are as follows. First, observations of lower cycle threshold (Ct) values (*16*–*18*)—that is, higher viral load—support the idea that VOC may be more transmissible per contact with an infectious person than preexisting variants (hypothesis 1). Second, longitudinal testing data (*17*) suggest that VOC may be associated with a longer period of viral shedding and hence a potentially longer infectious period (hypothesis 2). Third, the ΔH69/ΔV70 deletion in spike contributed to immune escape in an immunocompromised patient (*7*), which suggests that immunity to preexisting variants may afford reduced protection against infection with VOC (hypothesis 3). Fourth, the initial spread of VOC during the November 2020 lockdown in England, during which schools were open, suggests that children may be more susceptible to infection with VOC than with preexisting variants (hypothesis 4). Children are typically less susceptible to SARS-CoV-2 infection than adults (*19*, *20*), possibly because of immune cross-protection due to other human coronaviruses (*21*), which could be less protective against VOC. Finally, VOC could have a shorter generation time than preexisting variants (hypothesis 5). A shorter generation time could account for an increased growth rate without requiring a higher reproduction number, which would make control of VOC 202012/01 through social distancing measures relatively easier to achieve.

We fit each model to time series of COVID-19 deaths, hospital admissions, hospital and ICU bed occupancy, polymerase chain reaction (PCR) prevalence, seroprevalence, and the proportion of community SARS-CoV-2 tests with S gene target failure across the three most heavily affected

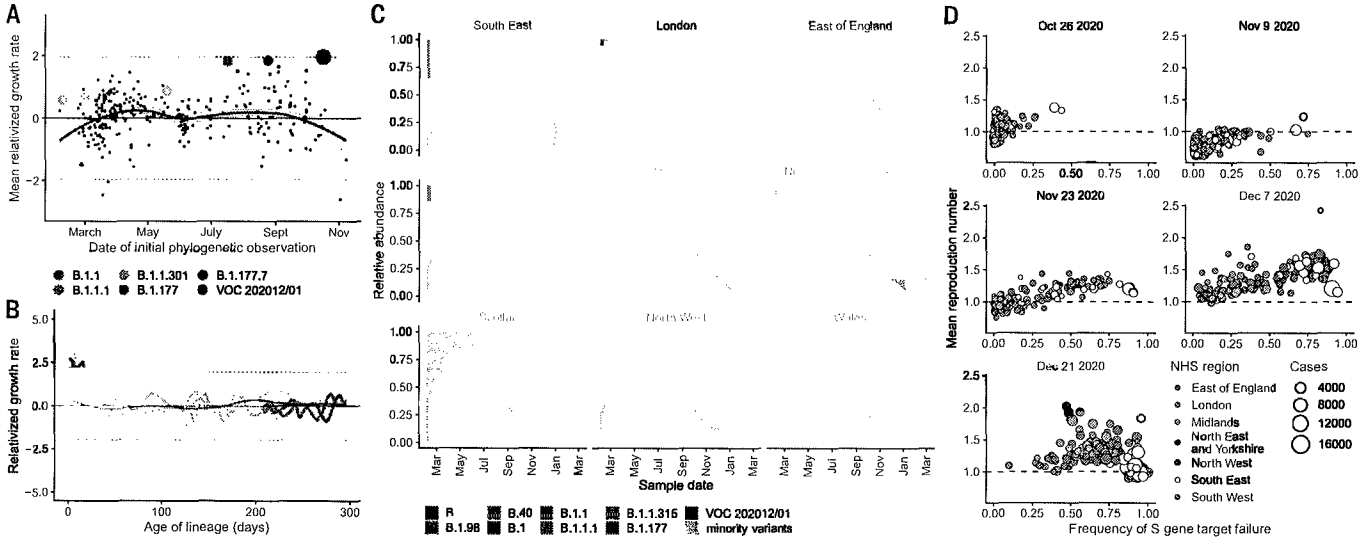

Fig. 2. Measuring the growth rate of VOC 202012/01. (**A**) Average relativized growth rate (i.e., a measure of variant fitness relative to other variants present during the 31 days after initial phylogenetic observation of a given variant) for all lineages in the COG-UK dataset, highlighting many lineages that have risen to prominence including B.1.177, the lineage with the highest relative abundance during the initial phylogenetic observation of VOC 202012/01. The shaded regions show conservative 95% rejection intervals; VOC 202012/01 is the first strain to exceed this threshold of faster relativized growth. Although many lineages exhibit above-average rates of growth, VOC 202012/01 has had the highest average relativized growth of any lineage in the history of COG-UK surveillance of SARS-COV-2. (**B**) Plotting all lineages' relativized growth rates [$\rho(t)$] as a function of lineage age with conservative 95% rejection intervals highlights the significantly faster growth of VOC 202012/01 relative to other lineages at comparable times since their initial observation. Later declines in VOC and B.1.177 correspond to highly uncertain estimates of growth rates for data that are yet to be backfilled, so these declines in $\rho(t)$ are sensitive to the processing of future sequences from recent dates (fig. S1). (**C**) Muller plots of the relative abundances of the major SARS-CoV-2 variants in the UK, based on a multinomial spline fit to COG-UK sequence data (Table 1 and table S1, separate-slopes multinomial spline model). A model extrapolation until 1 March 2021 is shown (shaded area). Minority variants are 440 circulating SARS-CoV-2 variants of low abundance. Specific colors represent the same lineages in (A) to (C). (**D**) Mean reproduction number over 7-day periods in 149 upper-tier local authorities of England (colored by the NHS England region they are within) plotted against the weekly proportion of Pillar 2 community SARS-CoV-2 tests with S gene target failure shows the spread of VOC 202012/01, a corresponding increase in the reproduction number in each local authority, and the eventual impact of targeted government restrictions from 20 December 2020. Testing data are shown for the week after the reproduction number estimates to account for delays from infection to test.

NHS England regions, over the period 1 March to 24 December 2020 (Fig. 3 and figs. S9 to S14). We assessed models using deviance information criteria (DIC) and compared model predictions to observed data for the 14 days after the fitting period (i.e., 25 December 2020–7 January 2021). Of the five hypotheses assessed, hypothesis 1 (increased transmissibility) had the lowest (i.e., best) combined DIC and predictive deviance. Hypothesis 2 (longer infectious period) and hypothesis 4 (increased susceptibility in children) also fitted the data well, although hypothesis 4 is not well supported by household secondary attack rate data (fig. S15) or by age-specific patterns of S gene target failure in the community (fig. S16), neither of which identify a substantial increase in susceptibility among children. Hypothesis 3 (immune escape) and hypothesis 5 (shorter generation time) fit poorly (Fig. 3A and table S4). In particular, hypothesis 5 predicted that the relative frequency of VOC 202012/01 should have dropped during stringent restrictions in late December 2020, because when two variants have the same effective reproduction number $R_t < 1$ but different generation times, infections decline faster for the variant with the shorter generation time.

We fitted a combined model incorporating the five hypotheses above, but it was not able to identify a single consistent mechanism across NHS England regions; hence, a wide range of parameter values are compatible with the observed growth rate of VOC 202012/01 (fig. S14). On the basis of our analysis, we identify increased transmissibility as the most parsimonious model, but we emphasize that the five mechanisms explored here are not mutually exclusive and may be operating in concert.

The increased transmissibility model does not identify a clear increase or decrease in the severity of disease associated with VOC 202012/01, finding similar odds of hospitalization given infection [odds ratio, 0.92; 95% credible interval (CrI), 0.77 to 1.10], critical illness [odds ratio, 0.90 (CrI, 0.58 to 1.40)], and death [odds ratio, 0.90 (0.68 to 1.20)] when the model was fitted to the three most heavily affected NHS England regions (Fig. 3B). These estimates should be treated with caution, as we would not expect to identify a clear signal of severity when fitting to data up to 24 December 2020, given delays between infection and hospitalization or death. However, the fitted model finds strong evidence of higher relative transmissibility, estimated at 65% (CrI, 39 to 93%) higher than preexisting variants for the three most heavily affected NHS England regions, or 82% (CrI, 43 to 130%) when estimated across all seven NHS England regions (Table 1, model 5a). These estimates of increased transmissibility are consistent with our statistical estimates and with a previous estimate of a 70% increased reproduction number for VOC 202012/01 (*16*). This model reproduces observed epidemiological dynamics for VOC 202012/01 (Fig. 3C and fig. S17). Without the introduction of a new variant with a higher growth rate, the model is unable to reproduce observed dynamics (Fig. 3, D and E, and figs. S17 to S19); these findings lend further support to the idea that changing contact patterns do not explain the spread of VOC 202012/01.

Implications for COVID-19 dynamics in England

Using the best-performing transmission model (increased transmissibility) fitted to all seven NHS England regions, we compared projected epidemic dynamics under different assumptions about control measures from mid-December 2020 to the end of June 2021. We compared four scenarios for nonpharmaceutical interventions

Table 1. Estimates of increased reproduction number for VOC 202012/01. Means and 95% CIs (GLMM) or 95% CrIs (R_t regression, transmission model) are shown. GLMM models do not estimate a baseline growth rate or reproduction number. Increases in the reproduction number assume a generation interval of 5.5 days. See table S1 for full details.

Model type	Model	Model assumptions	Data	Geography	Baseline growth rate	Additive increase in growth rate, Δr	Baseline reproduction number	Multiplicative increase in reproduction number
GLMM	1a	Separate-slopes multinomial spline model*	Sequence	Regions of UK	–	0.104 [0.100, 0.108]	–	77% [73, 81]
GLMM	1b	Common-slope multinomial model*	Sequence	Lower-tier local authorities of UK	–	0.093 [0.091, 0.095]	–	67% [65, 69]
GLMM	2h	Separate-slope binomial spline model†	S gene target failure‡	Regions of England	–	0.109 [0.107, 0.111]	–	83% [81, 84]
R_t regression	4a	Regional time-varying baseline	S gene target failure	Upper-tier local authorities of England	0.007 [0.002, 0.012]	0.067 [0.060, 0.073]	1.04 [1.01, 1.07]	43% [38, 48]
R_t regression	4b	Regional static baseline	S gene target failure	Upper-tier local authorities of England	0.007 [0.002, 0.012]	0.085 [0.079, 0.091]	1.04 [1.01, 1.07]	57% [52, 62]
Transmission model	5a	Increased transmissibility	S gene target failure‡	Regions of England	−0.001 [−0.017, 0.012]	0.118 [0.067, 0.168]	1.01 [0.94, 1.09]	82% [43, 130]
GLMM	3a	Common-slope binomial model†	Sequence	Regions of Denmark	–	0.080 [0.067, 0.092]	–	55% [45, 66]
GLMM	3b	Common-slope binomial model†	Sequence + RT-PCR rescreening	Regions of Switzerland	–	0.101 [0.092, 0.109]	–	74% [66, 82]
GLMM	3c	Common-slope binomial model†	S gene target failure‡	States of USA	–	0.084 [0.080, 0.088]	–	59% [56, 83]

*VOC 202012/01 versus B.1.177. †VOC 202012/01 versus all other variants. ‡Binomial counts adjusted for the true positive rate (proportion of S gene target failures that are VOC 202012/01), estimated from misclassification model (for UK) or a binomial GLMM fitted to sequencing data of S gene target failures (for US).

(NPIs) introduced on 1 January 2021: (i) a moderate-stringency scenario with mobility levels as observed in the first half of October 2020; (ii) a high-stringency scenario with mobility levels as observed during the second national lockdown in England in November 2020, with schools open; (iii) the same high-stringency scenario, but with schools closed until 15 February 2021; and (iv) a very-high-stringency scenario with mobility levels as observed during the first national lockdown in early April 2020, with schools closed (fig. S20). In combination with these NPI scenarios, we considered three vaccination scenarios: no vaccinations; 200,000 vaccinations per week; and 2 million vaccinations per week. We assumed that vaccine rollout starts on 1 January 2021 and that vaccinated individuals have a 95% lower probability of disease and a 60% lower probability of infection than unvaccinated individuals. For simplicity, we assume that vaccine protection is conferred immediately upon receipt of one vaccine dose. Note that these projections serve as indicative scenarios rather than formal predictive forecasts.

Regardless of control measures, all regions of England were projected to experience a new wave of COVID-19 cases and deaths in early 2021, peaking in February 2021 if no substantial control measures were introduced, or in mid-January 2021 if strong control measures succeeded in reducing R below 1 (Fig. 4A). In the absence of substantial vaccine rollout, the numbers of COVID-19 cases, hospitalizations, ICU admissions, and deaths in the first 6 months of 2021 were projected to exceed those in 2020, even with stringent NPIs in place (Table 2). Implementing more stringent measures in January 2021 (scenarios iii and iv) led to a larger rebound in cases when simulated restrictions were lifted in March 2021, particularly in those regions that had been least affected up to December 2020 (fig. S21). However, more stringent measures may buy time to reach more widespread population immunity through vaccination. Vaccine rollout further mitigated transmission, although the impact of vaccinating 200,000 people per week—similar in magnitude to the rates reached in December 2020—was relatively small (Fig. 4B and fig. S22). An accelerated uptake of 2 million people fully vaccinated per week (i.e., 4 million doses for a two-dose vaccine) had a much more substantial impact (Fig. 4C and fig. S23). However, accelerated vaccine rollout had a relatively limited impact on peak burden, as the peak was largely mediated by the stringency of NPIs enacted in January 2021, before vaccination had much of an impact. The primary benefit of accelerated vaccine rollout lies in helping to avert a resurgence of cases after the relaxation of NPIs, and in reducing transmission after the peak burden has already been reached.

As a sensitivity analysis, we also ran model projections with a seasonal component such that transmission is 20% higher in winter than in summer (*22*), but this did not qualitatively affect our results (fig. S24 and table S5).

Discussion

Combining multiple behavioral and epidemiological data sources with statistical and dynamic modeling, we estimated that the SARS-CoV-2 variant VOC 202012/01 has a 43 to 90% (range of 95% CrIs, 38 to 130%) higher reproduction number than preexisting variants of SARS-CoV-2 in England, assuming no changes to the generation interval. On the basis of early population-level data, we were unable to identify whether the new variant is associated with higher disease severity. Theoretical considerations suggest that, in some cases, natural

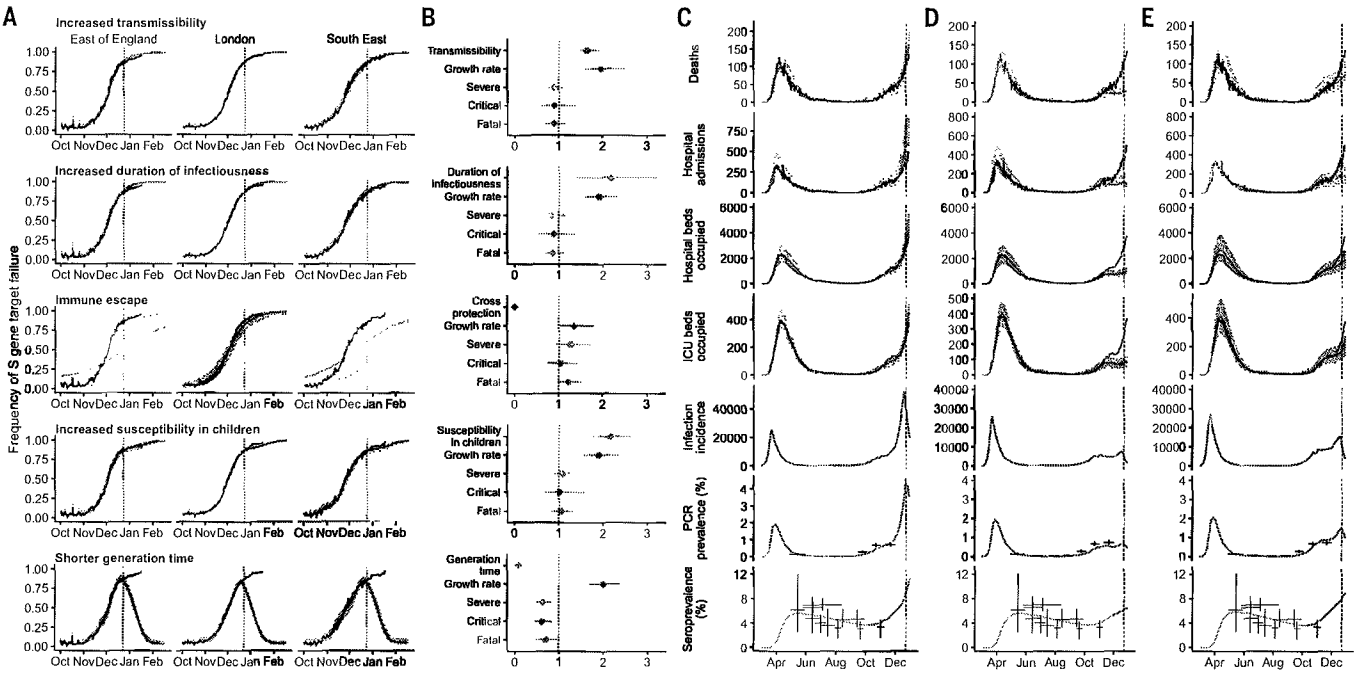

Fig. 3. Comparison of possible biological mechanisms underlying the rapid spread of VOC 202012/01. Each row shows a different assumed mechanism. (**A**) Relative frequency of VOC 202012/01 (black line and ribbon respectively denote observed S gene target failure frequency with 95% binomial credible interval; purple line and ribbon respectively denote mean and 95% credible interval from model fit). (**B**) Posterior estimates (mean and 95% credible intervals) for relative odds of hospitalization (severe illness), relative odds of ICU admission (critical illness), relative odds of death (fatal illness), growth rate as a multiplicative factor per week [i.e., $\exp(7 \cdot \Delta r)$], and the parameter that defines the hypothesized mechanism; all parameters are relative to those estimated for preexisting variants. (**C** to **E**) Illustrative model fits for the South East NHS England region: (**C**) fitted two-strain increased transmissibility model with VOC 202012/01 included; (D) fitted two-strain increased transmissibility model with VOC 202012/01 removed; (E) fitted single-strain model without emergence of VOC 202012/01. Black lines denote observed data; error bars denote the date range and 95% credible intervals for observed PCR prevalence and seroprevalence; colored lines and ribbons denote median and 95% credible intervals from model fit.

selection may favor reduced severity of disease in pathogens (*23*). For this to be true, however, the pathogen's infectious period must be truncated by disabling symptoms or death often enough that a less-virulent mutant generates more secondary infections despite potentially being less transmissible per contact, to the extent that decreased virulence and decreased transmissibility are consequences of the same mutation (e.g., one that decreases viral load). It is far from clear that this condition holds for SARS-CoV-2, given substantial transmission before the onset of severe symptoms. Regardless, without strengthened controls, there is a clear risk that future epidemic waves may be larger—and hence associated with greater burden—than previous waves. The UK government initiated a third national lockdown on 5 January 2021 in response to the rapid spread of VOC 202012/01, including school closures. Educational settings are among the largest institutions linked to SARS-CoV-2 clusters that remained open during the November 2020 lockdown (*24*), which means that the enacted school and university closures may have substantially assisted in reducing the burden of COVID-19 in early 2021.

The increase in transmission associated with VOC 202012/01 has crucial implications for vaccination. First, it means that prompt and efficient vaccine delivery and distribution are even more important to reduce the impact of the pandemic in the near future. Increased transmission resulting from VOC 202012/01 will raise the herd immunity threshold, so that the potential burden of SARS-CoV-2 is larger and higher vaccine coverage will be required to achieve herd immunity. It is therefore extremely concerning that VOC 202012/01 has spread to at least 114 countries globally (*2*). Although VOC 202012/01 was first identified in England, a rapidly spreading variant has also been detected in South Africa (*25*, *26*), where there was a marked increase in transmission in late 2020. Another variant exhibiting immune escape has emerged in Brazil (*27*, *28*). Thus, vaccination timelines will also be a crucial determinant of future burden in other countries where similar new variants are present. Second, there is a need to assess how VOC 202012/01 and other emerging lineages affect the efficacy of vaccines (*29*, *30*). Vaccine developers may need to consider developing formulations with variant sequences, and they may want to initiate post-licensure studies to detect differences in efficacy between the preexisting and new variants. Licensing authorities may need to clarify abbreviated pathways to marketing for vaccines that involve altering strain formulation without any other changes to their composition.

There are limitations to our analysis. We have considered a small number of intervention and vaccination scenarios, which should not be regarded as the only available options for policy-makers. Our transmission model does not explicitly capture nursing home or hospital transmission of SARS-CoV-2, and we fit the model to each region of England separately rather than pooling information across regions and explicitly modeling transmission between regions. There are also uncertainties in the choice of model used to generate these predictions, and the exact choice will yield differences in the measures needed to control the epidemic. We note that even without increased susceptibility of children to VOC 202012/01, the more efficient spread of the variant implies that the difficult societal decision of closing schools will be a key public health question for multiple countries in the months ahead.

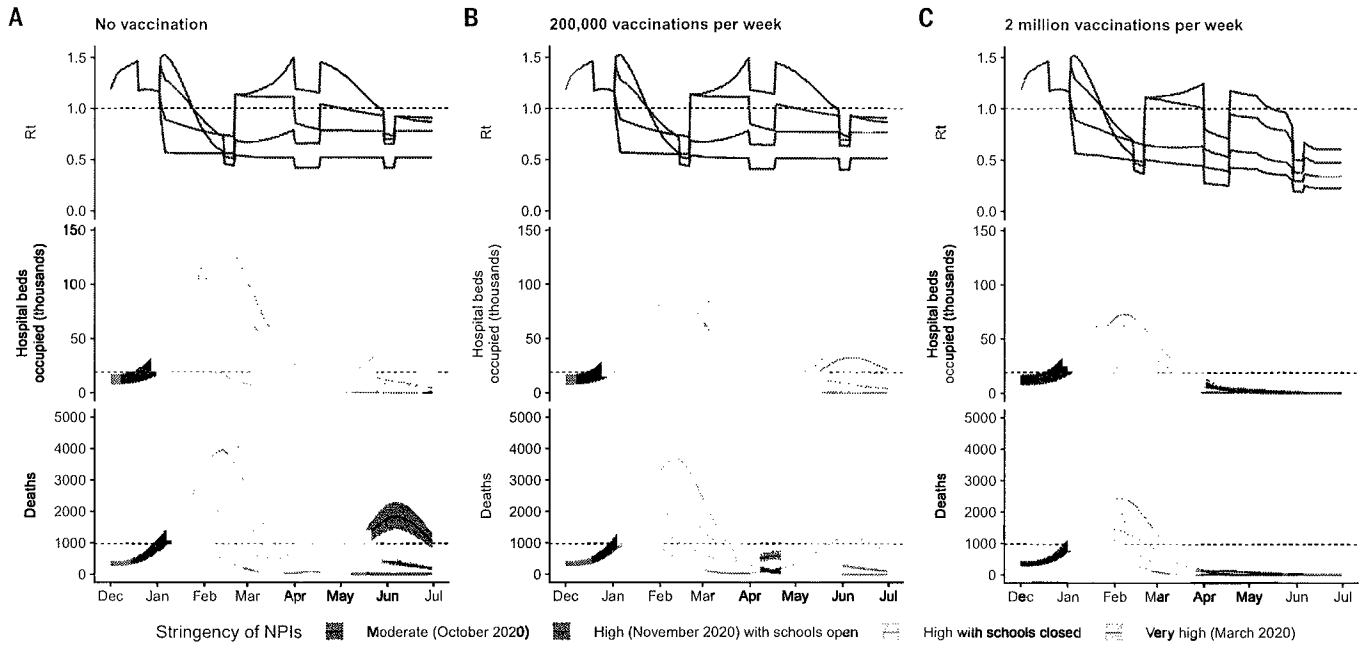

Fig. 4. Projections of epidemic dynamics under different control measures. We compare four alternative scenarios for nonpharmaceutical interventions from 1 January 2021: (i) mobility returning to levels observed during relatively moderate restrictions in early October 2020; (ii) mobility as observed during the second lockdown in England in November 2020, then gradually returning to October 2020 levels from 1 March to 1 April 2021, with schools open; (iii) as (ii), but with schools closed until 15 February 2021; (iv) as (iii), but with a lockdown of greater stringency as observed in March 2020 (fig. S20). (**A**) Without vaccination. (**B**) With 200,000 people vaccinated per week. (**C**) With 2 million people vaccinated per week. We assume that vaccination confers 95% vaccine efficacy against disease and 60% vaccine efficacy against infection, and that vaccination starts on 1 January 2021 with vaccine protection starting immediately upon receipt. This is intended to approximate the fact that vaccination started in early December, with full protection occurring after a time lag and potentially after a second dose. Vaccines are given first to people aged 70+ until 85% coverage is reached in this age group, then to people aged 60+ until 85% coverage is reached in this age group, continuing into younger age groups in 10-year decrements. Resurgences starting in March 2021 are due to the relaxation of nonpharmaceutical interventions, including reopening schools (fig. S20). Median and 95% credible intervals are shown. The dashed lines in rows 2 and 3 show peak hospitalizations and deaths from the first COVID-19 wave in England (April 2020).

Table 2. Summary of projections for England, 15 December 2020 to 30 June 2021. Median and 95% credible intervals are indicated.

	Moderate (October 2020)	High (November 2020) with schools open	High with schools closed	Very high (March 2020)
		No vaccination		
Peak ICU (relative to 1st wave)	274% (256–292%)	162% (151–172%)	130% (122–136%)	119% (112–124%)
Peak ICU bed requirement	9,980 (9,330–10,600)	5,880 (5,490–6,280)	4,720 (4,450–4,960)	4,310 (4,070–4,530)
Peak deaths	3,960 (3,730–4,200)	2,050 (1,920–2,160)	1,500 (1,440–1,570)	1,830 (1,670–2,000)
Total admissions	635,000 (604,000–659,000)	454,000 (432,000–472,000)	448,000 (425,000–466,000)	450,000 (425,000–472,000)
Total deaths	216,000 (205,000–227,000)	146,000 (138,000–152,000)	147,000 (139,000–155,000)	149,000 (140,000–157,000)
		200,000 vaccinations per week		
Peak ICU (relative to 1st wave)	269% (252–287%)	160% (149–170%)	130% (122–136%)	118% (112–124%)
Peak ICU bed requirement	9,790 (9,150–10,400)	5,810 (5,430–6,200)	4,710 (4,450–4,950)	4,310 (4,070–4,520)
Peak deaths	3,700 (3,500–3,920)	1,930 (1,820–2,040)	1,490 (1,430–1,550)	1,320 (1,280–1,380)
Total admissions	610,000 (580,000–634,000)	438,000 (416,000–454,000)	415,000 (394,000–430,000)	394,000 (373,000–413,000)
Total deaths	202,000 (192,000–213,000)	137,000 (130,000–143,000)	129,000 (123,000–135,000)	119,000 (112,000–125,000)
		2 million vaccinations per week		
Peak ICU (relative to 1st wave)	236% (221–252%)	149% (139–158%)	128% (121–134%)	118% (111–124%)
Peak ICU bed requirement	8,590 (8,050–9,170)	5,400 (5,070–5,760)	4,650 (4,390–4,880)	4,290 (4,060–4,500)
Peak deaths	2,470 (2,330–2,610)	1,510 (1,450–1,580)	1,390 (1,340–1,450)	1,290 (1,250–1,340)
Total admissions	483,000 (459,000–502,000)	353,000 (337,000–366,000)	277,000 (265,000–287,000)	190,000 (182,000–197,000)
Total deaths	140,000 (133,000–146,000)	98,900 (94,600–103,000)	81,000 (77,600–84,200)	58,200 (56,100–60,300)

We only assess relative support in the data for the mechanistic hypotheses proposed, but there may be other plausible mechanisms driving the resurgence of cases that we did not consider, and we have not identified the specific combination of mechanisms driving the increased transmission of VOC 202012/01. We identify increased transmissibility as the most parsimonious mechanistic explanation for the higher growth rate of VOC 202012/01, but a longer infectious period also fits the data well (table S4) and is supported by longitudinal testing data (*17*). Our conclusions about school closures were based on the assumption that children had reduced susceptibility and infectiousness relative to adults (*19*), but the precise values of these parameters and the impact of school closures remain the subject of scientific debate (*31*). We based our assumptions about the efficacy of NPIs on the measured impact on mobility of previous national lockdowns in England, but the impact of policy options cannot be predicted with certainty.

Despite these limitations, we found strong evidence that VOC 202012/01 is spreading substantially faster than preexisting SARS-CoV-2 variants. Our modeling analysis suggests that this difference could be explained by an overall higher infectiousness of VOC 202012/01, but not by a shorter generation time or immune escape alone. Further experimental work will provide insight into the biological mechanisms for our observations, but given our projections of a rapid rise in incidence from VOC 202012/01—and the detection of other novel and highly transmissible variants (*25*–*28*)—there is an urgent need to consider what new approaches may be required to sufficiently reduce the ongoing transmission of SARS-CoV-2.

Materials and methods

Summary of control measures in England in late 2020

After a resurgence of cases in September and October 2020, a second national lockdown was implemented in England, from 5 November to 2 December 2020. Restrictions included a stay-at-home order with exemptions for exercise, essential shopping, obtaining or providing medical care, education, and work for those unable to work from home. Schools were kept open. Non-essential shops and retail and leisure venues were required to close. Pubs, bars, and restaurants were allowed to offer takeaway services only. After the second national lockdown, regions in England were assigned to tiered local restrictions according to medium, high, and very high alert levels (Tiers 1, 2, and 3). In response to rising cases in southeast England and concerns over VOC 202012/01, the UK government announced on 19 December 2020 that a number of regions in southeast England would be placed into a new, more stringent "Tier 4," corresponding to a Stay at Home alert level. Tier 4 restrictions were broadly similar to the second national lockdown restrictions. As cases continued to rise and VOC 202012/01 spread throughout England, on 5 January 2021 a third national lockdown was introduced in England, with schools and universities closed and individuals advised to stay at home, with measures to be kept in place until at least mid-February 2021.

Data sources

To assess the spread of VOC 202012/01 in the UK, we used publicly available sequencing-based data from the COG-UK Consortium (*13*) (5 February 2020–6 January 2021) and Pillar 2 SARS-CoV-2 testing data provided by Public Health England (1 October 2020–7 January 2021) for estimating the frequency of S gene target failure in England. COG-UK sequencing data for Northern Ireland were excluded because of low sample sizes.

To assess the spread of VOC 202012/01 in Denmark, Switzerland, and the US, we used publicly available sequence data giving the incidence of VOC 202012/01 aggregated by week and region provided by the Danish Covid-19 Genome Consortium and the Statens Serum Institut (*32*) (15 October 2020–28 January 2021), sequence and RT-PCR 501Y.V1 rescreening data giving the incidence of VOC 202012/01 in different regions of Switzerland provided by Christian Althaus and Tanja Stadler and the Geneva University Hospitals, the Swiss Viollier Sequencing Consortium from ETH Zürich, the Risch laboratory, the University Hospital Basel, the Institute for Infectious Diseases, University of Bern, and the Swiss National Covid-19 Science Task Force (*33*, *34*) (2 November 2020–11 February 2021), and publicly available US nationwide Helix SARS-CoV-2 Surveillance data, comprising both S gene target failure data and randomly selected S-negative samples that were sequenced to infer the proportion of S-negative samples that were the VOC (***35***, *36*) (6 September–11 February 2020).

To estimate mobility, we used anonymized mobility data collected from smartphone users by Google Community Mobility (*11*). Percentage change in mobility per day was calculated for each lower-tier local authority in England, and a generalized additive model with a spline for time was fitted to these observations to provide a smoothed effect of the change in mobility over time (Fig. 1C).

To estimate social contact rates (Fig. 1D), we used data on reported social contacts from the CoMix survey (*12*), which is a weekly survey of face-to-face contact patterns, taken from a sample of ~2500 individuals broadly representative of the UK population with respect to age and geographical location. We calculated the distribution of contacts using 1000 bootstrap samples with replacement from the raw data. Bootstrap samples were calculated at the participant level, then all observations for those participants were included in a sample to respect the correlation structure of the data. We collected data in two panels that alternated weekly; therefore, we calculated the mean smoothed over the 2-week intervals to give a larger number of participants per estimate and to account for panel effects. We calculated the mean number of contacts (face-to-face conversational contact or physical contact) in the settings "home," "work," "education" (including childcare, nurseries, schools, and universities and colleges), and "other" settings. We calculated the mean contacts by age group and area of residence (those areas that were subsequently placed under Tier 4 restrictions on 20 December 2020 as they were experiencing high and rapidly increasing incidence, and those areas of England that were not placed under these restrictions). The mean number of contacts was influenced by a few individuals who reported very high numbers of contacts (often in a work context). The means shown here were calculated by truncating the maximum number of contacts recorded at 200 per individual per day. We compared R_t estimates derived from CoMix (*12*) to those derived from the REACT-1 prevalence survey (*9*) for England.

Statistical methods in brief

Growth of VOC 202012/01 after initial phylogenetic observation. For each lineage i in the COG-UK dataset, we pooled the number of sequences observed within that lineage across the UK for every day, t, yielding integer-valued sequence counts $N(i, t)$. We estimated the time-varying exponential growth rates of cases of each strain, $r(i, t)$, using a negative binomial state-space model correcting for day-of-week effects whose dispersion parameter was optimized for each strain by marginal likelihood maximization. We defined the relativized growth rate of a lineage i at time t as $\rho(i, t) = [r(i, t) - \bar{r}(t)]/\sigma_r(t)$, where $\bar{r}(t)$ is the average growth rate of all circulating strains at time t and $\sigma_r(t)$ is the standard deviation of growth rates across all lineages at time t, such that $\rho(i, t)$ is analogous to a z-statistic or Wald-type statistic and allows comparison of growth rate differences across time when the average growth rate and scale of growth rate differences varies.

Competitive advantage and increased growth rate of VOC-202012/01. To estimate the increase in growth rate of VOC 202012/01, we fitted a set of multinomial and binomial generalized linear mixed models (GLMMs), in which we estimated the rate by which the VOC displaces other resident SARS-CoV-2 variants across different regions in the UK, based on both the COG-UK sequence data and the S gene target failure data. In the analysis of the S gene target failure data, binomial counts were adjusted for the

true positive rate. For comparison, we also calculated the growth advantage of the VOC in Denmark, Switzerland, and the US based on both sequencing and S gene target failure data. All models took into account sample date and region, plus (if desired) their interaction, and all mixed models took into account possible overdispersion and for the UK further included local-tier local authority as a random intercept. From these models, we estimated the difference in Malthusian growth rate between other competing variants Δr, as well as the expected multiplicative increase in basic reproduction number R_t and infectiousness, assuming unaltered generation time, which can be shown to be equal to $\exp(\Delta r.T)$, where T is the mean generation interval. The multiplicative increase being equal to $\exp(\Delta r.T)$ is an approximation that holds for a delta-distributed generation interval, but we show in the supplementary materials that this is a good approximation for the gamma-distributed generation interval that we assume. In our calculations, we used estimated SARS-CoV-2 mean generation times T of either 5.5 days (*14*) (Table 1) or 3.6 days (*37*, *38*) (table S1).

R_t analysis. We calculated the weekly proportion of positive tests that were S gene–negative out of all positive tests that tested for the S gene by English upper-tier local authority. We used reproduction number estimates obtained using the method described in (*37*) and (*39*) and implemented in the EpiNow2 R package (*40*), downloaded from https://github.com/epiforecasts/covid-rt-estimates/blob/master/subnational/united-kingdom-local/cases/summary/rt.csv. We then built a separate model of the expected reproduction number in UTLA i during week t starting in the week beginning 5 October 2020 as a function of local restrictions, mobility indicators, residual temporal variation, and proportion of positive tests with S gene target failure. The residual temporal variation is modeled either as a region-specific thin-plate regression spline ("Regional time-varying") or a static regional parameter ("Regional static"). The key estimand is the relative change in reproduction number in the presence of S gene target failure that is not explained by any of the other variables.

Transmission dynamic model

We extended a previously developed modeling framework structured by age (in 5-year age bands, with no births, deaths, or aging due to the short time scales modeled) and by geographical region (*10*, *15*) to include two variants of SARS-CoV-2 (VOC 202012/01 and non-VOC 202012/01). The model is a discrete-time deterministic compartmental model that allows for arbitrary delay distributions for transitions between compartments. We fitted this model to multiple regionally stratified data sources across the seven NHS England regions as previously: deaths, hospital admissions, hospital bed occupancy, ICU bed occupancy, daily incidence of new infections, PCR prevalence of active infection, seroprevalence, and daily frequency of VOC 202012/01 across each of the regions as measured by S gene target failure frequency corrected for false positives. The model assumes that individuals with clinical symptoms are more infectious than individuals with subclinical infection (*19*). We assume that vaccinated individuals have a lower probability of both clinical and subclinical infection (fig. S9), but that vaccinated individuals who do develop clinical or subclinical infection are as infectious as unvaccinated individuals with clinical or subclinical infection. To model school closure, we removed all school contacts from our contact matrix based on POLYMOD data and varying over time according to Google Mobility indices, as described (*10*). See supplementary materials for details of Bayesian inference including likelihood functions and prior distributions.

Our individual transmission model fits to separate NHS regions of England produced independent estimates of parameters such as relative transmissibility and differences in odds of hospitalization or death resulting from infection with VOC 202012/01. To produce overall estimates for these parameters, we modeled posterior distributions from individual NHS regions as draws from a mixture distribution, comprising a normally distributed top-level distribution from which central estimates for each NHS region are drawn. We report the mean and credible intervals of the top-level distribution when reporting model posterior estimates for England.

In model fitting, we assume that our deterministic transmission model approximates the expectation over stochastic epidemic dynamics. This is not exact (*41*), but the error in this approximation is small for the population-level processes we are modeling, as it decays with $1/N$ (*42*). This approach is well developed for state-space models of communicable disease dynamics that fit an epidemic process to observed data via a stochastic observation process.

Apparent growth of VOC 202012/01 not a result of testing artifacts

The apparent frequency of VOC 202012/01 could be inflated relative to reality if this variant leads to increased test-seeking behavior (e.g., if it leads to a higher rate of symptoms than preexisting variants). However, this would not explain the growth in the relative frequency of VOC 202012/01 over time. Mathematically, if variant 1 has growth rate r_1 and variant 2 has growth rate r_2, the relative frequency over time is $a_2 \exp(r_2 t)/[a_1 \exp(r_1 t) + a_2 \exp(r_2 t)]$, where a_1 and a_2 are the frequency of variants 1 and 2, respectively, at time $t = 0$. However, if variant 1 has probability x of being reported and variant 2 has probability y, and both have growth rate r, the relative frequency over time is $a_2\, y \exp(rt)/[a_1\, x \exp(rt) + a_2\, y \exp(rt)]$, which is constant.

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SUPPLEMENTARY MATERIALS

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Dr. BYRAM BRIDLE
Plaintiff

-and-

UNIVERSITY OF GUELPH et al.
Defendants

Court File No. CV-22-00691880-0000

ONTARIO
SUPERIOR COURT OF JUSTICE

PROCEEDING COMMENCED AT TORONTO

MOTION RECORD OF DEFENDANT DAVID FISMAN
(Returnable November 19, 2024)

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ONTARIO
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and

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MOTION RECORD OF DEFENDANT DAVID FISMAN
(Returnable November 19, 2024)
Vol. II of II

June 30, 2023

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AND TO: **JANE OR JOHN DOE JUNIOR SCIENTIST**

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Court File No. CV-22-00691880-0000

ONTARIO
SUPERIOR COURT OF JUSTICE

B E T W E E N:

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Defendants

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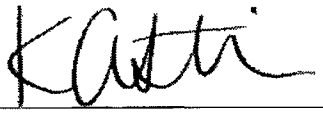
This is Exhibit "F" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.



Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Estimated transmissibility and impact of SARS-CoV-2 lineage B.1.1.7 in England

Nicholas G. Davies, Sam Abbott, Rosanna C. Barnard, Christopher I. Jarvis, Adam J. Kucharski, James D. Munday, Carl A. B. Pearson, Timothy W. Russell, Damien C. Tully, Alex D. Washburne, Tom Wenseleers, Amy Gimma, William Waites, Kerry L. M. Wong, Kevin van Zandvoort, Justin D. Silverman, CMMID COVID-19 Working Group1, COVID-19 Genomics UK (COG-UK) Consortium, Karla Diaz-Ordaz, Ruth Keogh, Rosalind M. Eggo, Sebastian Funk, Mark Jit, Katherine E. Atkins, and W. John Edmunds

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UK variant transmission

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has the capacity to generate variants with major genomic changes. The UK variant B.1.1.7 (also known as VOC 202012/01) has many mutations that alter virus attachment and entry into human cells. Using a variety of statistical and dynamic modeling approaches, Davies *et al.* characterized the spread of the B.1.1.7 variant in the United Kingdom. The authors found that the variant is 43 to 90% more transmissible than the predecessor lineage but saw no clear evidence for a change in disease severity, although enhanced transmission will lead to higher incidence and more hospital admissions. Large resurgences of the virus are likely to occur after the easing of control measures, and it may be necessary to greatly accelerate vaccine roll-out to control the epidemic.

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Preventive Medicine

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Pfizer-BioNTech vaccine effectiveness against Sars-Cov-2 infection: Findings from a large observational study in Israel

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ABSTRACT

Development of an effective vaccine against Covid-19 is crucial to reducing infection. mRNA BNT162b2, developed and manufactured by Pfizer-BioNTech, was one of the first FDA-approved vaccinations reporting high efficacy (95%) and minimal side effects. Evaluating effectiveness of BNT162b2 in a general population has been made possible after the implementation of a nation-wide vaccination program in Israel.

This retrospective cohort study was carried out in Maccabi HealthCare services, Israel among 1.6 million members aged 16 and over. The population was divided into those who were at least seven days post- second vaccination and those who had not been vaccinated. Number of days till the end of the study or Covid-19 infection, Covid-19-related hospitalization and mortality was calculated for each participant between 18.1.2021 to 25.4.2021. Participants who had reached day eight after second vaccination during the study period could contribute days to both groups. Vaccine efficacy (VE) was calculated using a conditional Poisson model, controlling for age group, gender, hypertension, diabetes and obesity, fitted within clusters defined by geographical statistical area and calendar week.

BNT162b2 was found effective for the total population group for infection, hospitalization and mortality, with adjusted VE of 93·0% (CI:92·6–93·4%), 93·4% (CI:91·9–94·7%) and 91·1% (CI:86·5–94·1%) respectively. VE for infection was lower for participants aged 75 and over, and for those with hypertension, diabetes and obesity.

This study strengthens the evidence that the Pfizer-BioNTech vaccination is effective in preventing infection, hospitalization and mortality.

1. Introduction

A number of vaccines were authorized for emergency use by the U.S. Food and Drug Administration (FDA), after reporting high efficacy and safety results in their respective randomized control trials (RCT) (Fu et al., 2020). Pfizer-BioNTech reported 95% efficacy (CI: 90.3%–97.6%) for their vaccine, with low serious adverse event rates in both the vaccinated and control groups (Polack et al., 2020). The vaccine was initially FDA-approved for ages 16 and over, requiring two doses at a recommended interval of 21 days. After two doses, RCT results indicated a significant divergence in infection rates between the intervention and control group after day seven (FDA, 2020). With an average of two months follow-up from day seven after the second vaccination, only eight new cases of 18,198 infection naïve participants developed symptoms in the intervention group, compared with 162 new cases in a similar population group that received the placebo (Polack et al., 2020).

Israel was among the first countries to implement a national drive to vaccinate its population, employing the mRNA BNT162b2 vaccine, as recommended. All four health maintenance organizations (HMOs) that provide health coverage for all 9.3 million of the population, offered the vaccine free of charge. From 20.12.2020, the vaccine was offered initially to healthcare workers and members aged 60 and over (Rosen et al., 2021), with the target population progressively broadened to include all citizens aged 16 and over by 4.2.2021 (Rossman et al., 2021). At the same time, Israel – like many of the European countries – was experiencing its third wave of the epidemic (Rosen et al., 2021). This

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wave was more severe than previous waves, secondary to the introduction of the B.1.1.7 variant of the SARS-CoV-2 virus into the country (Israel Ministry of Health, 2021), that had a much higher transmissibility than earlier viral forms (Leung et al., 2021; Galloway et al., 2021). The early introduction of the national campaign, combined with the speed with which the majority of the population was vaccinated (end of March), affords Israel the opportunity to evaluate effectiveness of the vaccine for a longer follow-up period than in other countries.

Clalit, the largest HMO in Israel, was the first to report vaccine effectiveness (92%), but with a very short follow-up period (15 days) (Dagan et al., 2021). National population-based data reported 94% vaccine effectiveness for infection with a 45-day follow-up period (Haas et al., 2021). The current study, carried out at Maccabi HealthCare Services (MHC), followed up the vaccine target population for a maximum 98 days. MHC is the second largest HMO in Israel and responsible for the care of over 2.5 million residents. MHC operated 95 vaccination sites across the country, including mobile sites and ambulance transport for homebound patients to reach vaccination sites.

MHC maintains a comprehensive database allowing the HMO to measure vaccine effectiveness in a broad range of population groups. The database includes demographic data, laboratory results, vaccination and hospitalization data of all members, including services not provided directly by the MHC (such as hospitalizations and vaccinations carried out by the ambulance service among nursing home residents). The database also maintains a number of chronic illness registries, based on algorithms incorporating data from multiple sources (medications, tests, diagnoses and procedures), as well as an obesity registry based on routinely recorded height and weight measurements. Persons suffering from chronic illnesses, such as hypertension, diabetes and obesity are at increased risk of Covid-19 infection (Tadic et al., 2020; Peric and Stulnig, 2020; Zhou et al., 2021) and little is known regarding their response to the vaccine.

The main objective of the study was to measure vaccine effectiveness regarding infection, hospitalization and mortality from Sars-Cov-2 virus after adjusting for both person-specific risk variables and virus exposure. The current study provides initial data comparing CoVid-19 incidence rates over a maximum potential 98-day follow-up period.

2. Methods

We conducted a retrospective cohort study based on vaccination, laboratory records and hospital data extracted from the MHC database. The study was approved by the MHC internal review board and by the Helsinki committee (#0178–20-MHS) and exempted from informed consent, after meeting guidelines for the protection of human subjects concerning safety and privacy.

2.1. Study population

The study population included all active HMO members (as of 18.1.2021) aged 16 and over that did not leave the HMO during the study period and had no evidence of prior infection (positive PCR or serology). The study population was divided into two dynamic groups: those who were at least seven days post- second vaccination between 18.1.2021 and 25.04.2021 (herein referred to as the 'vaccinated group'), and those who were not vaccinated (herein referred to as the 'unvaccinated group'). The groups were dynamic, such that people who were initially unvaccinated exited the 'unvaccinated group' on receipt of their first dose and entered the 'vaccinated group' eight days after receiving their second dose, provided that they had not been infected or died in the intervening period. Prior infection was defined for each group as follows: a positive PCR or IgG serology result prior to day eight after second dose of vaccination for the 'vaccinated group' and prior to 18.1.2021 for the 'unvaccinated group'.

Members who had vaccinated with one dose only before the 18.1.2021 and did not receive a second dose by the end of the study period (25.04.2021), and those who had died within seven days of receiving their second dose were excluded from the study.

2.2. Sars-Cov-2 testing

Polymerase chain reaction (PCR) testing is carried out for all HMO members presenting with symptoms or reporting exposure to a confirmed case, free of charge. PCR testing is required for all returning travelers from overseas, anyone who has been in contact with an infected person, or anyone presenting with flu-like symptoms. PCR testing does not require a physician's referral and is carried out upon member request. Serology testing in the general population and among health workers was carried out among sample populations at discrete points in time to assess infection rates (including those asymptomatic) and is carried out in the HMO using a nucleoprotein-based antigen with follow-up chemo-luminescence immunoassay.

2.3. Outcome measures

Outcome measures included Covid-19 infection, hospitalization and mortality. For each group, new PCR positive cases (based on test date), Covid-19-related hospitalizations and deaths were identified. Number of days each member contributed was calculated as follows for each of the outcome variables: for the 'vaccinated group', from day 8 after vaccination to end of study period (25.4.2021) or till date of outcome (positive PCR/hospitalization/death); for the 'unvaccinated group', from 18.1.2021 to end of study period, date of first vaccination or date of outcome.

2.4. Statistical analysis

Crude vaccine effectiveness (VE) rates were calculated as: 1 – (incidence rate in vaccinated/incidence rate in unvaccinated). Incidence rates were calculated as number of outcome events (first positive PCR results/first Covid-19-related hospitalization/Covid-19-related death) per 1000 person-days. Crude outcome VE rates were calculated for each age group, gender, socio-economic status (based on census and national survey classifications applied to home address), presence of hypertension and diabetes (based on registry data) and obesity (BMI $\geq$ 30). To account for risk according to exposure and individuals' characteristics, outcome rates were compared between the two groups within geographical statistical area (GSA) and calendar week using conditional Poisson regression analysis (Armstrong et al., 2014), adjusting for subject-specific variables: age group, gender, presence of hypertension, diabetes and obesity. GSAs (a smaller area unit, as defined by the Central Bureau of Statistics) and calendar week were selected as conditional variables as they reflect risk of infection for a particular neighborhood/area over time. The number of days contributed was calculated for each week and GSA. Adjusted VE rates were calculated as follows: one minus the exponent of the point estimates for vaccine status. Analyses were carried out using R software, version 3.6.2. Packages "gnm" was used to run conditional Poisson models (Turner and Firth, 2020). Confidence intervals (CIs) were calculated using 95% confidence levels.

3. Results

Of all HMO members aged 16 and over with no prior infection as of 18.1.2021 (N = 1,658,604), 7719 members were excluded: 7670 that had only one vaccination dose and 49 who died within seven days of the second dose. Due to the dynamic nature of the study population, participants could contribute to both the 'vaccinated' and 'unvaccinated' groups. Demographic characteristics of the study population (presented in Table 1) have therefore been split into three groups: 'only vaccinated' - those who were already seven days post- second vaccination at the beginning of the study period and therefore contributed all their days to this group, 'became vaccinated' – those who contributed days to both

Table 1
Demographic characteristics of study population by vaccination status, Maccabi HealthCare Services, Israel.

Characteristic	Only vaccinated*	Became vaccinated*	Only unvaccinated*
	N = 575,259	N = 772,717	N = 302,909
Gender			
Female	302,693 (52.62%)	399,047(51.64%)	160,371(52.94%)
Male	272,566 (47.38%)	373,670(48.36%)	142,538(47.06%)
Age Group (years)			
16–44	101,686 (17.68%)	550,538(71.25%)	195,313(64.48%)
45–59	190,838 (33.17%)	175,277(22.68%)	61,007(20.14%)
60–74	208,725 (36.28%)	35,568(4.6%)	31,603(10.43%)
75+	74,010(12.87%)	11,334(1.47%)	14,986(4.95%)
SES			
Low	68,710(11.94%)	146,347(18.94%)	97,674(32.25%)
Middle	280,615 (48.78%)	390,498(50.54%)	152,572(50.37%)
High	225,934 (39.28%)	235,872(30.53%)	52,663(17.39%)
Health Status			
Hypertension	201,292 (34.99%)	64,134(8.3%)	37,984(12.54%)
Diabetes	92,369(16.06%)	27,846(3.6%)	16,562(5.47%)
Obese	135,486 (23.55%)	109,120(14.12%)	45,692(15.08%)

* Population group to which the individual member contributed to, wherein the 'only vaccinated' contributed only to the vaccinated population, the 'became vaccinated' contributed days to both the unvaccinated and vaccinated group and the 'only unvaccinated' contributed only to the unvaccinated group.

the 'unvaccinated' and 'vaccinated' groups (presented for vaccination later and therefore reached day eight post-second vaccination during the study period), and 'only unvaccinated' – those who had not vaccinated (no dose) by the end of the study period.

Of the 1,650,885 study participants, 34.9% were in the 'only vaccinated' group, 46.8% 'became vaccinated' during the study period and 18.3% were in the 'only unvaccinated' group. The 'only vaccinated' participants were more likely to be older than those in the other two groups, and thus also more likely to have hypertension, diabetes and suffer from obesity. Though somewhat younger, the demographic and health characteristics of the 'became vaccinated' group were more analogous to the 'only vaccinated' than the 'only unvaccinated' groups. The 'only unvaccinated' group were more likely to come from a lower socioeconomic bracket than those vaccinated (either group).

Of the total study population, 1.7% (N = 28,042) became PCR positive during the study period, of whom 3.7% were hospitalized (N = 1047) and 0.5% died (N = 164). Infection, hospitalization and mortality incidence rates between the 'vaccinated' and 'unvaccinated' groups are presented in Table 2. Incidence rates for all three outcomes were much lower for the 'vaccinated group' than the 'unvaccinated group'. The 'vaccinated group' had on average 23 days more follow-up than the 'unvaccinated group' (Table 2). This is attributed to the relatively short number of days that those who 'became vaccinated' during the study period contributed to the 'unvaccinated group' before getting their first vaccination.

Crude vaccine effectiveness rates for all three outcome measures are presented in Table 3 and adjusted coefficient estimates presented in Table 4. Adjusted VE rates for infection, Covid-19-related hospitalization and Covid-19-related mortality were 93.0% (CI: 92.6–93.4), 93.4% (CI: 91.9%–94.7%) and 91.1% (CI: 87%–94%) respectively. Incidence of infection decreased with increasing age, but was higher for people suffering from diabetes and obesity. Crude VE for infection decreased by increasing age, but remained above 90% for those under the age of 75

Table 2
Covid-19 infection, hospitalization and mortality rates in study population by vaccination status, 18.1.2021–25.4.2021, Maccabi HealthCare Services, Israel.

Measure	Vaccinated population	Unvaccinated population
	N = 1,347,976	N = 1,075,626
Mean follow-up days (SD)*	63.3 (22.15)	40.5 (33.95)
Median follow-up days (min, max)*	64 (1,98)	28 (1,98)
Number infected (PCR positive)	1410	26,632
Proportion infected / 1000 persons	1.05	24.76
Incidence infection rate (cases/1000 person-days)	0.017	0.61
Number hospitalized	105	942
Proportion hospitalized / 1000 persons	0.078	0.876
Incidence hospitalization rate (hospitalized/1000 person-days)	0.001	0.018
Number deaths	33	131
Proportion died / 1000 persons	0.02	0.12
Mortality incidence rate (deaths/1000 person-days)	0.0004	0.0025

* For infection rate analysis.

(crude and adjusted rates). Adjusted VE for infection was 94.7% for the 16–44 age group and dropped to 84.0% for the 75+ age group. Adjusted VE for infection for the 70+ age group was calculated as 89.1% (CI:83%–93%). From the results of the conditional Poisson model, when the interaction of age with vaccine status was included (Table 4) vaccine effectiveness was 11% lower for those aged 75 and above compared to the youngest age group (16–44). For both hospitalization and mortality, the variation in vaccine effectiveness by age group was not significant, but this may be attributed to the small number of cases (Tables 3 & 4). For all three outcomes, males had consistently lower VE rates (crude and adjusted) than females, however, these differences were not significant (Table 4).

Adjusted VE rates for infection were lower for study participants with hypertension (89.7%, CI:88.6–91.7), diabetes (88.9%, CI: 87.3–90.2) and obesity (89.7%, 88.6–90.7) than total population VE for infection (93%, CI: 92.6–93.4). VE point estimates for hospitalization and mortality among those with hypertension, diabetes or obesity were not appreciably different from total population VE.

4. Discussion

In this study of over 1.6 million participants, Pfizer-BioNTech VE for infection adjusted for gender, age, hypertension, diabetes and obesity and conditioned on GSA and calendar week was 93% (CI:92.6–93.4). Based on an average follow-up period of 63 days for the two-dose vaccinated population and 40 days for those not vaccinated, the infection rates found here are slightly lower than those reported in the original Pfizer RCT (95% (CI: 90.3–97.6) with an average of 2 months follow-up) (Polack et al., 2020) but comparable with those of Clalit, another HMO in Israel (92% (CI: 88–95) with a maximum 15 day follow-up) (Dagan et al., 2021) and national data (94.1%, CI: 93.4–94.7) (Haas et al., 2021).

Adjusted VE for hospitalization in this study was 93.4% (CI:91.9%–94.7%). The Clalit study (Dagan et al., 2021) reported lower adjusted VE point estimates for hospitalization (87%, CI: 55%–100%). We suggest that the Clalit data under-estimate VE for hospitalization as a result of the small number of cases in the short time period available for analysis. National data (Haas et al., 2021), adjusted for age, gender and calendar week with a maximum follow-up period of 41 days reported a higher VE for hospitalization (96.2%, CI: 95.5–96.8) than the present study. We suggest that the inclusion of GSA in our model for adjustment of exposure, controlling for chronic illness conditions and the longer follow-up period, provides a more accurate estimate of VE for hospitalization.

Adjusted VE for mortality in this study was 91.1% (CI: 86.5%–94.1%). The initial Pfizer RCT (Polack et al., 2020) reported six deaths

Table 3
Crude incidence rates by selected demographic and crude vaccine effectiveness (VE) in study population by vaccination status, 18.1.2021–25.4.2021, Maccabi HealthCare Services, Israel.

Outcome	Population	Vaccinated group		Unvaccinated group		VE (CI)
		Cases / N (%)	Incidence rate/1000 person-days	Cases / N (%)	Incidence rate/1000 person-days	
Covid-19 infection	Total population	1410/1347974 (0·1%)	0·017(85367633)	26,632/1074275 (2·48%)	0·612(43548556)	0·972(0·971, 0·974)
	Females	723/701738(0·1%)	0·016(44301847)	14,543/558661 (2·6%)	0·631(23036470)	0·975(0·973, 0·976)
	Males	687/646236 (0·11%)	0·017(41065786)	12,089/515620 (2·34%)	0·589(20512086)	0·971(0·969, 0·973)
	16–44 years	378/650004 (0·06%)	0·011(33396806)	20,160/744834 (2·71%)	0·673(29952051)	0·984(0·982, 0·985)
	45–59 years	435/367678 (0·12%)	0·017(25077190)	4610/236005(1·95%)	0·564(8177652)	0·97(0·967, 0·973)
	60–74 years	393/244744 (0·16%)	0·02(19943541)	1388/67130(2·07%)	0·369(3766187)	0·946(0·939, 0·952)
	75+ years	204/85549(0·24%)	0·029(6950096)	474/26311(1·8%)	0·287(1652666)	0·899(0·881, 0·914)
	Hypertension	294/120575 (0·24%)	0·032(9329714)	1102/44404(2·48%)	0·533(2066733)	0·94(0·932, 0·947)
	Diabetes	512/266417 (0·19%)	0·025(20525175)	2156/102108(2·11%)	0·451(4779689)	0·945(0·939, 0·95)
	Obesity	405/244837 (0·17%)	0·024(16795802)	4369/154789(2·82%)	0·694(6294289)	0·965(0·962, 0·969)
Covid-19 hospitalization	Total population	105/1353847 (0·01%)	0·001(85846734)	942/1162033(0·08%)	0·018(52753054)	0·944(0·932, 0·955)
	Females	43/704587(0·01%)	0·001(44537129)	478/601350(0·08%)	0·017(27675270)	0·941(0·92, 0·957)
	Males	62/649260(0·01%)	0·002(41309605)	464/560689(0·08%)	0·019(25077784)	0·895(0·863, 0·919)
	16–44 years	6/652429(0%)	0(33563279)	312/803504(0·04%)	0·009(36431271)	–
	45–59 years	15/369323(0%)	0·001(25216862)	261/256605(0·1%)	0·026(10155430)	0·962(0·935, 0·977)
	60–74 years	28/245923(0·01%)	0·001(20058792)	200/74179(0·27%)	0·046(4383957)	0·978(0·968, 0·985)
	75+ years	56/86172(0·06%)	0·008(7007801)	169/27750(0·61%)	0·095(1782396)	0·916(0·886, 0·938)
	Hypertension	20/463454(0%)	0·001(31348964)	66/302181(0·02%)	0·006(10482442)	0·953(0·935, 0·967)
	Diabetes	60/673900(0·01%)	0·001(42356338)	472/580830(0·08%)	0·018(26284928)	0·951(0·937, 0·962)
	Obesity	25/216494(0·01%)	0·002(12141432)	404/279205(0·14%)	0·025(15985684)	0·976(0·962, 0·984)
Covid-19 mortality	Total population	33/1354444(0%)	0·0004(85894784)	131/1166487(0·01%)	0·0025(53150685)	0·84(0·766, 0·891)
	Females	23/649557(0%)	0·0002(44561168)	75/562886(0·01%)	0·0020(27881504)	0·9(0·804, 0·949)
	Males	10/704887(0%)	0·0006(41333616)	56/603607(0·01%)	0·0030(25269181)	0·8(0·681, 0·875)
	16–44 years	0/652535(0%)	0(33570019)	5/805124(0%)	0(36582830)	–
	45–59 years	0/369422(0%)	0(25224360)	15/257786(0·01%)	0·001(10263103)	–
	60–74 years	8/246077(0%)	0·0004(20071813)	40/75249(0·05%)	0·0089(4473540)	0·955(0·904, 0·979)
	75+ years	25/86410(0·03%)	0·0036(7028592)	71/28333(0·25%)	0·0388(1831212)	0·907(0·854, 0·941)
	Hypertension	18/121521(0·01%)	0·0019(9419554)	59/50100(0·12%)	0·0228(2581586)	0·917(0·859, 0·951)
	Diabetes	26/268205(0·01%)	0·0013(20691931)	90/112791(0·08%)	0·0156(5755391)	0·917(0·871, 0·946)
	Obesity	2/246346(0%)	0·0001(16929075)	5/172373(0%)	0·0006(8047035)	0·833(0·141, 0·968)

(two in the intervention group and four in the control group) but deemed all deaths to be unrelated to the vaccine. We have been able to present here mortality data that is illness-specific that perhaps reflects a more realistic assessment of mortality risk. In the national study (Haas et al., 2021), VE rates reported for mortality were slightly higher (93.3%, CI: 91.5–94.8) than those reported here. Clalit reported adjusted VE for severe disease (defined as severe disease or death) as 92%(CI:75%–100%) (Dagan et al., 2021).

The elderly population are at higher risk of morbidity and mortality from Sars-Cov-2 infection (Salzberger et al., 2021) and one of the greatest concerns regarding the vaccine is that it would be less effective in the elderly population (Soiza et al., 2021). We found that adjusted VE for infection was in fact lower in the 75+ age group (81%) than for those under age 75 (90 + %), with a VE for infection of 89% (CI: 83%–93%) for those aged 70 and over. Dagan et al., 2021[9] reported adjusted VE rates for infection of 95% (CI:87%–100%) for the 70+ age group. Their study excluded members from nursing homes, the homebound and those presenting to the healthcare system within three days. These groups were not excluded in this study. Inclusion of these population groups, and the longer follow-up period eight-plus days post- second vaccination may account for the lower VE for infection in this study.

Hypertension (Tadic et al., 2020), diabetes (Peric and Stulnig, 2020) and obesity (Zhou et al., 2021) have all been established as risk factors for Sars-Cov-2 infection. Independent of age, we wished to determine if

Table 4
Exponents of coefficients for infection, hospitalization and mortality, adjusted for sex, age group and morbidity, and conditioned on GSA and calendar week (using Conditional Poisson model) 18.1.2021–25.4.2021, Maccabi HealthCare Services, Israel.

Outcome	Variable	Without interaction	Sex	Age Group	Hypertension	Diabetes	Obesity
Infection	Vaccinated	***0·070	***0·066	***0·053	***0·059	***0·064	***0·066
	Sex(M)	***0·913	***0·908	***0·911	***0·912	***0·912	***0·913
	45–59	***0·910	***0·910	***0·913	***0·919	***0·915	***0·911
	60–74	***0·625	***0·625	***0·595	***0·625	***0·626	***0·625
	75 ≤	***0·577	***0·577	***0·945	***0·576	***0·579	***0·578
	Hypertension	*0·945	*0·945	*0·950	***0·878	*0·945	*0·944
	Diabetes	***1·124	***1·123	***1·125	***1·122	1·027	***1·121
	Obesity	***1·147	***1·147	***1·149	***1·150	***1·149	***1·132
	Vaccinated Male		1·111				
	Vaccinated 45–59			*1·163			
	Vaccinated 60–74			***1·600			
	Vaccinated 75 ≤			***2·996			
	Vaccinated Hypertension				***1·745		
	Vaccinated Diabetes					***1·755	
	Vaccinated Obesity						**1·218
Covid-19-related hospitalization	Vaccinated	***0·068	***0·058	***0·051	***0·050	***0·064	***0·090
	Sex(M)	***1·260	**1·231	***1·252	***1·256	***1·259	***1·263
	45–59	***2·622	***2·621	***2·688	***2·664	***2·627	***2·580
	60–74	***3·605	***3·601	***3·783	***3·675	***3·614	***3·540
	75 ≤	***8·112	***8·100	***7·09	***8·265	***8·134	***7·939
	Hypertension	***1·348	***1·347	***1·351	***1·276	***1·348	***1·717
	Diabetes	***1·715	***1·712	***1·719	***1·717	***1·695	***1·346
	Obesity	***1·347	***1·347	***1·351	***1·350	***1·348	***1·491
	Vaccinated Male		1·254				
	Vaccinated 45–59			0·795			
	Vaccinated 60–74			0·864			
	Vaccinated 75 ≤			2·37			
	Vaccinated Hypertension				*1·550		
	Vaccinated Diabetes					1·086	
	Vaccinated Obesity						***0·354
Covid-19-related mortality	Vaccinated	***0·089	***0·082	0	***0·089	***0·087	***0·087
	Sex(M)	***1·902	***1·852	***1·879	***1·902	***1·901	***1·901
	45–59	***10·062	***10·067	***10·851	***10·062	***10·092	***10·089
	60–74	***49·270	***49·299	***51·024	***49·264	***49·507	***49·417
	75 ≤	***208·388	***208·138	***189·483	***208·381	***209·351	***209·181
	Hypertension	***2·108	***2·104	***2·109	***2·108	***2·110	***2·110
	Diabetes	***2·692	***2·689	***2·687	***2·692	***2·657	***2·694
	Obesity	***0·070	***0·070	***0·070	***0·070	***0·070	***0·060
	Vaccinated Male		1·149				
	Vaccinated 45–59			0·091			
	Vaccinated 60–74			Inf.			
	Vaccinated 75 ≤			Inf.			
	Vaccinated Hypertension				1		
	Vaccinated Diabetes					1·061	
	Vaccinated Obesity						1·926

Si Significance codes: *** < 0·001, ** < 0·001, * < 0·05.

VE was lower for these conditions. It has been suggested that both Covid-19 infection and mRNA-based vaccines promote an ACE2 platelet receptor imbalance (Angeli et al., 2021) and poorer sero-conversion in these population groups (Watanabe et al., 2021). We found that presence of any of these conditions is accompanied by a lower adjusted VE for infection. Adjusted VE rates for infection for participants with these conditions in this study were consistently lower here than those reported by Dagan et al. (2021) (Dagan et al., 2021) (hypertension: 89.7% vs 93%, diabetes: 88.9% vs 91%, obesity: 89.7% vs 95%). Again, we suggest that our data reflect general population VE, given the minimal exclusion criteria.

The results presented here measured vaccine effectiveness at a time when the British (alpha) variant was predominant in Israel. After implementing the national vaccination drive, infection rates dropped to very low levels (Leshem and Wilder-Smith, 2021), until the introduction of the delta variant in Israel. Test-negative case-control studies comparing BNT162b2 efficacy by variant have shown comparable (Nasreen et al., 2021) or slightly lower VE rates (Lopez Bernal et al., 2021) against infection among those infected with the delta variant compared to those infected with the British variant.

Mehrota et al. (2021) (Mehrotra et al., 2020) suggest a number of clinical endpoints for evaluating the effectiveness of the Covid-19 vaccines: symptomatic infection, burden of infection (proportion with high morbidity/mortality) and asymptomatic infection. In this study, infection was identified by PCR results, irrespective of reported symptoms. In most cases, it can be assumed that the test was carried out secondary to symptom presentation. However, an unknown number of those tested will have done so for other reasons, such as exposure to an infected individual, prior to medical procedure or travel overseas. Asymptomatic illness has been estimated as 20% (Izda et al., 2021). While the VE for infection in this study may capture a proportion of this population group, it is likely to be small. Length of follow-up period is also an important component of measuring vaccine effectiveness, where the literature ideally recommends two years of follow-up (Mehrotra et al., 2020). IgG antibodies typically drop dramatically after 16 months with non-activated viral vaccines (Izda et al., 2021).

This study provides data on a longer follow-up period but only long-term studies with alternate methodology will be able to shed light on length of protection the mRNA BNT162b2 vaccine provides. As pointed out in other studies where national drives were implemented to vaccinate the population quickly (Dagan et al., 2021), the longer the follow-up period, the greater the difficulty in finding an adequately matched unvaccinated population. Over 86% of the 60+ population in Israel have been fully vaccinated to date (Israel Ministry of Health, 2021). Those

that have not been vaccinated are likely to include those that had been infected with the illness (and therefore not eligible for two dose vaccination according to current protocol), or unwilling to vaccinate. Those unwilling to vaccinate may be different in their use of healthcare and preventive services.

As an observational study, differences between those presenting for vaccination and those that did not, cannot be fully accounted for. Factors not included in the model here may have introduced bias, such as presence of heart disease and other chronic illnesses, and health worker employee status. Furthermore, PCR tests were performed upon request, most commonly because of accompanying symptoms. Asymptomatic members, unaware of their infection status, are less likely to have been tested and thus included in the study. Their inclusion in the seemingly unprotected 'unvaccinated' group would reduce infection incidence for that group, whilst their inclusion in the 'vaccinated group' may inflate VE estimates.

Finally, in our study we included MHC members only, who tend to be younger and come from a higher socioeconomic bracket compared to general population in Israel. However, given the similar findings in Clalit, an HMO with a larger elderly and lower socio-economic bracket population encourages us to assume that the impact on VE may be minimal.

5. Conclusions

This large observational study is based on an HMO population with few exclusion criteria. Our findings showed high vaccine effectiveness rates for the total population, with slightly lower rates for the elderly and those suffering from hypertension, diabetes or obesity. Our findings are slightly lower than those published by the manufacturer of the vaccine (based on a more restricted population group) but comparable with a study from another large HMO and national data. This study confirms earlier findings that the mRNA BNT162b2 vaccine provides high levels of protection to all major segments of the population, including the elderly. Whilst vaccination does not eliminate the risk of hospitalization or death, the risks are significantly reduced. Physicians should encourage vaccination in the general population. Continued follow-up is required to determine long term effectiveness of the vaccine.

Funding and disclosure of conflict of interest

No formal funding or conflict of interest to report. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The study was carried out (sponsored) on behalf of Maccabi HealthCare Services for the purposes of evaluating the effectiveness of the vaccine among its members. The sponsor had no role in the study design, collection, analysis and interpretation of the data, writing of the article or decision to submit the report for publication.

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This is Exhibit "G" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

ORIGINAL ARTICLE

Waning Immunity after the BNT162b2 Vaccine in Israel

Yair Goldberg, Ph.D., Micha Mandel, Ph.D., Yinon M. Bar-On, M.Sc., Omri Bodenheimer, M.Sc., Laurence Freedman, Ph.D., Eric J. Haas, M.D., Ron Milo, Ph.D., Sharon Alroy-Preis, M.D., Nachman Ash, M.D., and Amit Huppert, Ph.D.

ABSTRACT

BACKGROUND

In December 2020, Israel began a mass vaccination campaign against coronavirus disease 2019 (Covid-19) by administering the BNT162b2 vaccine, which led to a sharp curtailing of the outbreak. After a period with almost no cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, a resurgent Covid-19 outbreak began in mid-June 2021. Possible reasons for the resurgence were reduced vaccine effectiveness against the delta (B.1.617.2) variant and waning immunity. The extent of waning immunity of the vaccine against the delta variant in Israel is unclear.

METHODS

We used data on confirmed infection and severe disease collected from an Israeli national database for the period of July 11 to 31, 2021, for all Israeli residents who had been fully vaccinated before June 2021. We used a Poisson regression model to compare rates of confirmed SARS-CoV-2 infection and severe Covid-19 among persons vaccinated during different time periods, with stratification according to age group and with adjustment for possible confounding factors.

RESULTS

Among persons 60 years of age or older, the rate of infection in the July 11–31 period was higher among persons who became fully vaccinated in January 2021 (when they were first eligible) than among those fully vaccinated 2 months later, in March (rate ratio, 1.6; 95% confidence interval [CI], 1.3 to 2.0). Among persons 40 to 59 years of age, the rate ratio for infection among those fully vaccinated in February (when they were first eligible), as compared with 2 months later, in April, was 1.7 (95% CI, 1.4 to 2.1). Among persons 16 to 39 years of age, the rate ratio for infection among those fully vaccinated in March (when they were first eligible), as compared with 2 months later, in May, was 1.6 (95% CI, 1.3 to 2.0). The rate ratio for severe disease among persons fully vaccinated in the month when they were first eligible, as compared with those fully vaccinated in March, was 1.8 (95% CI, 1.1 to 2.9) among persons 60 years of age or older and 2.2 (95% CI, 0.6 to 7.7) among those 40 to 59 years of age; owing to small numbers, the rate ratio could not be calculated among persons 16 to 39 years of age.

CONCLUSIONS

These findings indicate that immunity against the delta variant of SARS-CoV-2 waned in all age groups a few months after receipt of the second dose of vaccine.

From Technion–Israel Institute of Technology, Haifa (Y.G.), the Hebrew University of Jerusalem (M.M.), and the Israeli Ministry of Health (O.B., E.J.H., S.A.-P., N.A.), Jerusalem, the Weizmann Institute of Science, Rehovot (Y.M.B.-O., R.M.), the Gertner Institute for Epidemiology and Health Policy Research, Sheba Medical Center Tel Hashomer, Ramat Gan (L.F., A.H.), Tel Aviv University, Tel Aviv (A.H.), and Ben Gurion University, Beersheva (E.J.H.) — all in Israel. Dr. Goldberg can be contacted at yairgo@technion.ac.il or at the Faculty of Industrial Engineering and Management, Technion–Israel Institute of Technology, Haifa 3200003, Israel.

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A key to the containment of the coronavirus disease 2019 (Covid-19) pandemic is mass vaccination of the population. However, the success of this policy is challenged by breakthrough infection and disease in fully vaccinated persons. One potential cause of breakthrough infection is the emergence of new variants of concern[1] that escape immunity, thus reducing the effectiveness of the vaccine. Several studies investigating the effectiveness of the BNT162b2 vaccine (Pfizer–BioNTech) against the beta (B.1.351)[2,3] and delta (B.1.617.2)[4-6] variants showed only modest rates of breakthrough infection and disease, whereas other studies showed higher rates.[7,8]

A second potential cause of breakthrough infection is waning of the immunity conferred by the vaccine. Mass vaccination with the BNT162b2 vaccine began in December 2020, and little is known about waning immunity over time. A recent study on longer-term follow-up of the participants in the phase 2–3 randomized trial of the BNT162b2 vaccine[9] showed a reduction in vaccine efficacy from 96% (in the period of 7 days to <2 months after receipt of the second dose) to 84% (in the period of 4 months to approximately 7 months after receipt of the second dose), which indicated a decrease in protection by a factor of four (i.e., [100−84]÷[100−96]). Preliminary reports of waning effectiveness of the same vaccine have come from a health maintenance organization in Israel[10] and from the United States,[11] and a decrease in vaccine-induced neutralization titers during the first 6 months after receipt of the second dose of vaccine has been reported.[12]

Israel conducted a very successful vaccination campaign using the BNT162b2 vaccine.[13-15] Starting in December 2020, more than half the adult population received two doses of vaccine within 3 months. The vaccination campaign, together with social measures, led to a sharp curtailing of the outbreak. By May 2021, infection rates had decreased to a few dozen cases daily, most of which were in unvaccinated persons or in persons returning from abroad. However, the number of polymerase-chain-reaction (PCR) tests that were positive for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) started to rise exponentially in June 2021, with a substantial number of infections being reported in vaccinated persons (Fig. 1). This rise in community transmission was followed by a concomitant increase in the numbers of severe cases and deaths, in both the vaccinated and unvaccinated populations. Genetic analysis showed that as of June 2021, more than 98% of the positive cases in Israel were attributed to the delta variant.[16] In this study, we estimated the role of waning immunity in the observed breakthrough against the delta variant.

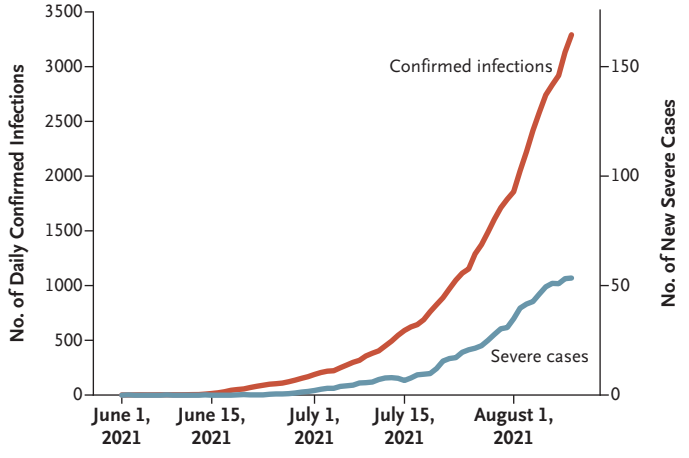

Figure 1. Daily Confirmed SARS-CoV-2 Infections and New Cases of Severe Covid-19 among Fully Vaccinated Persons in Israel, June through Early August 2021.

The graph shows increases in the numbers of daily severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections and new cases of severe coronavirus disease 2019 (Covid-19), on different scales, during the delta variant wave among persons who had received two doses of vaccine.

METHODS

DATA SOURCE

Data on all residents of Israel who had been fully vaccinated before June 1, 2021, and who had not been infected before the study period were extracted from the Israeli Ministry of Health database on September 2, 2021. We defined fully vaccinated persons as those for whom 7 days or more had passed since receipt of the second dose of the BNT162b2 vaccine. We used the Ministry of Health official database that contains all information regarding Covid-19 (see Supplementary Methods 1 in the Supplementary Appendix, available with the full text of this article at NEJM.org). We extracted from the database information on all documented SARS-CoV-2 infections (i.e., positive result on PCR assay) and on the severity of the disease after infection. We focused on infections that had been documented

in the period from July 11 through 31, 2021 (study period), removing from the data all confirmed cases that had been documented before that period. The start date was selected as a time when the virus had already spread throughout the entire country and across population sectors. The end date was just after Israel had initiated a campaign regarding the use of a booster vaccine (third dose). The study period happened to coincide with the school summer vacation.

We omitted from all the analyses children and adolescents younger than 16 years of age (most of whom were unvaccinated or had been recently vaccinated). Only persons 40 years of age or older were included in the analysis of severe disease because severe disease was rare in the younger population. Severe disease was defined as a resting respiratory rate of more than 30 breaths per minute, oxygen saturation of less than 94% while the person was breathing ambient air, or a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen of less than 300.[14] Persons who died from Covid-19 during the follow-up period were included in the study and categorized as having had severe disease.

During the study period, approximately 10% of the detected infections were in residents of Israel returning from abroad. Most residents who traveled abroad had been vaccinated and were exposed to different populations, so their risk of infection differed from that in the rest of the study population. We therefore removed from the analysis all residents who had returned from abroad in July.

VACCINATION SCHEDULE

The official vaccination regimen in Israel involved the administration of the second dose 3 weeks after the first dose. All residents 60 years of age or older were eligible for vaccination starting on December 20, 2020, thus becoming fully vaccinated starting in mid-January 2021. At that time, younger persons were eligible for vaccination only if they belonged to designated groups (e.g., health care workers and severely immunocompromised adults). The eligibility age was reduced to 55 years on January 12, 2021, and to 40 years on January 19, 2021. On February 4, 2021, all persons 16 years of age or older became eligible for vaccination. Thus, if they did not belong to a designated group, persons 40 to 59 years of age received the second dose starting in mid-February, and those 16 to 39 years of age received the second dose starting in the beginning of March. On the basis of these dates, we defined our periods of interest in half months starting from January 16; vaccination periods for individual persons were determined according to the time that they had become fully vaccinated (i.e., 1 week after receipt of the second dose). All the analyses were stratified according to vaccination period and to age group (16 to 39 years, 40 to 59 years, and ≥60 years).

STATISTICAL ANALYSIS

The association between the rate of confirmed infections and the period of vaccination provides a measure of waning immunity. Without waning of immunity, one would expect to see no differences in infection rates among persons vaccinated at different times. To examine the effect of waning immunity during the period when the delta variant was predominant, we compared the rate of confirmed infections (per 1000 persons) during the study period (July 11 to 31, 2021) among persons who became fully vaccinated during various periods. The 95% confidence intervals for the rates were calculated by multiplying the standard confidence intervals for proportions by 1000. A similar analysis was performed to compare the association between the rate of severe Covid-19 and the vaccination period, but for this outcome we used periods of entire months because there were fewer cases of severe disease.

To account for possible confounders, we fitted Poisson regressions. The outcome variable was the number of documented SARS-CoV-2 infections or cases of severe Covid-19 during the study period. The period of vaccination, which was defined as 7 days after receipt of the second dose of the Covid-19 vaccine, was the primary exposure of interest. The models compared the rates per 1000 persons between different vaccination periods, in which the reference period for each age group was set according to the time at which all persons in that group first became eligible for vaccination. A differential effect of the vaccination period for each age group was allowed by the inclusion of an interaction term between age and vaccination period. Additional potential confounders were added as covariates, as described below, and the natural logarithm of the number of persons was added as an offset. For each vaccination period and age group, an

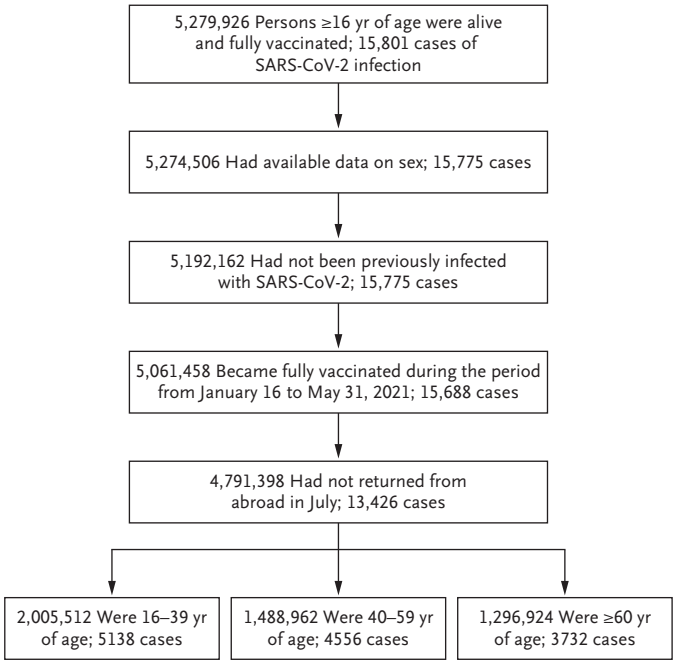

Figure 2. Study Population.

The population included persons who were fully vaccinated before June 1, 2021, were not abroad during July 2021, and had no documented SARS-CoV-2 infection according to polymerase-chain-reaction assay before July 11, 2021.

adjusted rate was calculated as the expected number of weekly events per 100,000 persons if all the persons in that age group had been vaccinated in that period. All the analyses were performed with the use of the glm function in the R statistical software package.[17]

In addition to age and sex, the regression analysis included as covariates the following confounders. First, because the event rates were rising rapidly during the study period (Fig. 1), we included the week in which the event was recorded. Second, although PCR testing is free in Israel for all residents, compliance with PCR-testing recommendations is variable and is a possible source of detection bias. To partially account for this, we stratified persons according to the number of PCR tests that had been performed during the period of March 1 to November 31, 2020, which was before the initiation of the vaccination campaign. We defined three levels of use: zero, one, and two or more PCR tests. Finally, the three major population groups in Israel (general Jewish, Arab, and ultra-Orthodox Jewish) have varying risk factors for infection. The proportion of vaccinated persons, as well as the level of exposure to the virus, differed among these groups.[18] Although we restricted the study to dates when the virus was found throughout the country, we included population sector as a covariate to control for any residual confounding effect.

We conducted several secondary analyses to test the robustness of the results, including calculation of the rate of confirmed infection in a finer, 10-year age grouping and an analysis restricted to the general Jewish population (in which the delta outbreak began), which comprises the majority of persons in Israel. In addition, a model including a measure of socioeconomic status as a covariate was fitted to the data, because this was an important risk factor in a previous study.[18] Since socioeconomic status was unknown for 5% of the persons in our study and the missingness of the data seemed to be informative, and also owing to concern regarding nondifferential misclassification (persons with unknown socioeconomic status may have had different rates of vaccination, infection, and severe disease), we did not include socioeconomic status in the main analysis. Finally, we compared the association between the number of PCR tests that had been conducted before the vaccination campaign (i.e., before December 2020) with the number that were conducted during the study period in order to evaluate the possible magnitude of detection bias in our analysis. A good correlation between past behavior regarding PCR testing and behavior during the study period would provide reassurance that the inclusion of past behavior as a covariate in the model would control, at least in part, for detection bias.

RESULTS

STUDY POPULATION

Among 5,279,926 fully vaccinated adults, we retained data on 4,791,398 persons for the main analysis (Fig. 2). Among these persons, 13,426 had a positive PCR test (confirmed SARS-CoV-2 infection) and 403 had severe Covid-19. Table 1 provides the number of events according to vaccination period, and Table S1 in the Supplementary Appendix provides a more detailed summary according to vaccination period and age group. Table 1 shows the characteristics of the study population according to vaccination period; Ta-

Table 1. Demographic and Clinical Characteristics of the Study Population According to Vaccination Period.*

Variable	Vaccination Period						
	Jan. 16–31 (N=1,076,708)	Feb. 1–15 (N=972,835)	Feb. 16–28 (N=747,788)	March 1–15 (N=819,040)	March 16–31 (N=749,422)	April 1–30 (N=325,201)	May 1–31 (N=100,404)
No. of positive SARS-CoV-2 PCR tests	3779	3182	2259	2146	1459	459	142
No. of cases of severe Covid-19	251	108	16	17	5	5	1
Male sex — no. (%)	518,196 (48)	459,251 (47)	380,135 (51)	410,371 (50)	358,398 (48)	153,619 (47)	46,352 (46)
Age group — no. (%)							
16–39 yr	125,977 (12)	195,961 (20)	352,722 (47)	549,090 (67)	496,779 (66)	217,731 (67)	67,252 (67)
40–59 yr	243,741 (23)	418,282 (43)	328,038 (44)	208,064 (25)	190,326 (25)	78,281 (24)	22,230 (22)
≥60 yr	706,990 (66)	358,592 (37)	67,028 (9)	61,886 (8)	62,317 (8)	29,189 (9)	10,922 (11)
No. of previous SARS-CoV-2 PCR tests — no. (%)†							
0	700,766 (65)	655,201 (67)	502,035 (67)	564,855 (69)	536,943 (72)	240,548 (74)	75,696 (75)
1	204,238 (19)	197,137 (20)	163,752 (22)	172,576 (21)	144,087 (19)	56,873 (17)	16,320 (16)
≥2	171,704 (16)	120,497 (12)	82,001 (11)	81,609 (10)	68,392 (9)	27,780 (9)	8,388 (8)
Population sector — no. (%)‡							
General Jewish	970,782 (90)	826,783 (85)	617,113 (83)	656,786 (80)	506,554 (68)	201,850 (62)	72,292 (72)
Arab	62,003 (6)	107,704 (11)	90,289 (12)	115,399 (14)	198,375 (26)	102,798 (32)	20,740 (21)
Ultra-Orthodox Jewish	43,923 (4)	38,348 (4)	40,386 (5)	46,855 (6)	44,493 (6)	20,553 (6)	7,372 (7)

* The numbers of persons in the column heads represent the numbers of persons who were fully vaccinated during that period. Positivity on the polymerase-chain-reaction (PCR) assay for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; i.e., confirmed infection) and cases of severe coronavirus disease 2019 (Covid-19) were assessed in the study period of July 11 to 31, 2021. Percentages may not total 100 because of rounding.
† Shown are the numbers of PCR tests that had been performed during the period of March 1 to November 31, 2020, which was before the initiation of the vaccination campaign.
‡ Population sector was determined on the basis of the area of residency, with the use of classifications provided by the Israeli Bureau of Statistics.

bles S2 through S4 show these data for each of the three age groups.

Because of the risk-based vaccination policy in Israel, persons who were vaccinated in January were older than those who were vaccinated later. In addition, the lower risk of Covid-19–related complications among younger persons may have caused a belief that vaccination was not urgent or even necessary, which also affected the age distribution of vaccination over the months.[19] The distribution of the number of previous PCR tests changed slightly between the periods, with 65% of the persons who became fully vaccinated in the second half of January having had no previous tests, as compared with 75% of those fully vaccinated in May. The number of tests seemed to be inversely correlated with age. A considerable difference was noted in the time of vaccination among the main population sectors: Arabs and ultra-Orthodox Jewish persons received vaccines later than did persons in the general Jewish population. Age and cultural differences contribute to these disparities.[18] (These differences in risk factors were adjusted for by their inclusion as covariates in the Poisson regression analysis.)

DESCRIPTIVE ANALYSIS

The rate of confirmed SARS-CoV-2 infection showed a clear increase as a function of time from vaccination. Among persons 60 years of age or older who were fully vaccinated in the second half of January, the rate was 3.3 confirmed infections per 1000 persons during the study period, as compared with 2.2 confirmed infections per 1000 persons who became fully vaccinated in the second half of February and 1.7 confirmed infections per 1000 persons fully vaccinated in the second half of March (Fig. 3A). Similar results were observed in the other age groups and when the analysis was categorized according to age in decades (Figs. 3A and S1). However, primarily health care workers and severely immunocompromised adults became fully vaccinated during the first three vaccination periods (January 16 to February 28) in the 16–39-year-old group and during the first two vaccination periods (January 16 to February 15) in the 40–59-year-old group; thus, the results for those vaccination periods in these age groups may be biased owing to selective samples and should be interpreted with caution.

A similar pattern was observed in the analysis of severe Covid-19 in the group of persons 60 years of age or older (Fig. 3B). In this analysis, vaccination periods were defined as January, February, March, and the combined April–May period because of the small numbers of severe cases in each age group. The rate of severe Covid-19 among persons 60 years of age or older who were fully vaccinated in January was 0.34 cases per 1000 persons over the study period and decreased to 0.26 cases per 1000 persons among those who were fully vaccinated in February, 0.15 cases per 1000 persons fully vaccinated in March, and 0.12 cases per 1000 persons fully vaccinated in the April–May period. The numbers of severe cases in the younger age groups were too small for conclusions to be drawn.

REGRESSION ANALYSIS

Tables 2 and 3 present the results of the regression analyses regarding confirmed SARS-CoV-2 infection and severe Covid-19, respectively; the complete set of estimated coefficients is provided in Tables S5 and S6. For each age group, the numbers in the tables show the ratios between the estimated rates in the first period when the persons in that group were eligible to become fully vaccinated (i.e., the second half of January for persons ≥60 years of age, the second half of February for those 40 to 59 years of age, and the first half of March for those 16 to 39 years of age) and the estimated rates in the other periods. The tables also include the adjusted rates for each vaccination period. In the group of persons 60 years of age or older, the rate of confirmed infection among those vaccinated in the second half of January was 1.1 times as high as the rate among those vaccinated in the first half of February. The rate ratio increased to 1.6 and 2.2 when comparing January vaccinees with those who were vaccinated in March and in April, respectively. The same phenomenon, of an increasing rate of confirmed infection with increased time since vaccination, was observed in all age groups.

Fewer cases of severe Covid-19 were noted in persons younger than 60 years of age, especially in the group of persons 16 to 39 years of age (Table S1), so the model could be fitted only to the groups of persons 40 to 59 years of age and those 60 years of age or older and only for the vaccination months of January through March.

The confidence intervals were wide; however, the results suggest a monotonic increase in the rate of severe disease as time since vaccination increased.

The analysis was repeated with socioeconomic status as an additional covariate, with the use of four categories (0 to 3 [indicating low socioeconomic level], 4 to 6 [indicating medium socioeconomic level], 7 to 10 [indicating high socioeconomic level], and unknown) and yielded similar results with only slightly smaller rate ratios (Table S8). Similar results were obtained when the analysis was restricted to the general Jewish population (Table S9).

DISCUSSION

The centralized health care system in Israel succeeded in vaccinating most of the Israeli population relatively early and in a short time.[13-15] This population is, therefore, useful for studying the effects of the BNT162b2 vaccine on the spread of SARS-CoV-2 infection and severity of Covid-19, as well as for studying the waning of vaccine protection over time. The appearance and rapid predominance of the delta variant in June 2021 resulted in a dramatic increase in the number of new SARS-CoV-2 infections among fully vaccinated persons, which aroused concern regarding

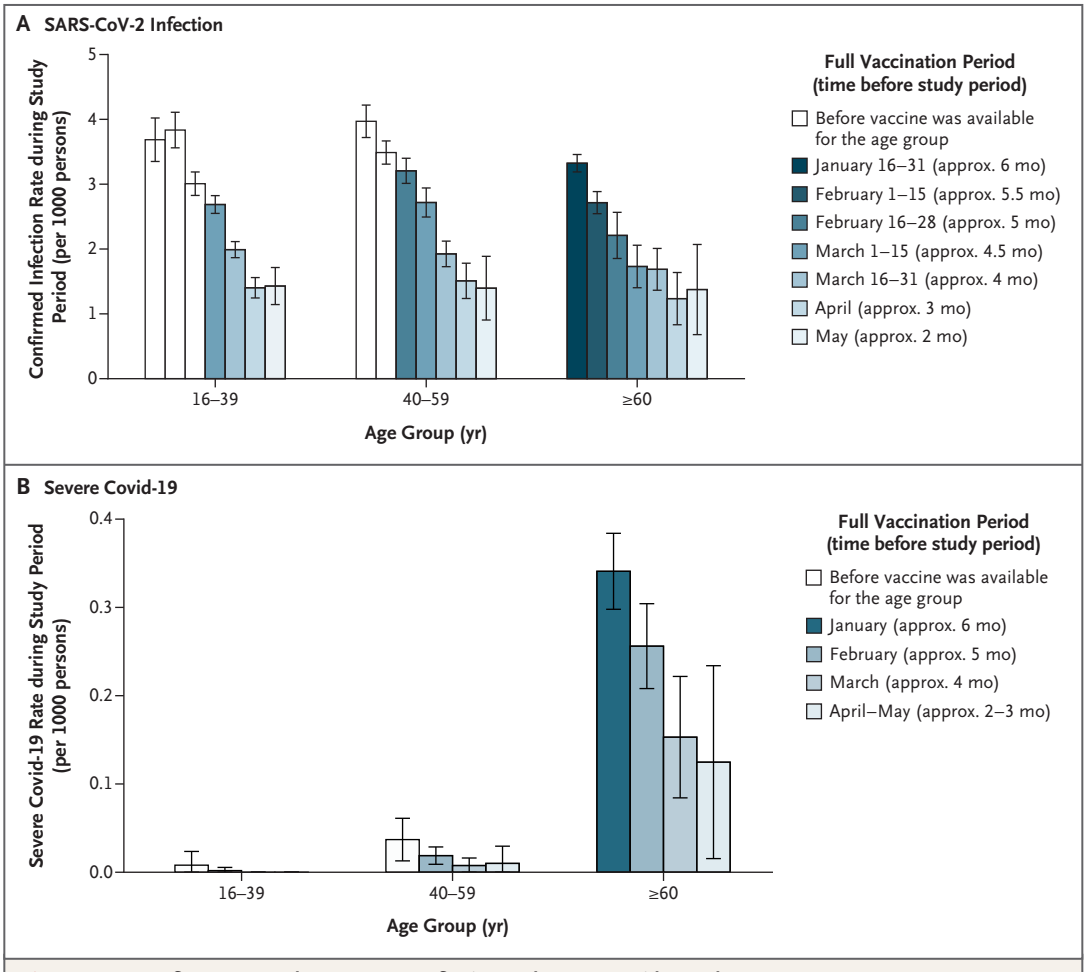

Figure 3. Rates of Documented SARS-CoV-2 Infection and Severe Covid-19, July 11 to 31, 2021.

Shown are the rates of documented infection per 1000 persons (Panel A) and rates of severe Covid-19 per 1000 persons (Panel B), according to period of second dose of Covid-19 vaccine and age group. In the analyses in the age groups younger than 60 years, white bars represent periods during which vaccination was restricted to only designated groups (e.g., health care workers and severely immunocompromised adults). I bars represent 95% confidence intervals, which are not adjusted for multiplicity. In Panel A, white bars represent half a month; in Panel B, white bars represent a month.

Table 2. Rate Ratios of Confirmed SARS-CoV-2 Infection According to Age Group and Vaccination Period.*

Age Group	Vaccination Period						
	Jan. 16–31	Feb. 1–15	Feb. 16–28	March 1–15	March 16–31	April 1–30	May 1–31
16–39 Yr							
Rate ratio of reference vs. period (95% CI)	0.8 (0.7–0.9)	0.7 (0.7–0.8)	0.9 (0.8–1.0)	Reference	1.2 (1.1–1.3)	1.5 (1.4–1.8)	1.6 (1.3–2.0)
Adjusted rate — no. of events/wk/100,000 persons	108.7	117.9	93.4	85.7	72.7	55.4	52.1
40–59 Yr							
Rate ratio of reference vs. period (95% CI)	0.9 (0.8–1.0)	1.0 (0.9–1.0)	Reference	1.1 (1.0–1.2)	1.4 (1.3–1.6)	1.7 (1.4–2.1)	2.1 (1.4–3.0)
Adjusted rate — no. of events/wk/100,000 persons	117.2	110.7	106.0	95.9	75.0	61.3	51.2
≥60 Yr							
Rate ratio of reference vs. period (95% CI)	Reference	1.1 (1.1–1.2)	1.3 (1.1–1.5)	1.6 (1.4–2.0)	1.6 (1.3–2.0)	2.2 (1.6–3.1)	2.2 (1.3–3.6)
Adjusted rate — no. of events/wk/100,000 persons	105.7	92.4	82.3	64.3	65.2	47.9	49.1

* Analyses were adjusted for week of infection, number of previous PCR tests (0, 1, or ≥2), population sector, and sex. Shown are rate ratios for confirmed SARS-CoV-2 infection during the period of July 11 through 31, 2021 (study period), as a function of time since full vaccination. We defined fully vaccinated persons as those for whom 7 days or more had passed since receipt of the second dose of the BNT162b2 vaccine. The comparison was between the estimated rate among persons who became fully vaccinated during the first vaccination period in which their age group was eligible (reference; i.e., January 16 to 31 for persons ≥60 years of age, February 16 to 28 for persons 40 to 59 years of age, and March 1 to 15 for persons 16 to 39 years of age) and the estimated rate among persons who became fully vaccinated in another vaccination period. For example, among persons 60 years of age or older, the rate of confirmed SARS-CoV-2 infection during the July 11–31 period among those vaccinated in January (105.7 events per week per 100,000 persons) was divided by the rate among those vaccinated in the second half of March (65.2 events per week per 100,000), yielding a rate ratio of 1.6. The 95% confidence intervals are not adjusted for multiplicity.

Table 3. Rate Ratios of Severe Covid-19 According to Age Group and Vaccination Period.*

Age Group	Vaccination Period		
	January	February	March
40–59 Yr			
Rate ratio of reference vs. period (95% CI)	0.6 (0.3–1.4)	Reference	2.2 (0.6–7.7)
Adjusted rate — no. of events/wk/100,000 persons	1.0	0.6	0.3
≥60 Yr			
Rate ratio of reference vs. period (95% CI)	Reference	1.2 (1.0–1.5)	1.8 (1.1–2.9)
Adjusted rate — no. of events/wk/100,000 persons	10.7	9.0	5.9

* For severe Covid-19, estimates are provided for the whole months of January, February, and March. Estimates are not provided for the youngest age group (16 to 39 years of age) and for the latest vaccination periods (April and May) because of very low case numbers. Analyses were adjusted for week of infection, number of previous PCR tests (0, 1, or ≥2), population sector, and sex. Shown are rate ratios during the period of July 11 through 31, 2021, as a function of time since full vaccination. The numbers in each age group are the ratios between the estimated rates in the first period when persons in that group were eligible to receive vaccination and the estimated rates in the other periods. The 95% confidence intervals are not adjusted for multiplicity.

decreased efficacy of the vaccine over time (Fig. 1).

A comparison of the rate of confirmed infection among persons vaccinated at different times revealed a clear increase in the rate as the time from vaccination increased in all age groups, with and without correction for measured confounding factors (Fig. 3A and Table 2). The rate of confirmed infection among persons 60 years of age or older who became fully vaccinated in the second half of January was 1.6 times as high as that among persons in the same age group who became fully vaccinated in March. The data show a similar increase in rate with increasing time since vaccination in the other age groups. The rate of severe Covid-19 cases also increased as a function of time from vaccination. Serologic studies in Israel have shown a correlated time-dependent reduction in neutralization titers,[12,20] which might be the biologic mechanism governing the observed waning immunity, and thus support the finding in this population-based research.

In contrast to early findings from the United Kingdom,[5] approximately two thirds of the cases of severe Covid-19 in Israel during the study period occurred in persons who had received two doses of the BNT162b2 vaccine. Two major differences exist between the studies. First, the current analysis used data from July 2021, a time when, for most of the Israeli population, at least 5 months had passed since receipt of their second dose of vaccine. The U.K. data were collected during the period of April through June 2021, with a much shorter time from vaccination to infection. Second, Israel has followed the original Pfizer–BioNTech protocol of administering the second dose 3 weeks (21 days) after the initial injection in most recipients, whereas the time between doses in the United Kingdom has typically been longer.[6]

A comparison of vaccinated persons with unvaccinated persons is of interest in order to predict the future burden on the health system. We therefore obtained data on the entire Israeli population from the Israeli Central Bureau of Statistics and calculated the number of unvaccinated persons indirectly. Moreover, unvaccinated persons might differ from the vaccinated population in important characteristics that could result in biased estimates. Nevertheless, we estimated the effectiveness of the vaccine against confirmed SARS-CoV-2 infection (see Supplementary Analysis 1). Vaccinated persons were found to be protected even after 6 months, as compared with unvaccinated persons. However, vaccine effectiveness was considerably lower than it had been closer to the vaccination date. Our findings are in line with findings from the randomized trial of the BNT162b2 vaccine, which showed a reduction in vaccine efficacy against symptomatic infection from 96% in the first 2 months after vaccination to 84% at 4 to 7 months after vaccination, when averaged over all age groups combined.[9]

Observational studies are subject to confound-

ing bias and detection bias. We examined these biases by using different sensitivity analyses (see Supplementary Analysis 2) and obtained similar results. Nevertheless, some sources of bias might remain; for instance, any effects that were due to differences in coexisting conditions between the vaccination periods could not be controlled for, because coexisting conditions are not recorded in the national database.

We did not separate the contribution of vaccine breakthrough due to waning immunity from the contribution due to the change in the dominant variant from alpha (B.1.1.7) to delta. Our analysis showed only the clear effect of waning vaccine-induced immunity against the delta variant. In addition, we were not able to quantify the extent of waning in the months immediately after vaccination (when the prevalence was extremely low in Israel).

Understanding the extent of waning immunity is critical for policy making, especially regarding vaccination strategies. The results presented here provided an epidemiologic basis for the decision by the Israeli Ministry of Health on July 30, 2021, to approve the administration of a booster (third dose) of Covid-19 vaccine to persons who had been vaccinated at least 5 months previously. The findings also suggest the need to follow the effects of waning immunity closely and to inform policymakers worldwide who are facing decisions regarding the administration of booster vaccinations.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

We thank Ofra Amir, Ronen Fluss, Sarah Goldberg, Boaz Lev, Ami Mizrahi, Geert Molenberghs, Rami Yaari, and Arnona Ziv for fruitful discussions.

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This is Exhibit "H" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

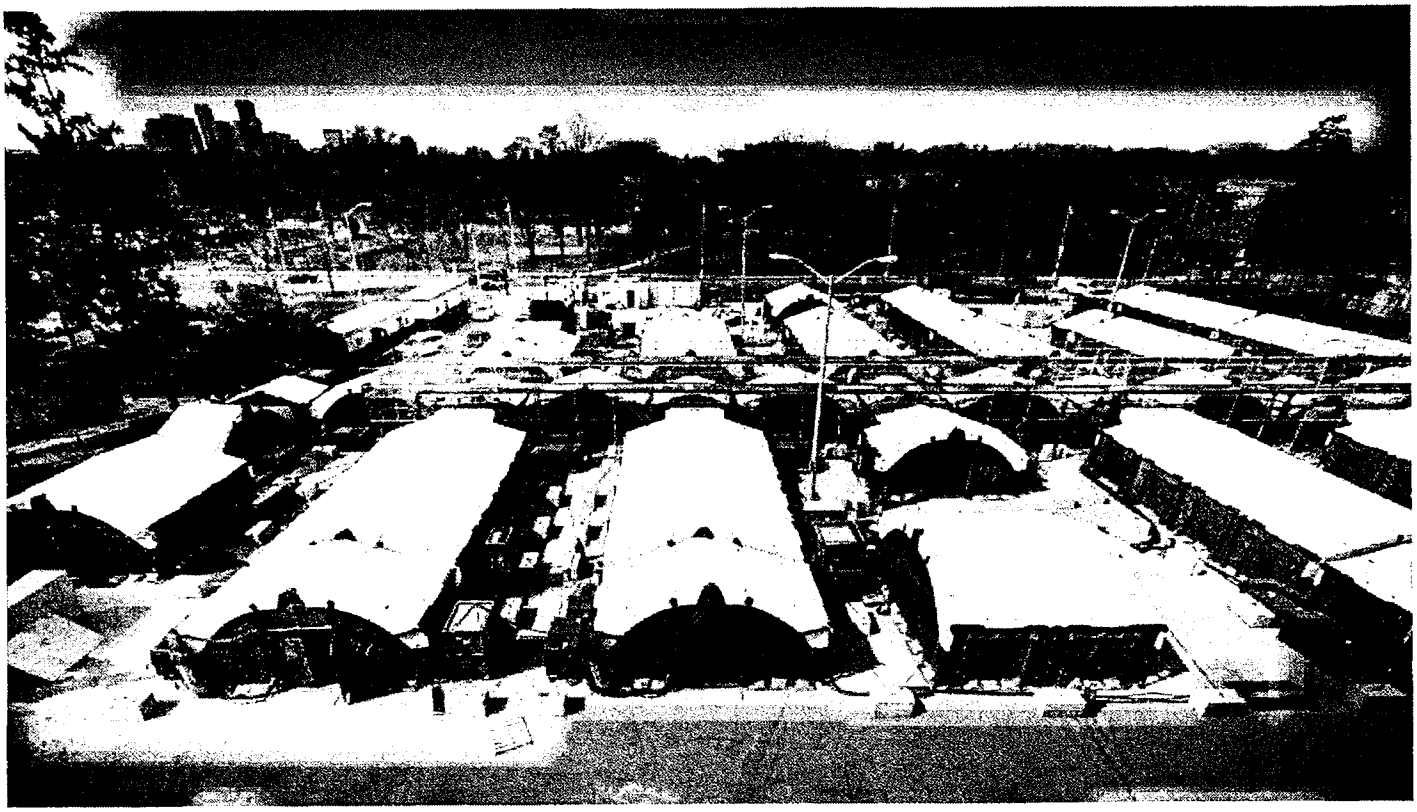
KATHERINE R. COSTIN

Toronto

New Toronto field hospital prepares to accept COVID-19 patients as ICUs overflow

'Last pieces' of tents in parking lot being put into place for mobile health unit, Sunnybrook says

Sannah Choi, Muriel Draaisma · CBC News · Posted: Apr 20, 2021 4:00 AM EDT | Last Updated: April 20, 2021



A Toronto field hospital, set up in a parking lot, is getting ready to accept COVID-19 patients. Sunnybrook Health Sciences Centre says its mobile health unit will likely open this week. (Evan Mitsui/CBC)

156 comments

A new field hospital built in a parking lot at Sunnybrook Health Sciences Centre in Toronto will likely be ready to accept patients this week as hospitals across the region try to deal with a record spike in COVID-19 caseloads.

The mobile health unit, as it is officially known, will provide care to patients who are recovering or have recovered from COVID-19. The unit will allow Sunnybrook to free up acute-care beds in hospitals during the third wave of the pandemic.

Contained in a series of green tents, supported by an aluminum frame, the 2,088-square-metre unit has 84 patient beds, with room to expand to 100 beds if needed. The hospital hopes to open 20 beds in the unit this week, according to the manager in charge of the temporary medical facility.

Robert Burgess, Sunnybrook's senior director of prehospital medicine, patient flow and emergency preparedness, said on Monday that the hospital is putting the finishing touches on the unit this week.

"We're literally at the last pieces in terms of the structural setup," Burgess said while wearing a mask inside the unit.

The unit is being prepared at a time when GTA hospitals are so overwhelmed due to record COVID-19 admissions that some patients are being transferred to other health-care centres outside the region, including southwestern Ontario.

A view of the inside of the mobile health unit at Sunnybrook Health Sciences Centre in Toronto on April 19, 2021. (Kevin Van Paassen/Sunnybrook Health Sciences Centre)

"We're preparing this week, as quickly as possible, to start bringing patients in," Burgess added.

"Obviously, when you open a new hospital, or you open a ward in a hospital, there's a lot of work to do around staffing plans. We're working through those to ensure we're doing this in a safe fashion," he added.

"Literally, with each hour that is passing, we're becoming closer and closer to the point where we can start to bring in patients on a routine basis. The hope is that we can start to bring some patients in this week. If it's safe to do so, we will proceed with that. We're all eager to start that process."

WATCH | Toronto field hospital 'tool in the toolbox' for surging patient load, says emergency planner.

Toronto field hospital 'tool in the toolbox' for surging patient load, says emergency planner

2 years ago | 8:02

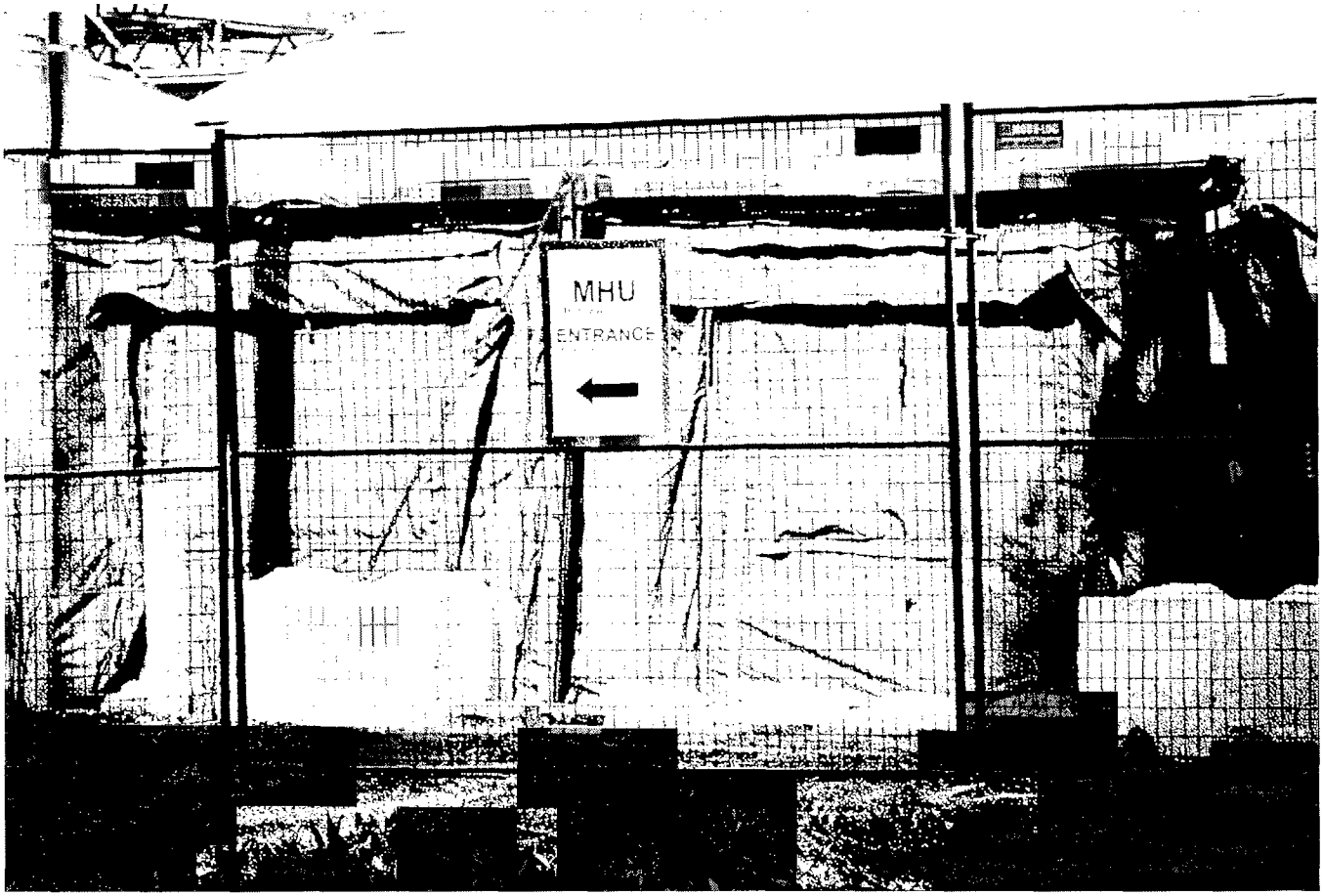
An 84-bed field hospital at Toronto's Sunnybrook Health Sciences Centre is close to being ready to accept recovering COVID-19 patients, says Robert Burgess, senior director of emergency preparedness at the hospital.

Burgess said the hospital wants to avoid bringing critically ill patients into the unit.

Each pod of eight-to-10 beds is self-contained. Several large generators provide power for the unit. He said there are many windows to provide light.

He said "it would be great" if pressures eased on the health-care system in Ontario, the hospital did not need to use the unit and could dismantle it soon.

"It's meant to be here as another tool in the toolbox for emergency preparedness. That is something that we would hope, but we're ready to help the province if it turns otherwise," he said.



Contained in a series of green tents, supported by an alumimum frame, the 2,088-square-metre unit has 84 patient beds, with room to expand to 100 beds if needed. (Evan Mitsui/CBC)

Burgess said staffing is being finalized with the help of the Ontario health ministry. The hospital is currently training staff members, providing orientation and doing simulations inside the unit to ensure people feel comfortable working there. "We are looking everywhere to find additional staff," he said.

He described the unit as a "system resource," which means it could provide relief to hospitals in other areas of the province where there are pressures on the health-care system.

Sunnybrook has been asked to ensure the unit will be up and running for a minimum of three months. That time period could be extended depending on needs and patient volumes, he said.

- **Ontario sees 4,447 new COVID-19 cases as admissions to ICUs top 750**
- **Trudeau pledges more health-care workers, rapid testing for pandemic-battered Ontario**

160

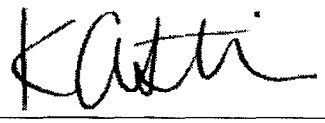
Burgess acknowledged that the unit looks like a number of tents from the outside and can be startling to see, but said the unit is sophisticated on the inside.

"These are structures that were developed for medical purposes. Once you're in, it's very sophisticated, it's very safe and very comfortable," he said.

"We've designed the structure to be safe for patients and staff. Hopefully, patients and staff will be pleasantly surprised when they see the inside for the first time."

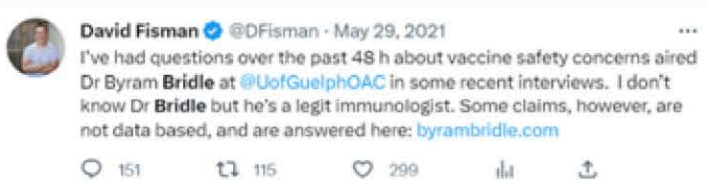
An under-construction field hospital on the grounds of Sunnybrook Heath Sciences Centre, in Toronto, is pictured on Apr. 6, 2021. (Evan Mitsui/CBC)

This is Exhibit "I" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.



Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN



This is Exhibit "J" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Dr. Byram Bridle

BYRAM BRIDLE IS A "VIRAL IMMUNOLOGIST WHO IS PASSIONATE ABOUT IMPROVING LIFE" ... ALBEIT NOT BY REDUCING THE SPREAD OF COVID-19 MISINFORMATION

WHO IS BYRAM BRIDLE?

Dr. Byram Bridle is an associate professor at the Ontario Veterinary College at the University of Guelph in Guelph, Ontario, Canada. **Dr. Bridle is not a medical doctor**. He conducts research on animals.

Dr. Bridle obtained a PhD in immunology under the supervision of Dr. Bonnie Mallard. They were early and prominent members of the **Canadian COVID Care Alliance**.

According to court filings, Dr. Bridle has **never treated** a (human) infectious disease, he has **never performed** a (human) childhood vaccination, **nor has he ever treated** a (human) adverse reaction to vaccine.

Dr. Bridle received funding to **develop his own COVID-19 vaccine**, raising questions of a **conflict of interest.**.

WHAT DOES HE CLAIM?

Bridle is most well known for **his claims about the biodistribution and 'toxicity' of the spike prote**

Bridle is also known for:

- Making similar claims on Fox News' *The Ingraham Angle*.
- Claiming he was being censored at a *televised press conference* on Canada's Parliament Hill. The press conference was organized by far-right politician Derek Sloan (who Bridle endorsed for Premi a month earlier).

RESPONSES TO HIS CLAIMS

FROM HIS COLLEAGUES

More than 80 of Dr. Bridle's colleagues at the University of Guelph signed a letter denouncing Bridle's ideas as misinformation.

- Being a special guest of (and speaking at an event organized by) far-right German politician Christine Anderson, whose views were called 'vile' by Conservative Leader Pierre Poilievre.
- His support of (and appearance at) the 'Freedom Convoy'.
- His opposition to many of the widely accepted COVID-19 public health interventions.

Bridle's claims were quickly rebutted by peers, fellow scientists, media watchdogs, and in the legal system. Despite claiming to be an advocate for free speech and debate, Bridle characterized these responses as censorship, defamation, and harassment.

> *"Therefore, we wish to state publicly that as scientists, faculty, and/or staff of the University of Guelph we stand firmly against the continued spread of factually incorrect and misleading information that is being disseminated by Dr. Bridle. We have confidence that the SARS-CoV-2 vaccines approved for use in Canada are safe and effective, and we wish to reassure the public that as members of the University of Guelph community we fully support evidence-based public health, which includes vaccination against COVID-19."*
>
> — Faculty and staff at the University of Guelph support SARS-CoV-2 vaccine saf

FROM THE MEDIA

15 articles were written refuting his (and similar) claims.

- Byram Bridle's claim that COVID-19 vaccines are toxic fails to account for key differences between spike protein produced during infection and vaccination, misrepresents studies. -- Health Feedbac
- This professor spreads false information about the consequences of corona vaccinations (*Dieser Professor verbreitet falsche Informationen über die Folgen von Corona-Impfungen*). -- AFP
- FALSE: Conspiracy theory that COVID-19 vaccines' spike proteins are 'cytotoxic' debunked by expe -- Fox2now
- Laura Ingraham guest pushes debunked claims that the COVID-19 vaccines are a "toxin". -- Media Matters
- Immunologist's misinterpretation of data fuels misleading Covid-19 vaccine claims. -- AFP
- Fact Check: Veterinary Professor Does NOT Prove Vaccine Causes Buildup Of Toxins. -- Lead Stories
- 'Toxic' spike protein claims misinterpret vaccine study. -- AAP
- No sign spike proteins from COVID-19 vaccines, including Novavax, are dangerous. -- PolitiFact
- Fact Check - No evidence spike proteins from COVID-19 vaccines are toxic. -- Reuters
- Claims that Covid vaccine spike proteins are harmful are unevidenced. -- Full Fact
- No sign that the COVID-19 vaccines' spike protein is toxic or 'cytotoxic'. -- PolitiFact
- Posts misrepresent US study on dangers of coronavirus spike protein. -- AFP
- Fact check: COVID-19 vaccines don't produce dangerous toxins. -- USA Today
- Fact Check - COVID-19 vaccines are not 'cytotoxic'. -- Reuters
- Spike protein produced by vaccine not toxic. -- AP

IN THE LEGAL SYSTEM

Bridle has served as an 'expert witness' in several legal cases opposing COVID-19 public health interventions.

" [238] The court accepts that Dr. Bridle is an immunologist and vaccinologist by training and that he has expert knowledge in these fields, in particular regarding the theory and science behind vaccines. ***However, for the reasons below, the court does not accept that Dr. Bridle is qualified to give opinion evidence with respect to the safety and efficacy of the Covid-19 vaccine for children****. ...*

[240] Dr. Bridle acknowledged that he is not a medical doctor. ***He has never vaccinated a child, he has never treated a child or an adult suffering from a reaction to a vaccine, nor has he ever treated a child or an adult who is suffering from an infectious disease.*** *...*

He did not seem to appreciate or accept the serious risks associated with contracting the Covid infection, including death, nor did he seem to appreciate the risks and effects of contracting Covid multiple times, including the risk of developing long Covid syndrome, of which he appeared to be oblivious. ...

[249] When asked by the court if he accepted that the Covid vaccine prevents serious illness and death, regardless of the shorter duration of immunity, Dr. Bridle would not acknowledge that receiving the vaccine prevented severe or serious illness and death. ...

[250] ***Respectfully, this is so far removed from the mainstream and widely accepted views of the Canadian and international medical and scientific community that the court cannot accept Dr. Bridle's evidence on the Covid vaccine as reliable****. ...*

[254] Dr. Bridle also testified that he is working on his own Covid vaccine, for which he has received government funding and is currently in the pre-clinical stage. ***The court was concerned that it is possible in Dr. Bridle's interest, consciously or not, to advance views that discredit the existing mRNA technology used in Covid vaccines because he is working on a competing technology****. "*

— B.C.J.B. v. E.-R.R.R., 2022 ONCJ 500 (Can

" [22] ***Dr. Bridle is neither a physician nor a veterinarian, and accordingly has had no experience in treating patients of any kind, including in relation to Covid-19****. Dr. Leis describes Dr. Bridle as a 'bench scientist' with expertise in 'immunology focused mainly on the pre-clinical development of therapies that can stimulate the immune response to fight cancer,'*

...

In discussing Dr. Bridle's report in this case, Dr. Leis says, for example, that:

*>> '****In reviewing Dr. Bridle's report, there are numerous scientific inaccuracies throughout the document and it would simply not be possible to address all of them in a succinct report****. However, before addressing the 16-points in the conclusion, a few major corrections should be noted that go against accepted medical science.'*

[37] This is a remarkable and singular kind of criticism to find in an expert report. While experts often vehemently disagree with one another's conclusions, it is rare to find an expert condemning the opposite expert's basic scientific premises in such emphatic language. ***In the discussion below, accordingly, I approach Dr. Bridle's views with caution, and carefully consider them against the backdrop of what Drs. Leis and Vaisman characterize as well-founded and generally accepted scientific concepts****. ...*

[101] More concerning, Seneca's experts note that there is no evidence that the vaccines potentially cause long term neurological damage, as Dr. Bridle alleges, and they observe that ***he overstates other risks of the vaccines and omits or ignores important studies in the area****.*

[102] Given the circumscribed nature and extent of Dr. Bridle's expertise, the fact that ***expresses opinions well outside the parameters of his expertise and apparently at odds with the prevailing state of medical and scientific knowledge, I prefer the opinions of Drs. Leis and Vaisman, and cannot accept Dr. Bridle's opinions underpinning the applicants' arguments described above****. "*

ABOUT THIS WEBSITE

GET IN TOUCH

— Costa, Love, Badowich and Mandekic v. Seneca College of, 2022 ONSC 5111 (CanLII)

Hi there 👋

I'm a Concerned Scientist. I created this website in response to the alarmist and misleading claims being made by Dr. Bridle about COVID-19 and the COVID-19 vaccines. Let's set the record straight:

- I am the sole author and creator of this website.
- I do not receive — nor have I ever received — any income or compensation related to this website.
- I have no conflicts of interest to declare with respect to the contents of this website.
- Byram Bridle does not own — nor has ever owned — byrambridle.com.
- No person or persons mentioned herein are affiliated with byrambridle.com

I do not share my name because of the vitriol of the anti-vaccine community, and their use of Strateg Lawsuits Against Public Participation (SLAPPs) to silence free speech (despite robust anti-SLAPP laws

I ❤ free speech.

Thank you to those who take the time to submit constructive feedback.

This is Exhibit "K" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Dr. Byram Bridle

Byram Bridle is a "viral immunologist who is passionate about improving life"... albeit not by reducing the spread of COVID-19 misinformation

Get Some COVID-19 Vaccine Facts **Learn More About Dr. Bridle's Claims**

Here are some examples of Dr. Bridle's claims...

General Claims About the Spike Protein

Bridle has made a number of claims about the spike protein — the protein that our bodies create from the mRNA instructions in the vaccine. He says the spike protein from the vaccine leaves the deltoid (your arm muscle), and...

- accumulates in the blood, spleen, and liver (it doesn't)
- accumulates in the bone marrow, and the adrenal glands (it doesn't)
- accumulates in the ovaries (it really doesn't)

Bridle seems to be confusing a number of different studies which have detected the spike protein in the body after a SARS-COV-2 infection, with what happens after you get the vaccine.

Had he walked down the hall, and consulted with his colleagues who have written extensively on this topic, they could tell him that the data shows no significant amount of the vaccine enters the circulation.

That's not only because the vaccine is injected into your muscle (not your blood stream), but because it's been given what's called a "transmembrane anchor" to keep it attached to the surface of your cells.

And, it's been inactivated — this isn't the same as the actual spike protein.

Claims About Who Should be Worried

Bridle has claimed — along with other proponents of "natural herd immunity" — that the risk of COVID-19 is overblown, people are too scared of the virus, and that we should end lockdowns and let the virus run its course through our vunerable populations.

Strangely, now he wants you to be afraid; he claims the spike protein can...

- pass through breast milk and harm infants (it can't)
- pass through blood donation and harm recipients (it can't)

There is simply no evidence for either of these claims. What his is proposing is that the spike protein may be passed to someone in a vunerable group (like the elderly) and injure them.

He seems to be ignoring that (as of this writing) we've already vaccinated over 90% of 80+ year olds in Canada. So they've already been exposed to this spike protein — and they're perfectly healthy.

Claims That "We" Made a Big Mistake

Bridle claims that "we made a big mistake — we didn't realize ... the spike protein itself was a toxin, and a pathogenic protein..."

Considering there's no evidence for these claims, one might wonder why Bridle thinks "we" made a mistake. Why would he want us to feel the vaccines aren't safe?

It's interesting to note that Bridle's lab was given $230,000 from the Ontario government to develop a competing vaccine he hoped to have ready in 4 years, that likely won't be needed if the existing vaccines succeed.

And who is Alex Pierson?

Bridle made these claims on Alex Pierson's radio show, on which he is a regular guest. I don't know much about Alex, because this is 2021 and nobody owns a radio, but here are some of her more recent works:

March 31, 2021

...Should we trust the WHO report on the origins of COVID-19?...

March 23, 2021

170

Are the lockdown measures just as deadly as the virus itself?

March 12, 2021

Why are mental health experts telling all levels of government Everything is Not Ok? Will China ever be held accountable for lies surrounding Covid?...

Feb. 19, 2021

A secretive and conflicted federal vaccine task force...

Feb. 2, 2021

Civil liberties are being sacrificed to help curb the spread of COVID-19, but to what end? And a new book by a former liberal MP that Trudeau might not want you to read

Dec. 11, 2020

Why did Trudeau invite the Chinese military to see our military secrets? Why don't we have rapid testing in place yet? And a conversation with Trudeau's half brother

Jul. 7, 2020

Environmentalist Michael Shellenberger apologizes for the "climate scare"

Thanks for Reading.

Remember kids, get vaccinated, and always buy your own domain name.

Get Some COVID-19 Vaccine Facts **Cheers!**

This is Exhibit "L" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

← Tweet

David Fisman
@DFisman

An excellent follow for good immune science from @UofGuelphOAC is Dr @glenpyle , who has addressed some of the misinformation in these interviews in his own tweets

> **Glen Pyle | #WomensHeartHealth** @glenpyle · May 29, 2021
> Replying to @nametobe1 @DFisman and @UofGuelphOAC
> The paper Byram cited doesn't support his claim. That's pretty telling that a study cited to support his claims actually goes against those claims.

7:23 AM · May 30, 2021

10 Retweets **36** Likes **4** Bookmarks

This is Exhibit "M" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

← Thread

David Fisman
@DFisman

The website debunking Dr. Bridle's covid-19 vaccine claims has been updated with lots of peer-reviewed science that attests to the safety of vaccines.
byrambridle.com
And for those who think I made or organized this website: nope. But grateful to the scientists who did.

2:25 PM · May 31, 2021

116 Retweets **33** Quotes **493** Likes **51** Bookmarks

David Fisman @DFisman · May 31, 2021
A friend indicates that Dr Bridle's interview caused his parents to cancel their vaccine appointments. This is not ok.

16 12 196

This is Exhibit "N" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

From: David N. Fisman <david.fisman@gmail.com>
Sent: 02 June 2021 12:46 PM
To: Funke, Daniel <DFunke@usatoday.com>
Cc: david.fisman@utoronto.ca <david.fisman@utoronto.ca>; J. Scott Weese <jsweese@uoguelph.ca>; Amy Greer <agreer@uoguelph.ca>; Glen Pyle <gpyle@uoguelph.ca>
Subject: Re: Media request from USA TODAY (Deadline: 5 p.m. ET)

Hi Daniel

I am actually headed into a busy afternoon and will answer this briefly but I don't think I can really do justice to this topic. Bridle (who I don't know) appears to be distorting scientific evidence that's emerging on covid vaccines to suggest that they are unsafe. In fact, what we are seeing right now with the massive rollout of mRNA vaccines is that they seem remarkably safe. Bridle is suggesting that a study that noted minuscule quantities of spike protein in blood after first dose represent a health hazard. That is poppycock: biologically implausible and not data based.

His assertions re: accumulation in ovaries is based on an odd document, the original of which is apparently not available, and which looks like it may be a safety study for lipid nanoparticles, not spike as Bridle asserts. This is some kind of drug safety study in rats, not people, and it's not possible to assess what was being done here, or even whether it's a real document.

This seems to be an organized disinformation op aimed at undermining vaccine confidence. I have no idea what the motivation would be behind such and exercise or who would be organizing this. I know that a number of us have been concerned that Dr Bridle (oddly for an immunologist with vaccine funding) seems to have been headed more and more in an "anti-vax" direction since last August.

As I don't have the ability to answer at greater length right now I have cc'd some colleagues from U of Guelph (all of whom are more knowledgeable than I am) who might be able to add to the conversation.

You may also want to check out https://byrambridle.com

D

Sent from my iPhone

> On Jun 2, 2021, at 12:14 PM, Funke, Daniel <DFunke@usatoday.com> wrote:
>
> Hi David,

I hope you're doing well. I'm a reporter for USA TODAY and I'm reaching out for your insight for a fact check I'm writing about the coronavirus vaccines.

I'm fact-checking a claim that coronavirus spike proteins resulting from the vaccines are dangerous toxins that linger in the body and potentially cause side effects. Here's an example of the articles I'm fact-checking: https://www.lifesitenews.com/news/vaccine-researcher-admits-big-mistake-says-spike-protein-is-dangerous-toxin

The source of the claim appears to be an interview with Byram Bridle, an associate professor at the University of Guelph. I saw you tweeted about him recently and would love your thoughts on this recent claim.

Is there any evidence to suggest the spike proteins that result from COVID-19 vaccines are dangerous toxins that adversely affect vital organs? What do you make of Bridle's claims in general?

I expect to file this fact check today around 5 p.m. ET.

Thanks so much!

<image003.png>

Daniel Funke

Fact Check Reporter

dfunke@usatoday.com

(813) 364-4369

This is Exhibit "O" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

FACT CHECK

fact-checking Add Topic

Fact check: COVID-19 vaccines don't produce dangerous toxins

Daniel Funke
USA TODAY

Published 11:14 p.m. ET June 8, 2021

The claim: Spike proteins from coronavirus vaccines are dangerous toxins that cause damage in the body

A Canadian immunologist who says he's "pro-vaccine" has recently become the source of misinformation about the safety of coronavirus vaccines.

Text in a June 3 Instagram photo says the coronavirus spike protein resulting from vaccination is a "toxin." The post cites a "doctor" as evidence.

"Doctor on COVID Vax: 'We Screwed-Up. We didn't realize the Spike Protein is a TOXIN,'" the text says. "Does this mean everyone vaccinated is manufacturing their own Spike Protein Toxins in their own bodies?"

The post is one of dozens of similar claims that have circulated on Facebook and Instagram over the past few weeks, according to CrowdTangle, a social media insights tool. The most widely shared version stemmed from a May 31 article by LifeSite News, which has previously made false claims about the safety of coronavirus vaccines.

The Instagram photo is a screenshot of a May 31 headline from the Hal Turner Radio Show. Turner, a far-right radio host, has previously published false claims about coronavirus vaccines on his website.

The spike protein is located on the surface of the coronavirus and is used by the virus to enter human cells. All three coronavirus vaccines approved for emergency use in the United States teach the body how to make antibodies against the spike proteins, eliciting an immune response.

Fact check: Moderna executive did not say mRNA vaccines alter recipient's DNA

The "doctor" the Hal Turner Radio Show and other websites cited is Byram Bridle, a viral immunologist and an associate professor in the Ontario Veterinary College at the University of Guelph. In a May 27 interview with Canadian broadcaster Alex Pierson cited in the Hal Turner Radio Show story, Bridle cast doubt on the safety of coronavirus vaccines by saying spike proteins are toxins that cause cardiovascular damage in vaccinated people.

"We made a big mistake," he said. "We never knew the spike protein itself was a toxin and was a pathogenic protein. So by vaccinating people, we are inadvertently inoculating them with a toxin. In some people, this gets into circulation, and when that happens in some people it can cause damage — especially in the cardiovascular system."

Bridle said his claims were "completely backed up by peer-reviewed, scientific publications."

But they're not.

An author of the study Bridle cited during the interview said Bridle "over-interpreted" its results. And several of Bridle's colleagues told USA TODAY his claims about spike proteins are wrong.

Public health officials say the coronavirus vaccines, which millions of Americans have received, are safe and effective at preventing severe COVID-19 cases.

Fact check: Peer-reviewed studies have shown safety, efficacy of COVID-19 vaccines

"Bridle is suggesting that a study that noted minuscule quantities of spike protein in blood after first dose represent a health hazard," David Fisman, an epidemiology professor at the University of Toronto, said in an email. "That is poppycock: biologically implausible and not data-based."

USA TODAY reached out to the Instagram user who shared the post for comment.

Vaccines teach body to make spike proteins

First, let's review how the coronavirus vaccines work.

Two vaccines approved for emergency use in the U.S., one from Pfizer-BioNTech and another from Moderna, use messenger RNA (mRNA) technology to inoculate people against the coronavirus.

More traditional vaccines contain weakened or inactivated viruses, that aren't capable of causing infection or disease themselves, to build up the body's immune response.

mRNA vaccines don't work like that. Instead, they carry genetic material with instructions that tell cells how to produce a protein or a piece of protein, which in turn activates the body's immune response and causes the production of antibodies.

All three vaccines approved for emergency use in the U.S. teach cells how to create the spike protein present on the surface of the coronavirus. The body then produces antibodies until all the spike proteins are destroyed, building up immunity for future coronavirus infections.

Carolyn Coyne, a professor of molecular genetics and biology at Duke University, previously told USA TODAY that spike proteins do stay in the body for some time. But the proteins are eventually broken down, and the vaccines are constructed in a way that limits the ability of the proteins to fully bind to cells and create more infectious particles.

"There is no scientific data to indicate that the spike protein is toxic or that it lingers at any toxic level in the body after vaccination," Abby Capobianco, press officer for the FDA, said in an email.

Fact check: No, the CDC did not release data showing 7 in 10 Americans are declining COVID-19 vaccine

The coronavirus vaccines are safe and effective at preventing serious coronavirus infections, according to data from clinical trials involving more than 100,000 participants. Officials briefly paused use of the Johnson & Johnson vaccine in April after some people who received it developed a rare and serious kind of blood clot. But the FDA and the U.S. Centers for Disease Control and Prevention say that vaccine's "known and potential benefits outweigh its known and potential risks."

Study author, Pfizer refute claims

During his interview with Pierson, Bridle cited two things: a study accepted for publication in the peer-reviewed journal Clinical Infectious Diseases and a document about Pfizer's coronavirus vaccine.

But neither source backs up his claims.

Bridle said the May 20 study showed how spike proteins produced by coronavirus vaccines could linger in the bloodstream and cause cardiovascular damage. An author of that study

says otherwise.

"My reading of the article you sent is Bridle is over-interpreting our results," David Walt, a professor at Harvard Medical School and the study's co-author, said in an email to USA TODAY.

The study measured proteins in plasma samples from 13 participants who received two doses of Moderna's coronavirus vaccine. It found that spike protein "was detectable in three of 13 participants an average of 15 days after the first injection."

But those results don't indicate the coronavirus vaccines are dangerous. It suggests the vaccines are working as designed.

"Our study simply validated that the mRNA vaccine is translated into the protein it is designed to encode," Walt said. "Because our method is 100-1000 fold more sensitive than others, we detected VERY low concentrations of the protein in most vaccinated individuals."

The paper's authors hypothesized that could be due to the body's immune response. During that process, T cells kill other cells that present the spike protein, causing an "additional release of spike into the bloodstream."

That phenomenon isn't a cause for concern, Walt said.

"While it is true that the spike protein has 'superantigen' properties, which means it has the POTENTIAL to cause adverse effects, we know that it doesn't cause these effects in many infected patients, it doesn't cause many of these superantigen effects in most vaccinated individuals, and the levels are incredibly low in the blood, suggesting this shouldn't be a concern," he said.

Fact check: No, the Oxford-AstraZeneca vaccine will not make your body Bluetooth connectable

The second source Bridle cited during his interview is a "biodistribution study" obtained from the Japanese Pharmaceuticals and Medical Devices Agency. He said the study shows how the coronavirus spike protein circulates in the bloodstream of vaccinated individuals and accumulates in their organs.

Pfizer told USA TODAY the document, which is in Japanese, doesn't back up Bridle's claims.

"The document is a real (common technical document), though it's not leaked – it's part of the submission data applied by Pfizer to PMDA (Japan's version of FDA) for its review," Kit

Longley, senior manager of science media relations, said in an email. "The document is about the pharmacokinetics overview seen from lab studies and we can confirm it's not about spike proteins from the vaccine resulting in dangerous toxins that linger in the body."

Colleagues say Bridle is off base

When USA TODAY reached out to Bridle via email for comment, an automatic reply addressing his comments on the coronavirus vaccines was returned.

"My answer to the question posed by the host was objective and founded on multiple reliable scientific sources," Bridle's automatic reply says. "I was simply fulfilling my duty as an academic public servant to disseminate information when it is asked of me."

Turner also defended Bridle's claims in an email to USA TODAY.

"The video contained in the article on my website says all that needed to be said," Turner said. "In it, you heard the doctor in his own words."

But Bridle's own colleagues at the University of Guelph's Ontario Veterinary College say the immunologist's claims are wrong.

"The bottom line is the vaccine contains an altered protein that is designed to prevent full activation, and it circulates for a short period of time at levels that are far below what would be a concern," W. Glen Pyle, a professor in the Department of Biomedical Sciences, said in an email.

J. Scott Weese, an associate professor in the Department of Pathobiology, said in an email that all evidence suggests the coronavirus vaccines are safe. Misinformation about the safety of the vaccines appears to be aimed at "creating fear and confusion during a critical time in this pandemic," he said.

"The efficacy and safety of mRNA vaccines is astounding, to me, particularly for a virus we've only known for a year and a half," Weese said. "mRNA vaccines have been used on millions of people, including extremely high rates of vaccination in high-risk populations (elderly, patients with other diseases), with incredibly low adverse event rates."

Fact check: No, COVID-19 vaccine isn't transmitted to others via contact

Public health officials have closely monitored the vaccine rollout for potential adverse effects. Amy Greer, an associate professor in the Department of Population Medicine at the

University of Guelph's Ontario Veterinary College, said in an email that the system appears to be working.

"Given the large number of mRNA vaccines administered to date, the absence of (safety concerns) with the mRNA vaccines is really a significant scientific achievement," she said.

Our rating: False

The claim that spike proteins from coronavirus vaccines are dangerous toxins that cause damage in the body is FALSE, based on our research. A recent study found spike protein in the blood of individuals vaccinated against COVID-19, but the levels were too low to cause damage, according to one of the study's authors. Pfizer and Bridle's colleagues also say his claims about spike proteins are wrong. Public health officials say the three coronavirus vaccines approved for emergency use in the U.S. are safe and effective at preventing serious infection.

Fact-check sources:

LifeSite, May 31, Vaccine researcher admits ‘big mistake,’ says spike protein is dangerous ‘toxin’
Hal Turner Radio Show, May 31, Doctor on COVID Vax: "We Screwed-Up. We didn't realize the Spike Protein is a TOXIN" Does this mean everyone vaccinated is manufacturing their own Spike Protein Toxins in their own bodies?
Clinical Infectious Diseases, May 20, Circulating SARS-CoV-2 Vaccine Antigen Detected in the Plasma of mRNA-1273 Vaccine Recipients
David Walt, June 2, Email exchange with USA TODAY
LifeSite News, accessed June 2, Facebook
USA TODAY, May 7, Fact check: COVID-19 vaccinated people don’t ‘shed’ viral particles from the vaccine
University of Guelph, accessed June 2, Byram W. Bridle
CrowdTangle, accessed June 2 and 8
USA TODAY, Dec. 19, Vaccines: Why do we need them and how do they work?
Knoxville News Sentinel, March 1, Does the new Johnson & Johnson COVID-19 vaccine stack up? Here's how it works
ON Point with Alex Pierson, May 27, New peer reviewed study on COVID-19 vaccines suggests why heart inflammation, blood clots and other dangerous side effects occur
David Fisman, June 2, Email exchange with USA TODAY

USA TODAY, March 5, How mRNA vaccines work
Centers for Disease Control and Prevention, accessed June 2, Understanding mRNA COVID-19 Vaccines
Food and Drug Administration, accessed June 2, COVID-19 Vaccines
Centers for Disease Control and Prevention, accessed June 2, Pfizer-BioNTech COVID-19 Vaccine Overview and Safety
Centers for Disease Control and Prevention, accessed June 2, Moderna COVID-19 Vaccine Overview and Safety
Centers for Disease Control and Prevention, accessed June 2, Johnson & Johnson's Janssen COVID-19 Vaccine Overview and Safety
USA TODAY, May 7, Fact check: COVID-19 vaccinated people don't 'shed' viral particles from the vaccine
USA TODAY, April 27, Fact check: No, interacting with a vaccinated person won't cause miscarriage or menstrual changes
NYU Langone Health, Jan. 28, How mRNA Vaccines Prevent COVID-19
W. Glen Pyle, June 2, Email exchange with USA TODAY
Reuters, March 11, Fact Check-COVID-19 vaccines using mRNA do not send the immune system into 'perpetual overdrive' by instructing cells to create the spike protein over and over again
Centers for Disease Control and Prevention, accessed June 2, COVID-19 Vaccinations in the United States
Food and Drug Administration, May 10, Pfizer-BioNTech COVID-19 Vaccine EUA Letter of Authorization reissued 05-10-2021
Food and Drug Administration, Feb. 25, Moderna COVID-19 Vaccine EUA Letter of Authorization
Food and Drug Administration, Feb. 27, Janssen COVID-19 Vaccine EUA Letter of Authorization
Chemical & Engineering News, Sept. 29, The tiny tweak behind COVID-19 vaccines
Centers for Disease Control and Prevention, accessed June 2, Possible Side Effects After Getting a COVID-19 Vaccine
J. Scott Weese, June 2, Email exchange with USA TODAY
Amy Greer, June 2, Email exchange with USA TODAY
Byram Bridle, June 2, Automatic email reply
Celeste McGovern, June 2, Email exchange with USA TODAY
Centers for Disease Control and Prevention, accessed June 3, CDC Recommends Use of Johnson & Johnson's Janssen COVID-19 Vaccine Resume

USA TODAY, May 12, CDC reports 13 additional cases of blood clots linked to J&J COVID-19 vaccine. All happened before 11-day pause in its use.
Children's Health Defense, accessed June 3
Kit Longley, June 3, Email exchange with USA TODAY
Food and Drug Administration, accessed June 3, COVID-19 Vaccine Safety Surveillance
Science Translational Medicine: In the Pipeline, May 4, Spike Protein Behavior
Abby Capobianco, June 4, Email exchange with USA TODAY
Natural News, June 2, Vaccine researcher admits 'big mistake,' says spike protein is dangerous 'toxin'
USA TODAY, May 25, Fact check: Moderna vaccine does not include poisonous substances
Hal Turner, June 2, Email exchange with USA TODAY

This is Exhibit "P" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

[redacted]

From: **Byram Bridle** <bbridle@uoguelph.ca>
Date: Mon, May 31, 2021 at 2:42 AM
Subject: Why Parents, Teens, and Children Should Question the COVID-19 Vaccine
To:

Dear recipient,

You are one of hundreds of people that have been blind carbon copied on this e-mail. Last Thursday evening I was interviewed on radio about COVID-19 vaccines for children and adolescents (https://omny.fm/shows/on-point-with-alex-pierson/new-peer-reviewed-study-on-covid-19-vaccines-sugge). This interview went viral around the world. Although I received hundreds of supportive e-mails and phone calls from around the globe, a vicious smear campaign has been initiated against me. This included the creation of a libelous website using my domain name. Such are the times that an academic public servant can no longer answer people's legitimate questions with honesty and based on science without fear of being harassed and intimidated.

However, it is not in my nature to allow scientific facts to be hidden from the public. I have attached a brief report that outlines the key science in support of what I said. This was written with my colleagues in the Canadian COVID Care Alliance (CCCA). We are a group of independent Canadian doctors, scientists, and professionals aiming to provide top quality, evidence-based information about COVID-19, intent on reducing hospitalizations and saving more lives. Our goal is to provide you with unbiased, peer-reviewed science that is relevant for you so that you can stay on the leading edge of the ever-evolving data, while at the same time focussing all of your efforts on your wellbeing, or, if you are a medical practitioner, the wellbeing of your patients.

Please feel free to send the attached brief report to as many people as possible. It is a very important message to get out to all Canadians.

The Canadian COVID Care Alliance is drafting a more extensive document that will dive into broader and deeper details about issues related to COVID-19 vaccines and youth. If you are interested in receiving this full article when it is ready, please sign-up to our e-mail distribution list at https://mailchi.mp/5666d252288c/canadian-covid-care-alliance. Please do not e-mail me directly for this request since my inbox is currently

unmanageable.

Because I care about our children,
Byram

Byram W. Bridle, PhD
Associate Professor of Viral Immunology
Office Room #4834
Lab Room #3808
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https://ovc.uoguelph.ca/pathobiology/people/faculty/Byram-W-Bridle

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This is Exhibit "Q" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Impact of population mixing between vaccinated and unvaccinated subpopulations on infectious disease dynamics: implications for SARS-CoV-2 transmission

David N. Fisman MD MPH, Afia Amoako MScPH, Ashleigh R. Tuite PhD MPH

Cite as: *CMAJ* 2022 April 25;194:E573-80. doi: 10.1503/cmaj.212105

Abstract

Background: The speed of vaccine development has been a singular achievement during the COVID-19 pandemic, although uptake has not been universal. Vaccine opponents often frame their opposition in terms of the rights of the unvaccinated. We sought to explore the impact of mixing of vaccinated and unvaccinated populations on risk of SARS-CoV-2 infection among vaccinated people.

Methods: We constructed a simple susceptible–infectious–recovered compartmental model of a respiratory infectious disease with 2 connected subpopulations: people who were vaccinated and those who were unvaccinated. We simulated a spectrum of patterns of mixing between vaccinated and unvaccinated groups that ranged from random mixing to complete like-with-like mixing (complete assortativity), in which people have contact exclusively with others with the same vaccination status. We evaluated the dynamics of an epidemic within each subgroup and in the population as a whole.

Results: We found that the risk of infection was markedly higher among unvaccinated people than among vaccinated people under all mixing assumptions. The contact-adjusted contribution of unvaccinated people to infection risk was disproportionate, with unvaccinated people contributing to infections among those who were vaccinated at a rate higher than would have been expected based on contact numbers alone. We found that as like-with-like mixing increased, attack rates among vaccinated people decreased from 15% to 10% (and increased from 62% to 79% among unvaccinated people), but the contact-adjusted contribution to risk among vaccinated people derived from contact with unvaccinated people increased.

Interpretation: Although risk associated with avoiding vaccination during a virulent pandemic accrues chiefly to people who are unvaccinated, their choices affect risk of viral infection among those who are vaccinated in a manner that is disproportionate to the portion of unvaccinated people in the population.

The remarkable speed of vaccine development, production and administration during the COVID-19 pandemic is a singular human achievement.[1] While the ability to vaccinate to herd immunity has been held back by the increasing transmissibility of novel SARS-CoV-2 variants of concern (e.g., Delta and Omicron variants),[2,3] and global distribution of vaccines is inequitable,[4] the effectiveness of SARS-CoV-2 vaccines in reducing severity of disease and disrupting onward transmission even when breakthrough infections occur is likely to have saved many lives. The emergence of the immune-evasive Omicron variant may undermine some of these gains, although provision of booster vaccine doses may restore vaccination to a high level of potency, and vaccines developed specifically to enhance immunity to the Omicron variant may emerge in 2022.[3,5–7]

However, antivaccine sentiment, fuelled in part by organized disinformation efforts, has resulted in suboptimal uptake of readily available vaccines in many countries, with adverse health and economic consequences.[8–10] Although the decision not to receive vaccination is often framed in terms of the rights of individuals to opt out,[11,12] such arguments neglect the potential harms to the wider community that derive from poor vaccine uptake. Nonvaccination is expected to result in amplification of disease transmission in unvaccinated subpopulations, but the communicable nature of infectious diseases means that this also heightens risk for vaccinated populations, when vaccines confer imperfect immunity. Although assortative (like-with-like) mixing[13] is characteristic of many communicable disease systems and may be expected to limit interaction between vaccinated

and unvaccinated subpopulations to some degree, the normal functioning of society means that complete like-with-like mixing is not observed in reality. Furthermore, the airborne spread of SARS-CoV-2[14–20] means that close-range physical mixing of people from vaccinated and unvaccinated groups is not necessary for between-group disease transmission.

Historically, behaviours that create health risks for the community as well as individuals have been the subject of public health regulation. This is true of communicable infectious diseases but also applies to public health statutes that limit indoor cigarette smoking[21] and legal restrictions on driving under the influence of alcohol and other intoxicants.[22,23]

Simple mathematical models can often provide important insights into the behaviour of complex communicable diseases systems.[13,24,25] To better understand the implications of the interplay between vaccinated and unvaccinated populations under different assumptions about population mixing, we constructed a simple susceptible–infectious–recovered model to reproduce the dynamics of interactions between vaccinated and unvaccinated subpopulations in a predominantly vaccinated population. We sought to contrast contribution to epidemic size and risk estimates by subpopulation, and to understand the impact of mixing between vaccinated and unvaccinated groups on expected disease dynamics.

Methods

Model

We constructed a simple compartmental model of a respiratory viral disease.[26] The model is described in Appendix 1 (available at www.cmaj.ca/lookup/doi/10.1503/cmaj.212105/tab-related-content). People are represented as residing in 3 possible "compartments:" susceptible to infection (S), infected and infectious (I), and recovered from infection with immunity (R). We divided the compartments to reflect 2 connected subpopulations: vaccinated and unvaccinated people. Susceptible people move into the infectious compartment after effective contacts (i.e., contacts of a nature and duration sufficient to permit transmission) with people who are infected. In the context of an airborne virus like SARS-CoV-2,[14–20] effective contact may be conceptualized as "sharing air" with an infective case. After an infectious period, infectious people with SARS-CoV-2 recover with immunity. We also assumed that some fraction of the unvaccinated population had immunity at baseline owing to previous infection and that a fraction of the population was vaccinated. We treated immunity after vaccination as an all-or-none phenomenon, with a fraction of vaccinated people (as defined by vaccine effectiveness) entering the model in the immune state and the remainder being left in the susceptible state. For example, a vaccine that is 80% efficacious would result in 80% of vaccinated people becoming immune, with the remaining 20% being susceptible to infection. We did not model waning immunity.

Humans do not mix randomly and exhibit a tendency to interact preferentially with others like themselves,[13,27] a phenomenon referred to as "assortativity." The relative frequency of interactions between people within different groups occurs on a spectrum that lies between high assortativity (i.e., like-with-like mixing) and random mixing. For instance, age-assortative mixing is frequently observed; children are more likely to interact with other children than would be expected if contacts occurred at random across all age groups. The use of matrices to govern such interactions are described in Appendix 1.

However, with respect to contacts between people from 2 different groups, relative frequency of contacts will depend both on the relative size of the 2 groups and the degree of like-with-like mixing. In our model, like-with-like mixing is determined by a constant (η), with random mixing occurring when $\eta = 0$, complete like-with-like mixing occurring when $\eta = 1$ and intermediate degrees of like-with-like mixing occurring at intermediate values. For our model, with 20% of the population unvaccinated, when random mixing is assumed ($\eta = 0$), 20% of the contacts a vaccinated person has would be expected to occur with unvaccinated people. When exclusively like-with-like mixing is assumed ($\eta = 1$), 0% of contacts a vaccinated person has would be with unvaccinated people. For intermediate levels of like-with-like mixing ($\eta = 0.5$), 10% of a vaccinated person's contacts would be with unvaccinated people.

We otherwise parameterized our base case model to represent a disease similar to SARS-CoV-2 infection with Delta variant, with a reproduction number of an infectious disease in the absence of immunity or control (R_0) of 6,[28] and we used higher values to capture the dynamics of the Omicron variant.[29] Our lower-bound estimate for vaccine effectiveness (40%) reflected uncertainty about the emerging Omicron variant,[3,7] whereas our upper bound (80%) reflected the higher effectiveness seen with the Delta variant.[30] Base case parameters, plausible ranges and relevant references are presented in Table 1.

We used the model to explore the impact of varying rates of immunization and different levels of like-with-like mixing on the dynamics of disease in vaccinated and unvaccinated subpopulations. We evaluated the absolute contribution to overall case counts by these subpopulations, and within-group and overall infection risk. We calculated attack rates as the cumulative number of infections divided by the population size. We calculated a quantity (ψ), which we defined as the fraction of all infections among vaccinated people that derived from contact with unvaccinated people, divided by the fraction of all contacts that occurred with unvaccinated people. Effectively, this represents a normalized index of the degree to which risk in one group may be disproportionately driven by contact with another. For example, if 10% of contacts among vaccinated people are with unvaccinated people, but 50% of infections among vaccinated people derive from these contacts, ψ would have a value of 5. If infection were simply a function of frequency of contact between the groups and prevalence was the same across groups, ψ would have a value of 1. The value of ψ would increase above 1 either because of an increased fraction of infections derived from contact with unvaccinated people or a decrease in the amount of contact between the groups (i.e., an increase in like-with-like mixing).

A version of the model in Microsoft Excel is available at 10.6084/m9.figshare.15189576.

Table 1: Model parameters

Parameter description	Symbol	Value	Plausible range	Reference
Probability of transmission per contact multiplied by contacts per year	β	437	164–728	Calculated
Rate of recovery from infection (per yr)	γ	73	41–91	Wolfel et al.[31]
Basic reproduction number	R_0	6	4–8	UK Health Security Agency,[3] Hogan et al.,[7] Xia et al.[28]
Mixing between subpopulations (0 = random, 1 = assortative)	η	0.5	0–0.9	Assumption (approach based on Garnett and Anderson[13])
Proportion vaccinated	P_v	0.8	0.6–0.99	Little[32]
Vaccine effectiveness	VE	0.8	0.4–0.8	UK Health Security Agency,[3] Hogan et al.,[7] Higdon et al.[33]
Approximate adult population of Ontario	N	10 000 000	—	Statistics Canada[34]
Baseline immunity in unvaccinated people		0.2	—	Assumption

Ethics approval

Because this study involved the use of publicly available aggregate data, approval by a research ethics board was not required.

Results

We present simulated epidemics that assume different amounts of mixing between vaccinated and unvaccinated groups in Figure 1. With 20% baseline immunity among unvaccinated people and 80% of the population vaccinated, we found that the absolute number of cases from vaccinated and unvaccinated groups was similar when mixing was random; however, after we adjusted for the substantially larger population in the vaccinated group, the risk of infection was markedly higher among unvaccinated people during the epidemic. With increased like-with-like mixing, differences in incidence between the vaccinated and unvaccinated groups became more apparent, with cases in the unvaccinated subpopulation accounting for a substantial proportion of infections during the epidemic wave. Like-with-like mixing uncoupled the dynamics of vaccinated and unvaccinated subpopulations, with unvaccinated subpopulations having higher and earlier peak incidence than vaccinated subpopulations. For example, with random mixing, peak incidence was simultaneous in the vaccinated and unvaccinated groups, but with strong like-with-like mixing the epidemic peak among vaccinated people occurred about 1 week later than among unvaccinated people; population-adjusted peak incidence was 4 times higher in the unvaccinated population than in the vaccinated population with random mixing, but about 30 times higher with strong like-with-like mixing (Figure 1).

We found that cumulative attack rates among vaccinated people were highest (15%) with random mixing and lowest (10%) with highly assortative mixing. In contrast, cumulative attack rates were lowest (62%) among unvaccinated people with random mixing, and highest (79%) with highly assortative mixing. The highest cumulative attack rates in the population overall were seen with intermediate levels of like-with-like mixing (27%) compared with random mixing (25%) and strong like-with-like mixing (24%) (Figure 1).

When we varied the degree of like-with-like mixing, changes in epidemic size in the vaccinated subpopulation occurred. As like-with-like mixing increased (i.e., with reduced contact between vaccinated and unvaccinated subpopulations), the final attack rate decreased among vaccinated people, but the contribution of risk to vaccinated people caused by infection acquired from contact with unvaccinated people (as measured by ψ) increased. The larger the value of ψ, the more unvaccinated people contributed to infections in the vaccinated subpopulation.

This pattern was consistent across a range of values for vaccine effectiveness and reproduction numbers (Figure 2). We found that increased like-with-like mixing reduced final outbreak size among vaccinated people most markedly at lower reproduction numbers but increased the value of ψ. With lower vaccine effectiveness, as observed with the Omicron variant, the effects of like-with-like mixing were attenuated. With either lower reproduction numbers or higher vaccine efficacy, transmission was more readily disrupted within the vaccinated subpopulation, such that risk arose increasingly from interactions with the unvaccinated subpopulation, where transmission continued. As like-with-like mixing increased, contribution to infection risk among vaccinated people was increasingly derived from (less and less common) interactions with unvaccinated people, increasing the value of ψ. We found similar patterns in sensitivity analyses in which vaccine coverage was increased from 80% to 99% (Figure 3). Increasing population vaccination coverage decreased the attack rate among vaccinated people (as expected, owing to indirect protective effects) but further increased the relative contribution to risk in vaccinated people by those who were unvaccinated at any level of like-with-like mixing.

Interpretation

We use a simple deterministic model to explore the impact of assortative mixing on disease dynamics and contribution to risk in a partially vaccinated population during a pandemic modelled

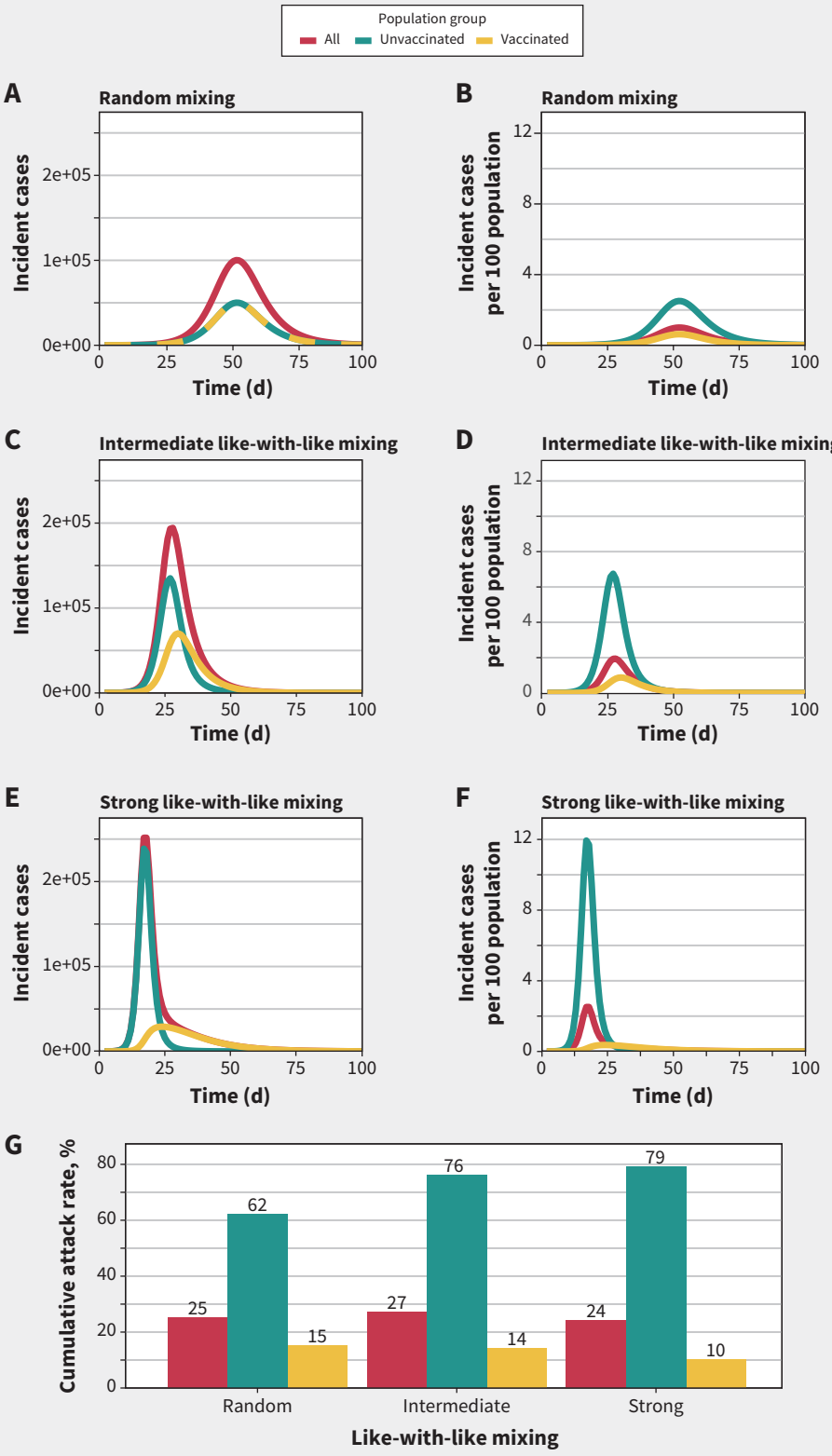

Figure 1: Simulated epidemics for different levels of mixing between vaccinated and unvaccinated populations. (A, C, E) Incident cases and (B, D, F) population-adjusted incidence per 100 population in unvaccinated, vaccinated and overall modelled populations. The degree of like-with-like mixing (assortativity, η) varies from (A, B) random mixing (η = 0) to (C, D) intermediate like-with-like mixing (η = 0.5) to (E, F) near exclusive mixing with people of the same vaccination status (η = 0.9). As like-with-like mixing increases, epidemic size among the vaccinated subpopulation is smaller in absolute terms than among the unvaccinated subpopulation and also has a different contour. (G) Increasing like-with-like mixing increased cumulative attack rates among unvaccinated people and decreased cumulative attack rates among vaccinated people. The highest overall attack rates were seen with intermediate levels of like-with-like mixing.

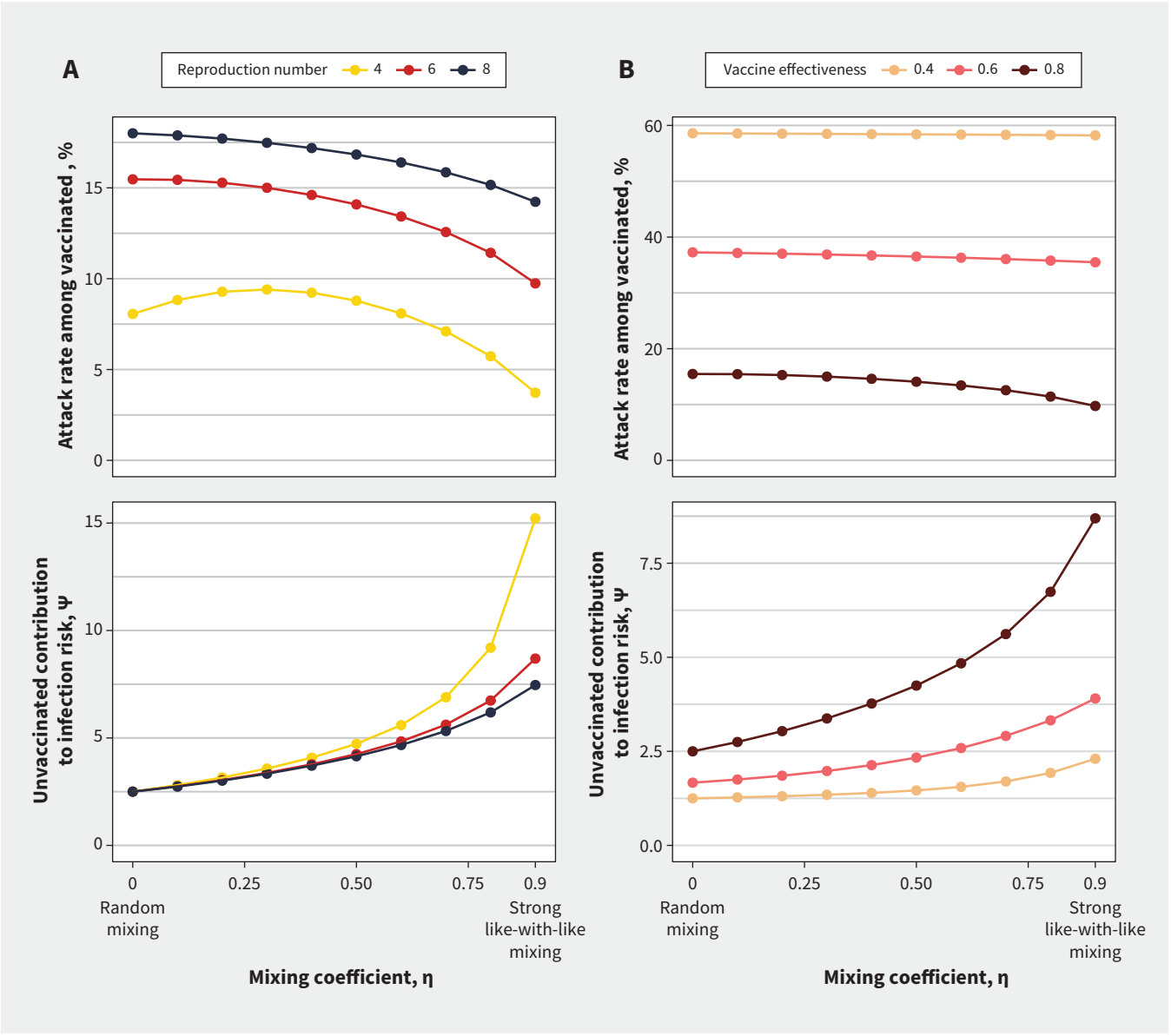

Figure 2: Impact of mixing between vaccinated and unvaccinated subpopulations on contribution to risk and final epidemic size for (A) varying reproduction numbers and (B) vaccine effectiveness. Both panels show the impact of increasing like-with-like mixing on outbreak size among the vaccinated subpopulation and contact-adjusted contribution to risk of infection in vaccinated people by unvaccinated people (ψ). As like-with-like mixing (η) increases, the attack rate among vaccinated people decreases, but ψ increases. This relation is seen across a range of (A) initial reproduction numbers and (B) vaccine effectiveness. These effects are more pronounced at lower reproduction numbers and are attenuated as vaccines become less effective. We used a base case estimate of 6 for the reproduction number in the sensitivity analysis on vaccine effectiveness and a base case estimate for vaccine effectiveness of 0.8 in the sensitivity analysis for *R*.

on the current pandemic of SARS-CoV-2. Notwithstanding the model's simplicity, it provides a graphical representation of the expectation that even with highly effective vaccines, and in the face of high vaccination coverage, a substantial proportion of new cases can be expected to occur in vaccinated people, such that rates, rather than absolute numbers, represent the appropriate metric for presenting the impact of vaccination. However, we find that the degree to which people differentially interact with others who are like themselves is likely to have an important impact on disease dynamics and on risk in people who choose to get vaccinated.

Vaccinated people were, as expected, at markedly lower risk of SARS-CoV-2 infection during the epidemic; however, when random mixing with unvaccinated people occurred, they decreased attack rates in the unvaccinated people, by serving as a buffer to transmission. As populations became more separate with progressively increasing like-with-like mixing, final epidemic sizes declined in vaccinated people, but rose in unvaccinated people because of the loss of buffering via interaction with vaccinated people. Many opponents of vaccine mandates have framed vaccine adoption as a matter of individual choice. However, we found that the choices made by people who forgo

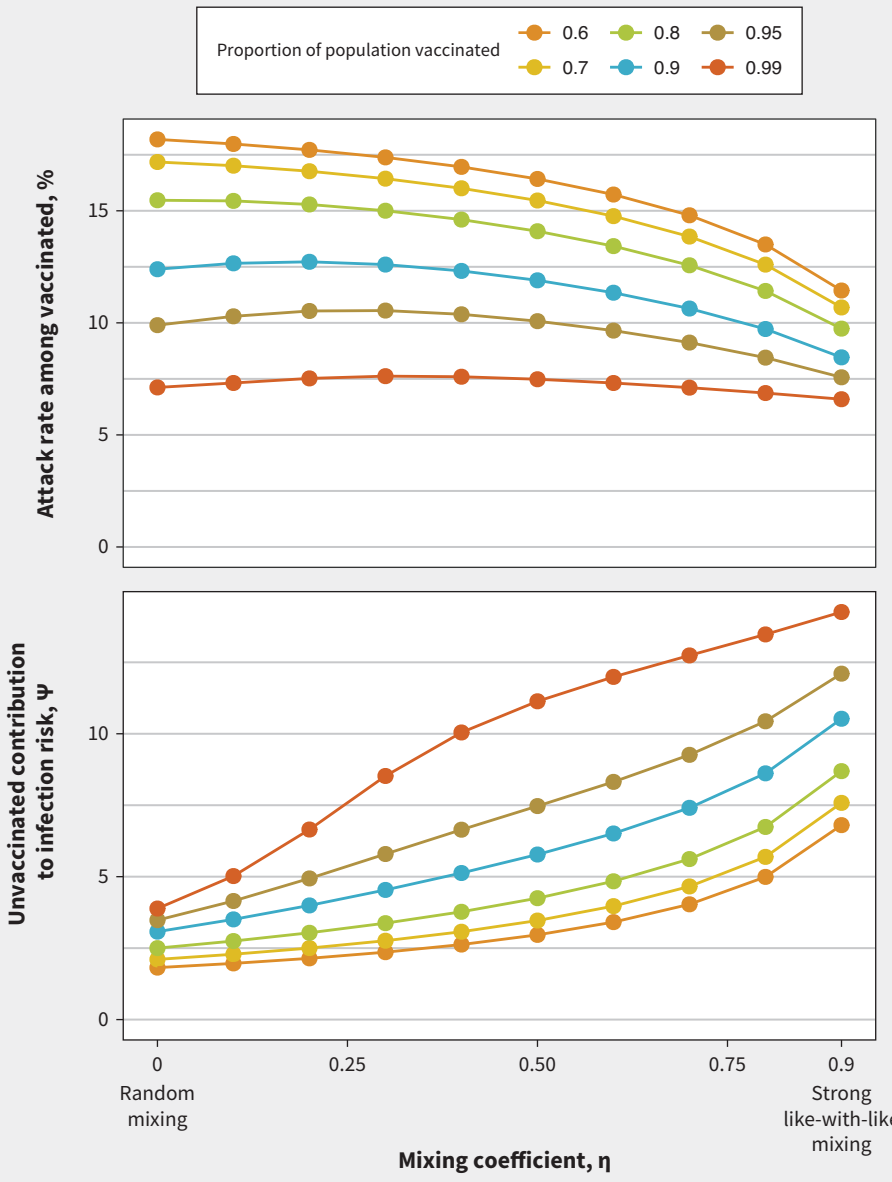

Figure 3: Impact of mixing between vaccinated and unvaccinated subpopulations on contribution to risk and final epidemic size with increasing population vaccination coverage. Increasing population vaccination coverage decreases the attack rate among vaccinated individuals and further increases the relative contribution to risk in vaccinated individuals by the unvaccinated at any level of like-with-like mixing. For levels of vaccination coverage that were evaluated, increasing like-with-like mixing decreases the attack rate among the vaccinated but increases the relative contribution to risk in vaccinated individuals by the unvaccinated.

vaccination contribute disproportionately to risk among those who do get vaccinated.

Increased mixing between vaccinated and unvaccinated groups increased final epidemic size among vaccinated people; conversely, more like-with-like mixing decreased final epidemic size among vaccinated people but resulted in enhancement of the degree to which risk among vaccinated people could be attributed to unvaccinated people. The fact that this excess contribution to risk cannot be mitigated by high like-with-like mixing undermines the assertion that vaccine choice is best left to the individual and supports strong public actions aimed at enhancing vaccine uptake and limiting access to public spaces for unvaccinated people, because risk cannot be considered "self-regarding."[35] There is ample precedent for public health regulation that protects the wider community from acquisition of communicable diseases, even if this protection comes at a cost of individual freedom.[36,37] We also note that the use of legal and regulatory tools for the prevention of behaviours and practices that create risk for the wider public also extend beyond communicable infectious diseases, such as statutes that limit indoor cigarette smoking.[21–23]

In the context of immune evasion seen with the newly emerged Omicron variant, we found that like-with-like mixing is less protective when vaccine effectiveness is low. This finding underlines the dynamic nature of the pandemic, and the degree to which policies need to evolve in a thoughtful manner as the nature of the disease and the protective effects of vaccines evolve. Boosting with mRNA vaccines appears to restore vaccine effectiveness at least temporarily against Omicron,[5] and it is likely that the higher vaccine effectiveness estimates used in our model will be relevant to public policy as booster campaigns are scaled up in Canada and elsewhere.

Despite reduced protection against infection by the Omicron variant, vaccinated people, including those who have not received third vaccine doses, have continued to receive strong protection against admission to hospital and death from SARS-CoV-2 infection.[38,39] This means that acceptance of vaccination is a means of ensuring that greater health care capacity is available for those with other illnesses. For example, in Ontario, capacity for COVID-19 cases in intensive care units was created by cancelling elective surgeries for cancer and cardiac disease, which resulted in extensive backlogs.[40] By contributing to these backlogs, unvaccinated people are creating a risk that those around them may not be able to obtain the care they need and, consequently, the risk they create cannot be considered self-regarding.

The robustness of our findings in the face of wide-ranging sensitivity analysis will allow this work to be applied in the future, when new variants arise, as we understand the length of time vaccination confers immunity and as new vaccine formulations become available.

Limitations

The simplicity of our model is both a strength (it is transparent and easily modified to explore the impact of uncertainty) and a weakness, because it does not precisely simulate a real-world pandemic process in all its complexity. For instance, we modelled vaccine effectiveness against infection but not the additional benefits of vaccination for preventing severe illness. Although this benefit is not captured by a simple model focused on transmission, an advantage of models such as ours is that they provide a ready platform for layering on increasing complexity, so our model can be adapted or expanded to consider impacts on the health system, or to incorporate additional structural elements or alternate assumptions. We have also likely underestimated vaccine benefit in this model, as we have not attempted to capture the impact of vaccines on prevention of forward transmission by vaccinated, infected individuals; this effect appears to be substantial.[41]

Conclusion

Using simple mathematical modelling, we have shown that, although risk associated with avoiding vaccination during a virulent pandemic accrues chiefly to those who are unvaccinated, the choice of some individuals to refuse vaccination is likely to affect the health and safety of vaccinated people in a manner disproportionate to the fraction of unvaccinated people in the population. Risk among unvaccinated people cannot be considered self-regarding, and considerations around equity and justice for people who do choose to be vaccinated, as well as those who choose not to be, need to be considered in the formulation of vaccination policy. It is unlikely that SARS-CoV-2 will be eliminated, and our findings will likely be relevant to future seasonal SARS-CoV-2 epidemics or in the face of emerging variants.

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Competing interests: David Fisman has served on advisory boards related to influenza and SARS-CoV-2 vaccines for Seqirus, Pfizer, AstraZeneca and Sanofi-Pasteur Vaccines, and has served as a legal expert on issues related to COVID-19 epidemiology for the Elementary Teachers Federation of Ontario and the Registered Nurses Association of Ontario. He also served as a volunteer scientist on the Ontario COVID-19 Science Advisory Table. Ashleigh Tuite was employed by the Public Health Agency of Canada when the research was conducted. The work does not represent the views of the Public Health Agency of Canada. No other competing interests were declared.

This article has been peer reviewed.

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Contributors: All of the authors made substantial contributions to the conception and design of this work, drafting and revision for important intellectual content, gave final approval of the version to be published and agreed to be accountable for all aspects of the work.

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Data sharing: A version of the model in Microsoft Excel is freely available at 10.6084/m9.figshare.15189576.

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Correspondence to: David Fisman, david.fisman@utoronto.ca

This is Exhibit "R" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

200

Fiction Disguised as Science to Promote Hatred

Disinformation Must Be Called Out

DR. BYRAM W. BRIDLE
APR 26, 2022

If you only have a few minutes, jump to the section below entitled "Proof that the Paper by Fisman, *et al.* Should be Retracted Immediately".

The legacy media has been like pigs at a trough today and yesterday with the publication of an atrocious 'peer-reviewed' 'scientific' article by Dr. David Fisman, Dr. Ashleigh Tuite, and a graduate student. After all, with raw public health data unable to support the only COVID-19 narrative that has been deemed acceptable, fresh fuel was apparently needed. Lots of media outlets have been reporting on this study; one of them being CTV News. Here is the headline for their article:

> *"Being with unvaccinated people increases COVID-19 risk for those who are vaccinated: modelling study"*

This kind of messaging will only fuel hatred and segregation and the potential development of harmful policies. And it is all in the name of bad science. As a researcher who has published and reviewed many scientific papers, I can tell you that the article by Fisman, *et al.* is the worst one that I have ever seen. The 'peer reviewers' of this article should be ashamed of themselves for allowing this to be published, and the editor even more so. If the *Canadian Medical Association Journal* does not promptly retract this article, they will have made themselves an embarrassment among scientific publishers.

This paper by Fisman, *et al.* is only thinly veiled hate speech under the guise of science. Before I walk you through the numerous massive errors in this paper, let me first show you one example of the messaging regarding the impact and relevance of the paper.

Fisman was quoted by CTV News as saying "We thought what was missing from that conversation was, what are the rights of vaccinated people to be protected from unvaccinated people?". The only reason why Fisman is trying to turn people against other people is due to the abject failure of the COVID-19 'vaccines' to function like vaccines. That is to say that the purpose of a vaccine is to protect people from a pathogen; to prevent both the disease and transmission of the causative agent. Such a medical product does not require one to pit people against people. Fisman has inappropriately labelled critically thinking people as an enemy when the actual culprits are SARS-CoV-2 and overly rushed jabs of exceptionally poor quality and highly questionable safety that have been pushed as the sole solution at the expense of all others.

One of the few accurate statements in the CBC News article was this quote about Fisman where: "he acknowledges that a simple mathematical model doesn't fully reflect the real world or the

diverse factors that must be taken into account when setting public health policy". This is an understatement if ever I saw one.

I already spend an inordinate amount of time and energy correcting misinformation and disinformation coming from scientists who should know better. As such, I am not going to conduct an exhaustive breakdown of the article by Fisman, *et al.* However, here are quite a few examples that make it clear that the science is flawed and the messaging not only biased, but completely incorrect...

1. 

ity has been held back by the
concern...". How about considering
real vaccines that they never
The authors of the paper are not
discussions have turned to the
seem to realize that the term
ase and transmission of the
would be an unrealistic goal. To do
/ is required. The current SARS-
d. This is painfully obvious by the
nic despite most of the population
ces and other organizations (and
SARS-CoV-2 run rampant through

2 *nized disinformation efforts, has*
resulted in suboptimal uptake of readily available vaccines".

In fact, the article by Fisman, *et al.* represents one of the most egregious examples of disinformation that I have seen.

Most of the people that remain 'unvaccinated' are not 'anti-vaccine' by any stretch of the imagination. Due to critical thinking and following the science, they are not in support of the current crop of COVID-19 jabs. Remember, the definition of a vaccine was changed to accommodate these jabs. They are nothing like any of the historically mandated vaccines, such as those used in the childhood immunization series.

People have been wary because the initial clinical trials to evaluate these 'vaccines' are still ongoing, literally meaning the jabs are still in the experimental phase. Other reasons for wariness include that the stated endpoint of the ongoing clinical trials is a reduction in cases of COVID-19, which they are failing to do. Hesitancy is because the placebo control arms of the clinical trials were removed (in contravention of study protocols) after a median follow-up time of only two months so no mid- to long-term safety signals can be detected via active monitoring. It is because the only safety monitoring left is via outdated and flawed passive voluntary monitoring systems that dramatically underestimate adverse events. It is because the vaccines behave in stark contrast to what was publicly stated. For example, they don't stay at the injection site. Rather, the mRNA vaccines get distributed throughout the body (see my recent article about this). It is because serious side-effects were only unveiled after the rollout into the public, leading to things like the AstraZeneca 'vaccine' being deemed too dangerous for Canadian adults (many other countries detected the blood clotting problem and avoided this jab and then had to teach Canadian health authorities about this problem) and the Moderna vaccine being declared too dangerous for young males by harming their hearts.

Other reasons for COVID-19 vaccine hesitancy include public health 'experts' disseminating disinformation like what is contained in the article by Fisman, *et al.* Also, many people recognize the validity of naturally acquired immunity and the massive accumulation of literature that shows it is superior in almost every way to the 'vaccine'-induced immune responses. And there are a myriad of other reasons.
I refer to this as a major issue because Fisman and his colleagues are labeling people and using subjective argumentation in an attempt to justify it. This sentence in the paper has no place in the world of objective science.

3. Major problem: *"Nonvaccination is expected to result in amplification of disease transmission in unvaccinated subpopulations, but the communicable nature of infectious diseases means that this also heightens risk for vaccinated populations"*. Some critical considerations have been missed here. The issue is not about nonvaccinated versus vaccinated people; it is about who is immune and who is not. Fisman, *et al.* really need some advanced immunology training before opining about vaccines. They need to realize that immune responses to vaccination follow a bell-shaped curve in an outbred population like humans. Most people respond moderately well. A few will respond very robustly; these are the high responders. And then a minority will be low- or non-responders. These individuals will have no protection against disease. As such, showing certification of having received two or more jabs does not guarantee immunity. And then there is the reality that the human immune system can work in the absence of external 'hand of man' interventions. This means that our immune systems can mount protective responses to natural infections. Having lived with SARS-CoV-2 for more than two years and with the Delta and Omicron variants having ripped through the population, a vast majority of 'unvaccinated' people would be expected to have naturally acquired immunity. Conflating the concepts of immunity and vaccination represents a major conceptual flaw with this paper. I recommend that Fisman and his colleagues review this video that I am in full agreement with: Fauci teaches that **natural** infection is the **best form of vaccination**.
4. Major issue that proved to be a fatal flaw: *"We also assumed that some fraction of the unvaccinated population had immunity at baseline owing to previous infection and that a fraction of the population was vaccinated"*. Initially, this seems reasonable. But then one learns that the number plugged into the simple mathematical model was only 20% of the population having naturally acquired immunity. Really?!? After more than two years of living with SARS-CoV-2 and and Delta and Omicron sweeping through Canada, one is to believe that only 20% have immunity? Where is the evidence for this? A paper showing that a vast majority of Canadians might have some degree of naturally acquired or cross-reactive immunity against SARS-CoV-2 can be found here. I would expect that even more unvaccinated Canadians would now have natural immunity. Unfortunately, the Canadian taskforce for gathering data about immunity among our population was disbanded so we missed the boat on tracking this incredibly important parameter that would have a major influence on any epidemiological model. So, what is the source of data used by Fisman, *et al.* to come up with their proposition of 20% immunity with a 'plausible' range of 10-50%? The source is stated as "assumption"! Isn't that interesting. This is notable because all mathematical models are only as good as the assumptions that are plugged into them. The old adage is 'garbage in, garbage out'. I will show you below how this single unjustified assumption biased the conclusions of the paper.

5. Fatal flaw: "*We treated immunity after vaccination as an all-or-none phenomenon, with a fraction of vaccinated people (as defined by vaccine effectiveness) entering the model in the immune state and the remainder being left in the susceptible state. For example, a vaccine that is 80% efficacious would result in 80% of vaccinated people becoming immune, with the remaining 20% being susceptible to infection.*" There are multiple major issues here. First, the COVID-19 'vaccines' fail to confer immunity, which is protection against disease and transmission. It is common knowledge that 'vaccinated' people get infected and sick at least as much as, if not more than the 'unvaccinated'. This is why a desperate emphasis has been placed on the debatable possibility that disease severity is merely dampened by 'vaccination'. A such, a biologically incorrect assumption was made here.
Also, the effectiveness of the vaccine that was plugged into the model started at 40% and went up from there. However, these high percentages represent relative risk reduction, not absolute risk reduction. Do you remember when we were told that Pfizer's 'vaccine' was 95% effective? That did not mean that 95% of the population was protected against getting COVID-19 (or 80% as used in the example in the paper). That 95% reduction was a relative risk reduction. What many people were not told is that most of the people in Pfizer's clinical trial never got COVID-19, which is the disease that can occur in some people who get infected with SARS-CoV-2. In fact, the absolute risk reduction at the population level in the study was a mere 0.84% as a result of 'vaccination'. As such, it is completely inappropriate for *Fisman, et al.* to plug values for relative risk reduction into their population-level models and treat them as though they represent absolute risk reduction. Conflating relative versus absolute risk reduction was misleading. As expert epidemiologists the authors must surely know the difference between absolute versus relative risk reduction. This is evidence that what they disseminated was disinformation.

6. Fatal flaw: "*We did not model waning immunity*". I was flabbergasted by this assumption. COVID-19 'vaccine'-induced immunity is ridiculously short-lived. In contrast, naturally acquired immunity is much longer-lived. This differential effect would have had a major influence on the outcome of the mathematical model. This assumption by the authors ignores obvious scientific facts.

7. Major issue: There was no discussion about assumptions with respect to the safety (or lack thereof) of the COVID-19 'vaccines' that might offset the perceived value of 'vaccination'. Regardless of their viewpoint, they should have discussed this and provided evidence for their stance.

8. Major issue: "*Boosting with mRNA vaccines appears to restore vaccine effectiveness at least temporarily against Omicron*". This is not an accurate assumption to make. The paper that was cited to justify this statement used data derived from a simplistic model in a petri dish. It provided evidence of the presence of SARS-CoV-2-neutralizing antibodies in the blood of people who received COVID-19 booster 'vaccines'. However, this lacked a functional context. First, these antibodies were measured in blood, but the virus infects the airways, so the anatomical location that was studied is irrelevant. Second, it is possible that there were non-neutralizing antibodies that could actually enhance disease but no testing was performed to detect these; non-neutralizing antibodies were ignored. There are other issues, but the point is that there was no way of knowing whether the subset of antibodies measured in the cited study would translate into any degree of protection against 'real world' infection. This shows a bias by Fisman, *et al.* in pushing booster doses with the weakest of data to support it.

9. A refreshing truth: "*The simplicity of our model is... a weakness, because it does not precisely simulate a real-world pandemic process in all its complexity*". This is an understatement and is one of the reasons why the paper should be retracted.
10. Major issue: The paper treats COVID-19 'vaccines' as though these are the only way to reduce the harm of COVID-19 among Canadians. There was no discussion of alternative strategies such as prophylaxis or treatment using re-purposed drugs, the promotion of vitamin D sufficiency (a vital molecule for optimal functioning of the immune system) among a population in a northern climate that is plagued with seasonal vitamin D insufficiency, *etc.*
11. Major issue: Fisman declared the following competing interests: "*David Fisman has **served on advisory boards related to** influenza and **SARS-CoV-2 vaccines for Seqirus, Pfizer, AstraZeneca and Sanofi-Pasteur Vaccines***"! These seem like inappropriate conflicts of interest, especially in light of the major flaws in his paper. In combination, these are suggestive of an inappropriate bias in judgement.
12. Minor issue: "*He also served as a volunteer scientist on the Ontario COVID-19 Science Advisory Table*". My issue here is not related to a conflict of interest, but rather the shoddy epidemiological modeling in his current paper raises serious questions about all of the historical modeling done with the Ontario Science Table. Journalist Brian Lilley published an article in the Toronto Sun on March 16, 2022 about the poor track record of mathematical modeling by the Ontario Science Table (of which Fisman is a former key member of the modeling group). It included these quotes...

> *"The self-appointed, self-important Science Table has been wrong with their modelling predictions more times, and in more ways, than I can count."*
> *"The Science Table has somehow achieved a revered status in Ontario, a sort of secular sainthood bestowed upon their members and their work despite a spotty track record of predicting how the pandemic is going. They're not an official government body; in fact, they have no official government role, but they are seen as and presented as the authoritative voice on COVID in the province."*
> *"I'm just judging the Science Table by their track record and if you did that, you wouldn't be listening to them either."*

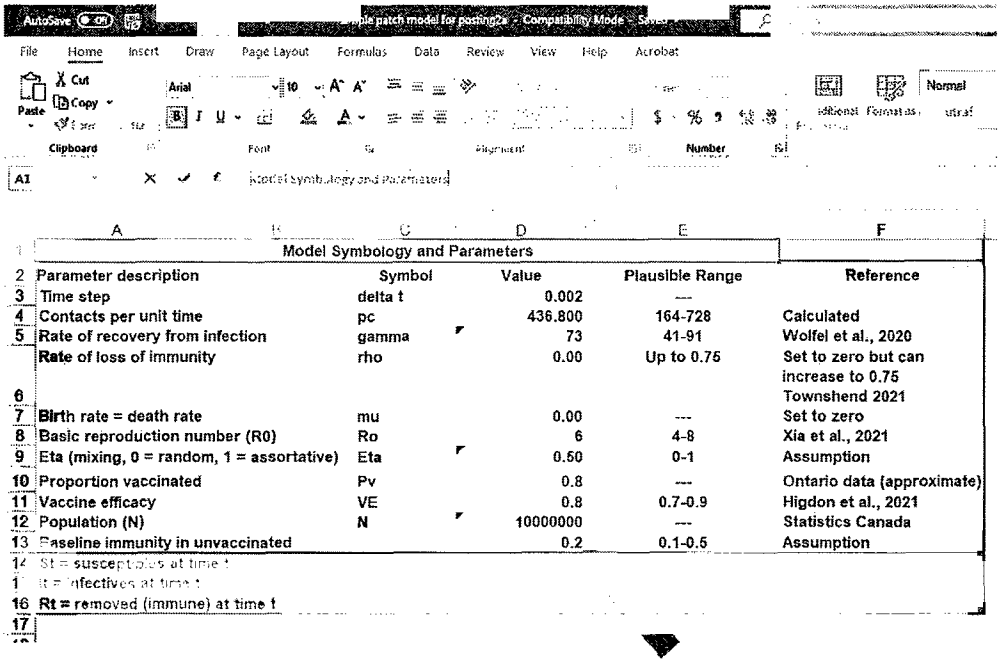
13. Major issue: The messaging in the paper by Fisman, *et al.* carries serious implications. It implies that the unvaccinated are selfishly causing harm to the 'vaccinated'. If the conclusions of the paper were to stand, they would logically lead to things like policies for forced vaccinations or stricter segregation of the 'unvaccinated' or harsher penalties against them. It is scary to see scientists using disinformation to promote hatred and division amongst Canadians.

Proof that the Paper by Fisman, *et al.* Should be Retracted Immediately

For the final 'nail in the coffin that is the paper by Fisman, *et al.*, let's make only one adjustment to their model. First, kudos to the authors for making their mathematical model available. Seeing the epidemiological models being used to inform COVID-19 policies has been a rarity over the past couple of years. Fisman did not make his model available in his previous paper (that was also published in the *Canadian Medical Association Journal*), nor for any models that he was involved with when serving on the Science Table. Unfortunately, the current disclosed model highlights exactly why nobody should ever trust any epidemiological model that has not been

fully disclosed. I encourage you to download the model. You can find it as a Microsoft Excel file here.

Now select the first tab, entitled "Patch Model". In the top left corner of this page you will see "Model Symbology and Parameters"...

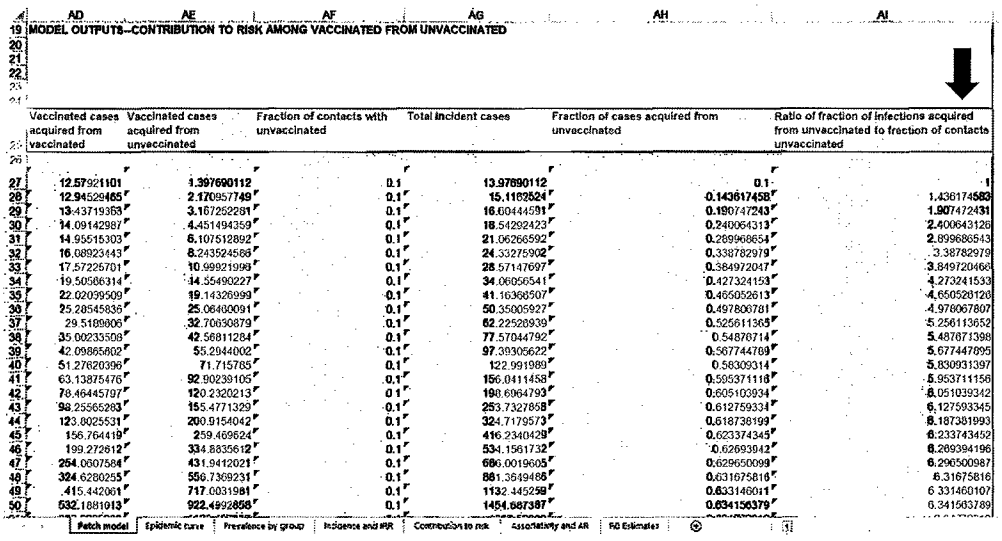


Model Symbology and Parameters

Parameter description	Symbol	Value	Plausible Range	Reference
Time step	delta t	0.002	---	
Contacts per unit time	pc	436.800	164-728	Calculated
Rate of recovery from infection	gamma	73	41-91	Wolfel et al., 2020
Rate of loss of immunity	rho	0.00	Up to 0.75	Set to zero but can increase to 0.75 Townshend 2021
Birth rate = death rate	mu	0.00	---	Set to zero
Basic reproduction number (R0)	Ro	6	4-8	Xia et al., 2021
Eta (mixing, 0 = random, 1 = assortative)	Eta	0.50	0-1	Assumption
Proportion vaccinated	Pv	0.8	---	Ontario data (approximate)
Vaccine efficacy	VE	0.8	0.7-0.9	Higdon et al., 2021
Population (N)	N	10000000	---	Statistics Canada
Baseline immunity in unvaccinated		0.2	0.1-0.5	Assumption
St = susceptibles at time t				
It = infectives at time t				
Rt = removed (immune) at time t				

Note that the default value used for baseline immunity among the 'unvaccinated' was a mere 20%, which was based on pure speculation (se the 'Reference' column, cell F13). Remember, I cited a peer-reviewed published scientific paper suggesting that ~90% have immunity.

If you scroll to the right on the same Excel page, you will find the following data starting at cell AD19...



MODEL OUTPUTS--CONTRIBUTION TO RISK AMONG VACCINATED FROM UNVACCINATED

Vaccinated cases acquired from vaccinated	Vaccinated cases acquired from unvaccinated	Fraction of contacts with unvaccinated	Total incident cases	Fraction of cases acquired from unvaccinated	Ratio of fraction of infections acquired from unvaccinated to fraction of contacts unvaccinated
12.57921101	1.397690112	0.1	13.97690112	0.1	1
12.94529465	2.170957749	0.1	15.1162524	0.14361745	1.436174583
13.43719353	3.167252281	0.1	16.60444581	0.190747243	1.907472431
14.09142987	4.451494359	0.1	18.54292423	0.240064313	2.400643126
14.95515303	6.107512892	0.1	21.06266592	0.289968654	2.899686543
16.08923443	8.243524586	0.1	24.33275902	0.338782979	3.38782979
17.57225701	10.99921998	0.1	28.57147697	0.384972047	3.849720466
19.50586314	14.55490227	0.1	34.06056541	0.427324153	4.273241533
22.02039509	19.14326999	0.1	41.16366507	0.465052613	4.650526128
25.28545836	25.06400091	0.1	50.35005927	0.497806781	4.978067807
29.5189806	32.70650879	0.1	62.22528939	0.525611365	5.256113652
35.00233508	42.56811284	0.1	77.57044792	0.54876714	5.487671398
42.09865602	55.2944002	0.1	97.39305622	0.567744789	5.677447895
51.27620396	71.715785	0.1	122.991989	0.58309314	5.830931397
63.13875476	92.90239105	0.1	156.0411458	0.595371118	5.953711156
78.46445797	120.2320213	0.1	198.6964793	0.605103934	6.051039342
98.25565283	155.4771329	0.1	253.7327858	0.612759334	6.12759345
123.8025531	200.9154042	0.1	324.7179573	0.618736199	6.187381993
156.764419	259.469624	0.1	416.2340429	0.623374345	6.233743452
199.272612	334.8835612	0.1	534.1561732	0.62693942	6.269394196
254.0607584	431.9412021	0.1	686.0019605	0.629650099	6.296500987
324.6280255	556.7369231	0.1	881.3649486	0.631675816	6.31675816
415.442061	717.0031981	0.1	1132.445259	0.633146011	6.331460107
532.1881013	922.4992858	0.1	1454.687387	0.634156379	6.341563789

Patch model | Epidemic curve | Prevalence by group | Incidence and IRR | Contribution to risk | Susceptibility and AR | RO Estimates

...below the purple arrow are numbers that provide a theoretical indication of the proportion of cases of COVID-19 that 'vaccinated' people got from the 'unvaccinated' after normalizing for their amount of contact people with the 'unvaccinated' group. A number larger than '1' indicates that cases of COVID-19 among the 'vaccinated' came disproportionately from contact with the 'unvaccinated'. The first row of data represents the starting point of a theoretical wave of COVID-19, hence the reason why column AI27 starts at '1'. As you move down the column, the time into the modeled wave of cases of COVID-19 increases. Note that the ratios rapidly rise well above '1', suggesting a bias in transmission coming from the 'unvaccinated'. This is the basis for Fisman, *et al.* promoting fear of the 'unvaccinated'.

However, I have provided an excellent-quality peer-reviewed scientific article as a reference that suggests that immunity among 'unvaccinated' people in British Columbia was ~90%. To be conservative, let's say it is only ~85% across Canada, despite this likely being an underestimate as most Canadians were exposed during the record-shattering waves caused by the Delta and Omicron variants sweeping through the country. So, let's change this single parameter in the mathematical model to see what happens. I changed the proportion of the 'unvaccinated' with immunity from 20% to a justifiable 85%...

	A	B	C	D	E	F
1	Model Symbology and Parameters					
2	**Parameter description**	**Symbol**		**Value**	**Plausible Range**	**Reference**
3	Time step	delta t		0.002	---	
4	Contacts per unit time	pc		436.800	164-728	Calculated
5	Rate of recovery from infection	gamma		73	41-91	Wolfel et al., 2020
	Rate of loss of immunity	rho		0.00	Up to 0.75	Set to zero but can increase to 0.75
6						Townshend 2021
7	Birth rate = death rate	mu		0.00	---	Set to zero
8	Basic reproduction number (R0)	Ro		6	4-8	Xia et al., 2021
9	Eta (mixing, 0 = random, 1 = assortative)	Eta		0.50	0-1	Assumption
10	Proportion vaccinated	Pv		0.8	---	Ontario data (approximate)
11	Vaccine efficacy	VE		0.8	0.7-0.9	Higdon et al., 2021
12	Population (N)	N		10000000	---	Statistics Canada
13	Baseline immunity in unvaccinated			0.85	0.1-0.5	Assumption
14	St = susceptibles at time t					
15	It = infectives at time t					
16	Rt = removed (immune) at time t					

...now look at the effect this had on the data (look under the purple arrow)...

	AD	AE	AF	AG	AH	AI
19	MODEL OUTPUTS--CONTRIBUTION TO RISK AMONG VACCINATED FROM UNVACCINATED					
20						
21						
22						
23						
24						
25	Vaccinated cases acquired from vaccinated	Vaccinated cases acquired from unvaccinated	Fraction of contacts with unvaccinated	Total incident cases	Fraction of cases acquired from unvaccinated	Ratio of fraction of infections acquired from unvaccinated to fraction of contacts unvaccinated
26						
27	12.57921101	1.397690112	0.1	13.97690112	0.1	1
28	12.94529405	1.377313697	0.1	14.32260835	0.090163608	0.961638082
29	13.31240981	1.360435465	0.1	14.67284527	0.092717904	0.927179044
30	13.68111687	1.340823905	0.1	15.02794077	0.089213211	0.892132111
31	14.05194626	1.330269429	0.1	15.38221569	0.086837191	0.868371911
32	14.42540157	1.32857958	0.1	15.75398113	0.084332941	0.843329409
33	14.80196188	1.323577651	0.1	16.12553953	0.082079589	0.820795886
34	15.18208398	1.321101658	0.1	16.50318563	0.080051312	0.800513117
35	15.56620141	1.321003046	0.1	16.88720746	0.078225074	0.782250736
36	15.95474138	1.323145781	0.1	17.27788712	0.076580299	0.76580299
37	16.34809635	1.327405291	0.1	17.67550164	0.075098592	0.750985923
38	16.74665591	1.333667793	0.1	18.08032371	0.073763491	0.737634909
39	17.15079306	1.341829292	0.1	18.49262235	0.07256025	0.725602405
40	17.56086868	1.351794943	0.1	18.91266382	0.071475851	0.714756509
41	17.97723281	1.363478344	0.1	19.34071116	0.070497839	0.704978391
42	18.40022588	1.376800912	0.1	19.77702678	0.069616173	0.696161727
43	18.8301797	1.391691302	0.1	20.221871	0.068821095	0.688210948
44	19.26741869	1.408084871	0.1	20.67550358	0.068104018	0.681040182
45	19.71226097	1.425923184	0.1	21.13818385	0.067457223	0.674572231
46	20.16501781	1.445153565	0.1	21.61017137	0.066873767	0.66873767
47	20.62599747	1.465728671	0.1	22.09172015	0.066347404	0.663474039
48	21.09550299	1.487606113	0.1	22.58310911	0.065872511	0.658725114
49	21.57383437	1.510748094	0.1	23.08458247	0.065440292	0.654402918
50	22.06129897	1.535121089	0.1	23.59641006	0.0650574	0.650574001

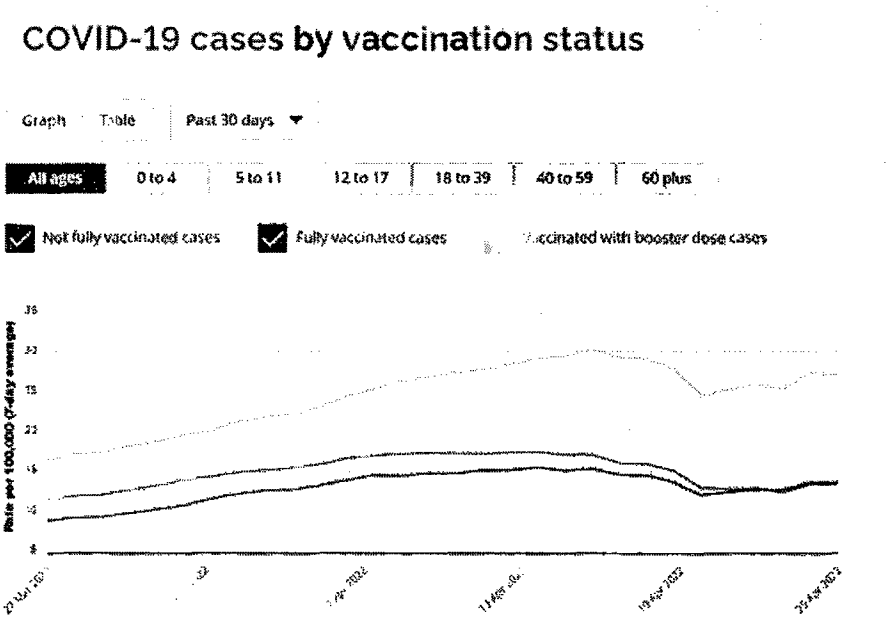
Patch model | Epidemic curve | Prevalence by group | Incidence and IRR | Contribution to risk | Assortativity and AR | SD Estimates

...remarkable, isn't it! **Correcting only this one assumption completely *reverses* the conclusions of the paper.** All of a sudden every ratio drops below '1', meaning that transmission is occurring disproportionately from 'vaccinated' people. Now the 'unvaccinated' are serving as a protective buffer for the 'vaccinated'.

Now one must ask how skewed the conclusions of the paper would go in the opposite direction if the several other incorrect assumptions were to be corrected. As you can plainly see, this paper by Fisman, *et al.* is nothing short of preposterous. How could a paper be allowed to be published in what used to be a respected medical journal when its conclusions get completely reversed when only one of several reasonable corrections are made! The paper by Fisman, *et al.* is a classic demonstration of 'garbage in, garbage out'. The authors are very intelligent, well-trained scientists who ply their trade well. They know what they are doing. As such, in my expert opinion, their paper represents **dis**information (*i.e.*, the deliberate dissemination of misinformation). I would be willing to stand in a court to justify my opinion. I honestly believe that the authors need to be investigated, as does the editor who allowed the paper to be published; and it would be great if the names of the reviewers who supported its publication could be made public. And the legacy media outlets that are promoting this 'scientific' garbage should be ashamed. They could use some immunology expertise and a return to investigative journalism to separate themselves from being labeled as propaganda artists.

Modeling Should Predict 'Real World' Data

Here are some 'real world' data that Fisman, *et al.* would be wise to consider in their modeling. This was copied from the website of the Ontario Ministry of health on April 26, 2022...



At the end of their paper, Fisman, *et al.* strongly promoted booster doses as a way to reduce 'infections'. Yet the 'real world' data clearly show that the boosted sub-population is being diagnosed with disproportionately more cases than the 'not fully vaccinated' group, which includes the 'unvaccinated' and people who received a single dose. Why would someone want to take a booster and more than double their risk of getting diagnosed with COVID-19?!? Which is

stronger evidence, the public health data shown above or contradictory messaging based on inappropriate massaging of a purely theoretical model? When mathematical models are misused they become no more utilitarian than toys.

A Direct Message to Fisman, *et al.*

Fisman and Tuite: Your paper, which can be flipped on its head by correcting just one of your multiple incorrect immunological assumptions, stigmatizes the 'unvaccinated' and could potentially be used to justify policies as draconian as forced inoculations. Why are you even promoting messaging based on theoretical models using assumptions that you are not qualified to opine on when concrete scientific evidence about transmission could be generated via biological sampling? You owe Canadians an apology for disseminating harmful disinformation. Should you wish to contest my immunological critiques, I would invite you to arrange a forum where we can have an objective third party moderate a respectful discussion about COVID-19 vaccinology in front of the Canadian public with equal representatives on both sides of the debate.

A Message to the Administration of the University of Toronto

You should launch an investigation into the academic conduct of Drs. Fisman and Tuite.

A Message to the College of Physicians and Surgeons of Canada

You should investigate Dr. Fisman and the harms that may be caused by his actions as a physician in publishing a misleading scientific paper in a medical journal.

A Message to the *Canadian Medical Association Journal*

Do the right thing and immediately retract the paper by Fisman, *et al.* There is a rumour that you may have sent this paper directly to Canadian physicians. If true, make it very clear to them that the paper represented disinformation disseminated by a physician who knows better. You should reconsider future recruitment of the services of the reviewers that promoted the publication of this paper and recommend that their academic institutions review their conduct. You should also review the conduct of your editorial board and peer review process.

A Message to the Legacy Media

Make the right choice and do everything that you can to blunt the profound harms caused by your rampant and widespread dissemination of misinformation. It is promoting hatred against critically thinking people who made highly informed and justified choices to avoid inoculations that are still in their initial clinical experimentation phase and for about which there is profound scientific debate. Protect the people that you have inappropriately placed at risk. You know where it will lead if you promote polarization of two groups of people and fuel feelings of anger and hatred in one of them against the other. Replace journalists with those who are willing to think critically and who will not support censorship of legitimate experts with 'dissenting' views. Nor should peer-reviewed scientific papers be treated like the gospel truth. The anonymous peer review process is fallible.

A Message to All Canadians

We are more alike than different. Do not fall into the trap set in the paper by Fisman, *et al.* to equate 'likeness' with 'vaccination' status. As an expert vaccinologist who has been closely following the accumulating science and, more importantly, as a fellow human being, I implore you to promote unity.

Correcting the Disinformation

Fisman had Tweeted, "*Our paper supports the idea that the decision to remain unvaccinated confers risk not only on the unvaccinated individual but (disproportionate to contact rates) on vaccinated individuals too*".

If Fisman were to demonstrate objectivity as a scientist, he would update his Tweet to state something like the following: "*After correcting just one of the several inappropriate immunological assumptions, our paper now supports the idea that the decision to get 'vaccinated' confers risk not only on the 'vaccinated' individual but (disproportionate to contact rates) on 'unvaccinated' individuals too; unlike our previous conclusion, this corrected model matches real-world data. Thank-you to those who chose to remained 'unvaccinated' since you are now selflessly serving as a buffer to the 'vaccinated'. We are sorry to the field of public health modeling for disclosing how easy it is for the conclusions of our models to be manipulated by assumptions that we sometimes pull out of thin air. We also apologize to 'unvaccinated' people (most of whom have received legitimate vaccines throughout their lifetimes) for misleading media organizations around the world into promoting hatred against you. Now that our model has been shown to point to the 'vaccinated' as the main culprits of transmission of SARS-CoV-2, we implore you to not promote hatred against us like we have done to you.*"

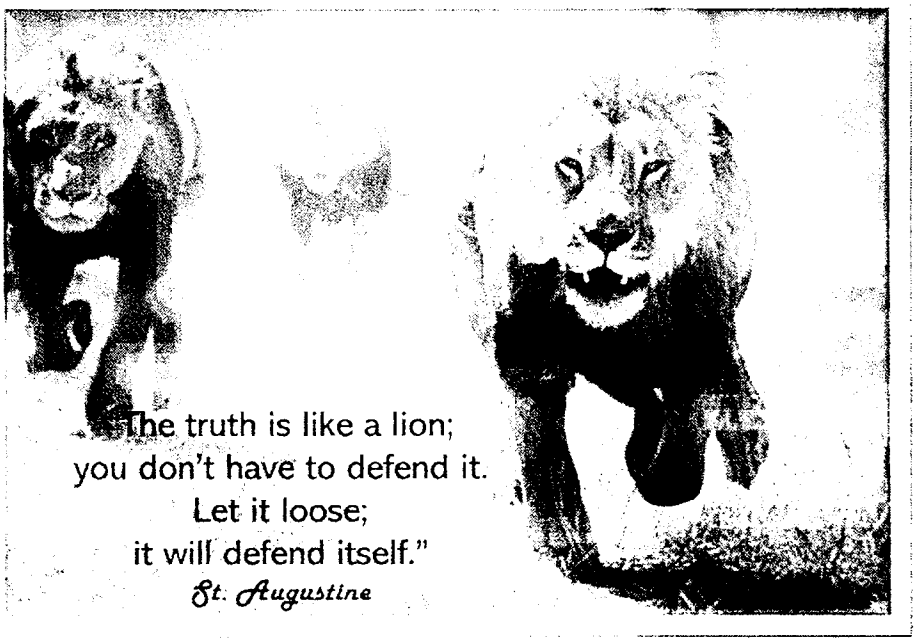
...don't worry Dr. Fisman, I can tell you sincerely and from experience that the vast majority of 'unvaccinated' people will not return ill wishes in kind, even as more hypocritical messaging is unveiled. I for one want to see unity restored among Canadians. Hatred, segregation, and mislabeling of people have no place here. Much healing needs to happen and your current misdeed has been very counterproductive.

Overall Conclusions

No government should be allowed to implement policies based on results of epidemiological modeling prior to full disclosure of the data, the model used to generate predictions, and the conflicts of interests of the modelers. As demonstrated by Fisman, *et al.*, public health models in the hands of the wrong people can result in devastatingly harmful and brutally misleading public messaging. This, in turn, could lead to the introduction of inappropriate health policies. And finally, multiple objective immunologists should always be consulted to advise on the immunological parameters being plugged into any health-related models.

In the name of good science, the correction of mis/dis-information, and in a desperate attempt to maintain some public faith in vaccinology,

Byram



724 Likes

322 Comments

Write a comment...

StellaMaris Apr 26, 2022 ♥ Liked by Dr. Byram W. Bridle

OMG!!!!!!!!!!!!!!! This was one of your best posts!!!! Knowing you are the side of getting the truth out makes me hopeful....you are doing such amazing work...we are all better and blessed because of your efforts and contributions!!! Forever grateful...."Three things cannot be long hidden: the sun, the moon, and the truth." Buddha

LIKE (125) REPLY ···

R. Jones Apr 26, 2022 ♥ Liked by Dr. Byram W. Bridle

I am both disgusted and horrified by what I have just read in the CTV article by Morgan Lowrie. Thank you Dr. Bridle for being a voice of sanity and common sense in this grotesque societal manipulation that is being orchestrated.

LIKE (105) REPLY ···

2 replies

320 more comments...

This is Exhibit "S" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Raymond MacNeil <raymond.macneil@utoronto.ca>
Reply-To: raymond.macneil@utoronto.ca
To: editorial@cmaj.ca, kim.barnhardt@cmaj.ca, cmajgroup@cmaj.ca, raymond.macneil@utoronto.ca

Wed, May 4, 2022 at 2:21 AM

05/03/2022

Dear CMAJ Editor

"Trust in public health is at an all-time low.." Dr. Makary, Johns Hopkins School of Medicine

I ask that the CMAJ retract the April 25, 2022 paper by Fisman et al. As a medical journal that shapes Canadian health policy, the CMAJ has an obligation to uphold scientific integrity and public trust. Fisman et al. (2022) Impact of population mixing between vaccinated and unvaccinated subpopulations on infectious disease dynamics: implications for SARS-CoV-2 transmission represents an abject failure of these obligations and questions the CMAJ's editors/peer review process, as well as the integrity and scientific acumen of your organization.

The Fisman et al. paper is inflammatory and has a sinister undertone. Among other serious concerns, the conclusions made by Fisman et al. (2022) overlook noteworthy factors around the realities of vaccine effectiveness and the percentage of the public with naturally acquired immunity. They also did not factor that current vaccines do not protect against disease or transmission, that 'vaccinated' people get infected and sick at least as much as, if not more than the 'unvaccinated, or that vaccines are subject to significant wane in protection over time. The authors make illogical, outrageous comparisons and conclusions that are not science. Rather, it is flagrant propaganda that fuels fear, incites hatred/ contempt of the unvaccinated and support of authoritarian, forced vaccination and segregation policies.

It cannot be overlooked that David Fisman is a strong fan of chilling COVID-Zero strategies (think Shanghai at present) and has financial conflicts of interest with COVID vaccine manufacturers, teachers unions, WE Charity etc. that may seriously bias his findings. His abrupt resignation from the controversial, self appointed Ontario Science table, was likely due to public exposure of these conflicts of interests. David Fisman is also well known for past, exaggerated modelling that served to unduly scare the public. It is troubling therefore, that the CMAJ saw fit to publish his work without robust and intense scrutiny.

I have provided links below to critical commentary from various sources expressing concern over the Fisman et al. paper. Since I am not a scientist, I ask you to refer the arguments made in the first article by Dr. Byram Bridle, and the second article by Dr. Jessica Rose to support my request to retract this both egregious and erroneous paper. As such, please answer to the fatal flaws, and the major and minor issues that Dr. Bridle describes and comment on the concerning modelling errors noted by mathematician Dr. Jessica Rose

Thank you for your time. Due to the serious threat posed by this dangerous paper that is being boldly paraded across the press as the gospel truth, your prompt reply is warranted.

Sincerely

Raymond MacNeil,raymond.macneil@utoronto.ca

Vancouver,BC

1. Dr Bridle: Fiction designed to promote hatred https://viralimmunologist.substack.com/p/fiction-disguised-as-science-to-promote?token=eyJ1c2VyX2lkIjozNzI2NzUwNSwicG9zdF9pZCI6NTI4MzY0MTAsIl8iOiJIdkxSWiIsImlhdCI6MTY1MTAyOTM3MSwiZXhwIjoxNjUxMDMyOTcxLCJpc3MiOiJwdWItNTkyMjE0Iiwic3ViIjoicG9zdC1yZWFjdGlvbiJ9.Qq0XD6T0r0slxhuTzDA7_AfjeoJUk20JEfcjrlS0_FQ&s=r

2. https://jessicar.substack.com/p/call-for-retraction-of-paper-entitled?token=eyJ1c2VyX2lkIjozNzI2NzUwNSwicG9zdF9pZCI6NTMwMTU4ODUsIl8iOiJIdkxSWiIsImlhdCI6MTY1MTE5NTAzOCwiZXhwIjoxNjUxMTk4NjM4LCJpc3MiOiJwdWItNTE2ODk2Iiwic3ViIjoicG9zdC1yZWFjdGlvbiJ9.aHLBSikmFKPR6wNjl8Zzt0dWg_NhLFuF5ZcqhqLv-9U&s=r

3. OCLA Statement on CMAJ Fisman et al. Article Claiming Disproportionate Infection Risk from Unvaccinated Population, and on Negligent Media Reporting

https://ocla.ca/ocla-statement-on-cmaj-fisman-et-al/

4. Lawyer Knia Singh: Human rights and legal critique of the recently published article regarding the impact of viral transmission between the vaccinated and the unvaccinated. https://www.instagram.com/tv/CcyxL2RlSc4/?utm_source=ig_web_copy_link

5. Jestre: Impact of population mixing: The tractability problem

https://jestre.substack.com/p/impact-of-population-mixing-the-tractability?s=r

6. David Chesnut: New Study Claiming Unvaccinated Spread COVID More Than Vaccinated – Pseudoscience, Propaganda, and Misleading Lies https://www.facebook.com/101852609908112/posts/5130922020334454/?sfnsn=mo

7. https://nakedemperor.substack.com/p/a-picture-is-worth-a-thousand-words-1fc/comments?token=eyJ1c2VyX2lkIjo0MDY2Njk5MCwicG9zdF9pZCI6NTMwMzE2MTgsIl8iOiJCbjhvMSIsImlhdCI6MTY1MTE1MzM1NCwiZXhwIjoxNjUxMTU2OTU0LCJpc3MiOiJwdWItNjAyMzczIiwic3ViIjoicG9zdC1yZWFjdGlvbiJ9.uXGhUF1KsN82khtO3VoxprS_RNRcjrrTnZx07zb949c&s=r

8. Press: Life Site Newshttps://www.lifesitenews.com/blogs/canadian-doctor-compares-unvaccinated-to-murderous-drivers-citing-insane-study/

Gmail

David N. Fisman <david.fisman@gmail.com>

Retract Fisman et al. 2022!

Sybilla Rulf <sybilla1@gmaail.com> Tue, May 3, 2022 at 1:12 PM
Reply-To: sybilla1@gmaail.com
To: editorial@cmaj.ca, kim.barnhardt@cmaj.ca, cmajgroup@cmaj.ca, sybilla1@gmaail.com

05/03/2022

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Thank you for your time. Due to the serious threat posed by this dangerous paper that is being boldly paraded across the press as the gospel truth, your prompt reply is warranted.

Sincerely

Sybilla Rulf,sybilla1@gmaail.com

Vancouver,B.C.

1. Dr Bridle: Fiction designed to promote hatred https://viralimmunologist.substack.com/p/fiction-disguised-as-science-to-promote?token=eyJ1c2VyX2lkIjozNzI2NzUwNSwicG9zdF9pZCI6NTI4MzY0MTAsIl8iOiJIdkxSWiIsImlhdCI6MTY1MTAyOTM3MSwiZXhwIjoxNjUxMDMyOTcxLCJpc3MiOiJwdWItNTkyMjE0Iiwic3ViIjoicG9zdC1yZWFjdGlvbiJ9.Qq0XD6T0r0sIxhuTzDA7_AfjeoJUk20JEfcjrlS0_FQ&s=r

2. https://jessicar.substack.com/p/call-for-retraction-of-paper-entitled?token=eyJ1c2VyX2lkIjozNzI2NzUwNSwicG9zdF9pZCI6NTMwMTU4ODUsIl8iOiJIdkxSWiIsImlhdCI6MTY1MTE5NTAzOCwiZXhwIjoxNjUxMTk4NjM4LCJpc3MiOiJwdWItNTE2ODk2Iiwic3ViIjoicG9zdC1yZWFjdGlvbiJ9.aHLBSikmFKPR6wNjI8Zzt0dWg_NhLFuF5ZcqhqLv-9U&s=r

3. OCLA Statement on CMAJ Fisman et al. Article Claiming Disproportionate Infection Risk from Unvaccinated Population, and on Negligent Media Reporting

https://ocla.ca/ocla-statement-on-cmaj-fisman-et-al/

-

4. _Lawyer Knia Singh: Human rights and legal critique of the recently published article regarding the impact of viral transmission between the vaccinated and the unvaccinated. https://www.instagram.com/tv/CcyxL2RlSc4/?utm_source=ig_web_copy_link

5. Jestre: Impact of population mixing: The tractability problem

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6. David Chesnut: New Study Claiming Unvaccinated Spread COVID More Than Vaccinated – Pseudoscience, Propaganda, and Misleading Lies https://www.facebook.com/101852609908112/posts/5130922020334454/?sfnsn=mo

7. https://nakedemperor.substack.com/p/a-picture-is-worth-a-thousand-words-1fc/comments?token=eyJ1c2VyX2lkIjo0MDY2Njk5MCwicG9zdF9pZCI6NTMwMzE2MTgsIl8iOiJCbjhvMSIsImlhdCI6MTY1MTE1MzM1NCwiZXhwIjoxNjUxMTU2OTU0LCJpc3MiOiJwdWItNjAyMzczIiwic3ViIjoicG9zdC1yZWFjdGlvbiJ9.uXGhUF1KsN82khtO3VoxprS_RNRcjrrTnZx07zb949c&s=r

8. Press: Life Site Newshttps://www.lifesitenews.com/blogs/canadian-doctor-compares-unvaccinated-to-murderous-drivers-citing-insane-study/

This is Exhibit "T" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN


BRIGHT LIGHT NEWS INVITES TIMOTHY CAULFIELD, DR THERESA TAM AND OTHERS FOR A RESPECTFUL DEBATE ON COVID-19 SCIENCE WITH OUR PANELISTS JAN 28th
Open Invite to Covid-19 Panel Debate
Dr Byram Bridle, Viral Immunologist
Dr Mark Trozzi, ER Doctor
Timothy Caulfield, Prof. of Health and Law
Dr Theresa Tam, CPHO Canada
Dr Kieran Moore, CMOH Ontario
Dr Patrick Phillips, ER, Family Doctor
Rodney Palmer, Former Journalist
David Fisman, Epidemiologist
MOH Toronto
GlobalNews Reporter
Jan 28th 7pm
RSVP: info@brightlightnews.com
BUY TICKETS: brightlightnews.com/panel
LOCATION: The Zoetic, 526 Concession St, HAMILTON, ON
BRIGHT LIGHT NEWS

This is Exhibit "U" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

1  LILLEY: WestJet pilots to strike because $300K a year ain't enough TRENDING

2  MAPLE LEAFS NOTES: Coyotes failed arena deal should end Austo... TRENDING

3  Jays fa* a... TREI

Ontario

Pro-trucker docs push to end COVID mandates

Postmedia News

Published Feb 08, 2022 • 1 minute read

Join the conversation

Anti-vaccination protesters holding signs take part in a rally against Covid-19 vaccine mandates. PHOTO BY RINGO CHIU /AFP via Getty Images

part in a public scientific forum on Friday at the Marriott Hotel in Ottawa.

A press release from Road Warrior News states that Dr. Byram Bridle, Dr. Paul Alexander, and Dr. Roger Hodkinson have invited federal senior health officials, including Dr. Theresa Tam and Dr. Howard Njoo, a health discussion.

Bridle wants to see the evidence used by federal officials in the decision to lock down the country during COVID-19.

The doctors' letter to Tam claims, in part:

"Since the beginning of this declared pandemic, Canadian experts in immunology, virology, epidemiology, evidence-based medicine, academic science, and clinical medicine have been

justify the restrictions and mandates.

“Given that the government claims we are in a state of emergency, we feel this discussion needs to be expedited and brought before the Canadian public.

“Specifically, we are requesting that the health authorities supply the associated evidence and data that demonstrably justify their case for the public health emergency and the initiation and continuation of mandates.”

The doctors are calling upon the governments of Canada at every level to publicly declare an end to the health emergency and an end to all COVID mandates. And, they claim, “the evidence is clear that there is no health emergency, and the mandates cannot be supported scientifically.”

The invitation comes at a time when other countries, such as the U.K., are dropping their COVID mandates.

The letter notes that Canada’s national media outlets are invited to the event, which will be livestreamed.

As of Feb. 8, there were 5.75 million deaths worldwide from COVID-19, with the United States responsible for the lion’s share with 904,000 deaths. India is a distant second with 504,000 deaths.

Postmedia is committed to maintaining a lively but civil forum for discussion and encourage all readers to share their views on our articles. Comments may take up to an hour for moderation before appearing on the site. We ask you to keep your comments relevant and respectful. We have enabled email notifications—you will now receive an email if you receive a reply to your comment, there is an update to a comment thread you follow or if a user you follow comments. Visit our Community Guidelines for more information and details on how to adjust your email settings.

JOIN THE CONVERSATION

TRENDING

1 
LILLEY: WestJet pilots to strike because $300K a year ain't enough

2 
MAPLE LEAFS NOTES: Coyotes failed arena deal should end Auston Matthews rumours

3 
Jays manager chews the fat about dugout dustup

4 
SIMMONS: Is this finally it for Coyotes in Arizona?

5 
GOTCHA! Escaped killer Joshua Kohl who beat man to death recaptured

LATEST N/

THIS WEEK IN FLYERS

This is Exhibit "V" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

GUELPHTODAY.com

Controversial U of G prof called as vaccine 'expert' in family court fight

GuelphToday Staff
Nov 11, 2022 8:00 AM

Byram Bridle, associate professor in U of G's Department of Pathobiology. | Kenneth Armstrong/GuelphToday file photo

Listen to this article
00:03:26

A Toronto mother chose a controversial University of Guelph professor as her expert witness in a battle with the father over who should should have the final say in their son's vaccinations.

U of G professor Byram Bridle, whose stance on COVID-19 vaccines was front page news during the peak of the pandemic and has been criticized by much of the scientific and medical

over who would control if and what vaccines the boy would receive.

The father wanted the boy to receive standard vaccinations and the COVID vaccine. The mother did not.

The court ruling determined the father, who does not have custody of the child, was best to make those decisions.

"In deciding this issue, the court must determine which parent is best capable of making vaccination decisions in their child's best interests," said Justice Sheilagh O'Connell in her written decision granting control to the father.

The child had COVID last February, reinforcing the father's quest to get him vaccinated for COVID.

The father's expert witness was Dr. Abdu Sharkawy, an internal medicine and infectious diseases specialist at the University Health Network. He testified that the 11 year old should receive the COVID vaccine at the appropriate time.

"Dr. Sharkawy also discussed the concerns raised by some, including the mother's proposed expert, about the MRNA technology used to create the vaccine. He explained that this technology is not new and that it has been around for many years," said the written judgment.

"Dr. Sharkawy's medical practice is at Toronto Western Hospital, where he has worked on the Covid ward and the Intensive Care Unit (ICU) attending Covid patients since the beginning of the pandemic. He is trained in pediatric infectious diseases. He has had extensive first-hand, front line "real world" experience treating Covid patients throughout the pandemic."

The judge noted that Sharkawy's opinions regarding vaccines for children are shared by numerous health organizations around the world.

While acknowledging that Bridle is an expert in his research field, she ruled he was not qualified to give expert opinion on this case.

"However ... the court does not accept that Dr. Bridle is qualified to give opinion evidence with respect to the safety and efficacy of the Covid-19 vaccine for children," the ruling states.

"Dr. Bridle acknowledged that he is not a medical doctor. He has never vaccinated a child, he has never treated a child or an adult suffering from a reaction to a vaccine, nor has he ever treated a child or an adult who is suffering from an infectious disease."

illness and death, regardless of the shorter duration of immunity, Bridle would not acknowledge that receiving the vaccine prevented severe or serious illness and death. In fact, he stated that vaccinated people are at greater risk than unvaccinated people given his interpretation of hospital admissions.

"Respectfully, this is so far removed from the mainstream and widely accepted views of the Canadian and international medical and scientific community that the court cannot accept Dr. Bridle's evidence on the Covid vaccine as reliable," the judge ruled.

This is Exhibit "W" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

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KATHERINE R. COSTIN

https://www.guelphmercury.com/news/u-of-guelph-professor-spoke-at-event-for-far-right-german-politician/article_c336e83f-2120-5895-a1bb-8b7c4aed24c4.html

NEWS

U of Guelph professor spoke at event for far-right German politician

After lunch meeting with 3 MPs became public last week, Conservative leader Pierre Poilievre called Anderson's views "vile" and that her "racist, hateful views are not welcome here"

By **Graeme McNaughton** Guelph Mercury

Mar 2, 2023

A University of Guelph professor was a featured speaker at a Toronto event headlined by a politician from a far-right German political party classified as a suspected threat to democracy by that country's domestic intelligence agency.

Dr. Byram Bridle, a viral immunologist with the Ontario Veterinary College at the University of Guelph, spoke for approximately 30 minutes at the Feb. 21 event at the Eglinton Grand Theatre in Toronto, headlined by Christine Anderson, a member of the European Parliament for Alternative for Germany (AfD).

The University of Guelph declined to comment on this story. Bridle did not respond to several requests for comment prior to publication.

ARTICLE CONTINUES BELOW

Bridle's talk, video of which was obtained by the Mercury Tribune, focused on his views on the efficacy and safety of COVID-19 vaccinations — something several immunologists and scientists

potentially dangerous.

Bridle is currently suing the University of Guelph, along with a number of school administrators and researchers working both with and independently from the university, for $3 million, alleging "overlapping conspiracies" against him due to his views on COVID-19 vaccinations. His allegations have not yet been tested in court.

WHO IS CHRISTINE ANDERSON?

Anderson has been a member of AfD since 2013, and was the party's parliamentary group leader between 2013 and 2016, and would go on to be elected to the European Parliament in 2019.

ARTICLE CONTINUES BELOW

Christine Anderson, a member of European Parliament for Alternative for Germany (AfD) | ChristineAnderson.eu

immigration views, made waves in the European Parliament last year after claiming Islam was at fault for the loss of women's rights in Afghanistan: "Call the devil by its name and stop using apologetic terms to downplay the true nature of the most despicable and horrific ideology women suffer from worldwide," she said at the time.

Anderson's influence also extends to Canada, where she's found support from the "Freedom Convoy" movement after voicing her support for the protests with YouTube videos and her appearances on the right-wing website Rebel News.

"It is simply not enough for just a handful of politicians to stand up and raise their voices against this whole COVID madness, this is not enough," she told Rebel's Ezra Levant at the time. "We need the people taking to the streets."

Anderson also made headlines in Canada after rebuking Prime Minister Justin Trudeau during his speech to the European Parliament last year, calling him "a disgrace to democracy." At her Toronto event, Anderson recreated part of that speech to a man introduced as a Trudeau impersonator.

Last month, the German politician took part in a cross-country tour, dubbed "What Would Christine Anderson Do?", seeing her speak at events in Alberta, Ontario and Quebec. Other scheduled speakers at these events included Dr. Crystal Luchkiw, a Barrie-based doctor who had her medical licence suspended for, among other things, spreading false information about COVID-19 and revealing identifying information about a patient during a talk show; and Patrick Provost, a Quebec professor suspended without pay in mid-2022 by Université Laval for comments he made about COVID-19 vaccinations.

> We're deeply concerned by CPC MPs @LeslynLewis @DeanAllisonMP @ColinCarrieCPC meeting with @AndersonAfDMdEP — a member of the far-right German AFD Party known for Islamophobic and anti-immigrant views. We raised this directly with @CPC_HQ. pic.twitter.com/DgG8m5UjPk
>
> — CIJA (@CIJAinfo) Feb. 24, 2023

Anderson's Canadian tour drew controversy late last week when three Conservative MPs — Dean Allison of Niagara West, Colin Carrie of Oshawa and Leslyn Lewis of Haldimand-Norfolk — were photographed with her at a restaurant.

In a statement released Feb. 24, Conservative leader Pierre Poilievre called Anderson's views "vile" and that her "racist, hateful views are not welcome here."

WHAT IS THE AfD?

Anderson's party, the AfD, was officially formed in early 2013, initially as a conservative option for those opposed to the European Union. However, the party would move further to the right in subsequent years, basing part of its platform in 2016 on opposing Islam, calling for national bans on facets of the religion such as public calls to prayer and burqas and using the slogan "Islam is not a part of Germany."

RELATED STORIES

Immunologists raise concerns on U of Guelph prof's views on COVID-19 vaccine safety

Professor files $3M lawsuit against University of Guelph, researchers

Oshawa MP Colin Carrie said he 'was not aware' he was meeting with European politician who espouses 'racist, hateful views'

Niagara MP facing criticism for meeting with far-right German politician

An AfD poster the following year, showing a pregnant white woman, said "New Germans? We make them by ourselves."

According to a 2020 profile of the party published by the BBC, a former leader of the party, Frauke Petry, said German police should "if necessary" shoot at migrants seeking to enter the country illegally.

That same profile noted some of the party's rhetoric "has been tinged with Nazi overtones," pointing to party leaders' comments Germans should be "proud" of its soldiers in both world wars and condemning a Holocaust memorial in Berlin.

A German court ruled in March 2022 that AfD could be classified as a suspected threat to democracy, with The Guardian reporting at the time the administrative court in Cologne determined there were "sufficient indications of anti-constitutional goals within the AfD." The ruling cleared the way for the country's domestic intelligence agency, BfV, to put a radical faction of the party, Der Flügel (The Wing), under surveillance.

At the time of its dissolution in 2020 and shortly after BfV first announced it would be putting the faction under surveillance, Anderson was chair of Der Flügel in the Hesse region, according to German media reporting at the time.

ARTICLE CONTINUES BELOW

Graeme McNaughton
Graeme McNaughton is a reporter/photographer with the Guelph Mercury Tribune.

Read more about

REPORT AN ERROR JOURNALISTIC STANDARDS ABOUT US

This is Exhibit "X" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

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KATHERINE R. COSTIN

constitutionalrightscentre.ca/dr-byram-bridle-sues/

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Incorporated November, 2004

Rocco Galati - Executive Director (Founder) • Paul Slansky • Amina Sherazee - Operational Co-Directors

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Published : DECEMBER 22, 2022 | Vol. I Issue: 38

Dr. Byram Bridle sues University of Guelph, their professors, and others for Tortious conduct against him.

PUBLISHED: DECEMBER 22, 2022 | by the Constitutional Rights Centre

Dr. Byram Bridle sues University of Guelph, their professors, and others for Tortious conduct against him.

On December 19th, 2022, Dr. Byram Bridle issued a Statement of Claim in Ontario Superior Court. The expert vaccinologist, and viral immunologist, states that he has been viciously and falsely attacked by some of his colleagues, with the complicity of the University administration. Some of the Defendants include the President of University of Guelph, Dean Wichtel, Administrator Arnott, Professor Pyle, Professor Weese, and Dr. David Fisman at the University of Toronto.

Click here for the PDF Statement of Claim.

Rocco Galati, BA, LLB, LLM, Executive Director

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This is Exhibit "Y" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

Court File No. CV-22-00691880-0000

ONTARIO
SUPERIOR COURT OF JUSTICE

B E T W E E N:

DR. BYRAM BRIDLE

Plaintiff

and

UNIVERSITY OF GUELPH, JEFFREY WICHTEL, LAURIE ARNOTT, CHARLOTTE YATES, SCOTT WEESE, GLEN PYLE, ANDREW PEREGRINE, DOROTHEE BIENZLE, AMY GREER, DAVID FISMAN, NICK DULEY, JANE OR JOHN DOE JUNIOR SCIENTIST

Defendants

REQUEST TO INSPECT DOCUMENTS

You are requested to produce for inspection the following documents referred to in your Statement of Claim:

1. All documents, emails, Twitter posts, and other social media statements referred to in the Statement of Claim allegedly made by the Defendant Dr. Fisman. In particular, the Defendant Dr. Fisman requests to inspect:

   (a) The social media statements referred to at paragraph 58 of the Statement of Claim alleged to demonstrate the beginning of "a targeted and vicious campaign of personal attacks and harassment aimed at the Plaintiff, over social media, in order to label the Plaintiff, a career vaccinologist, as "an 'antivaxx-er' and disseminator of 'misinformation', in order to silence and discredit him" by Dr. Fisman and Defendants Pyle and Weese;

(b) The email(s) referred to at paragraph 59 of the Statement of Claim that allegedly "clearly manifest a conspiracy hatched and instigated by Fisman, agreeing with Pyle and Weese to destroying [sic] the Plaintiff's reputation and work";

(c) The social media posts referred to at paragraph 60 of the Statement of Claim that allegedly show that after "every interview, speech or article written by the Plaintiff," Dr. Fisman, along with Defendants Pyle and Weese, "would immediately post over social media labelling him as providing 'misinformation', and/or seeking to discredit the Plaintiff as an individual who provides 'misinformation'";

(d) The social media posts on Twitter in which Dr. Fisman allegedly directed people to a false website, "byramdridle.com", referred to in paragraph 64 of the Statement of Claim;

(e) The social media post from May 31, 2021, made by @byrambridle, and the social media post by Dr. Fisman that the @byrambridle post was allegedly responding to, referred to in paragraph 66(a) of the Statement of Claim;

(f) The social media posts referred to at paragraph 68 of the Statement of Claim in which Dr. Fisman allegedly references the "byrambridle.com" website;

(g) The social media posts referred to at paragraph 68 of the Statement of Claim in which @byrambridle reposts tweets by Dr. Fisman;

(h) The May 29, 2021, social media post by @DFisman referred to at paragraph 73(a) of the Statement of Claim;

(i) The May 30, 2021, social media post by @DFisman referred to at paragraph 73(d) of the Statement of Claim;

(j) The social media posts in which Dr. Fisman allegedly continues to "smear the Plaintiff's reputation online" and refers to the Plaintiff as an "anti-vaxxer" and "purveyor of 'misinformation'" referred to at paragraph 83 of the Statement of Claim;

(k) The social media posts and correspondence with the press referred to at paragraph 85 of the Statement of Claim through which Dr. Fisman allegedly co-conspired with the Defendants, Weese and Pyle, "to personally malign the Plaintiff as 'dangerous' and harass the Plaintiff as a purveyor of 'misinformation' to the public at large through social media and to the press";

(l) The email(s), dated on or around June 2, 2021, from Dr. Fisman and the Defendant Pyle to a *USA Today* journalist referred to at paragraph 85 of the Statement of Claim;

(m) A copy of the workplace harassment report, including against Dr. Fisman, filed by the Plaintiff on June 2, 2021, referred to at paragraphs 86 and 87 of the Statement of Claim;

(n) The social media posts from @DFisman from in or around June 2021 referred to at paragraph 89 of the Statement of Claim;

(o) The emails from the Plaintiff to Dr. Fisman on June 12 and 14, 2021, including any attachments, referred to at paragraph 90 of the Statement of Claim;

(p) The email from the Plaintiff to Dr. Fisman on June 14, 2021, including any attachments, referred to at paragraph 91 of the Statement of Claim;

(q) The email in which the Plaintiff sent "COVID-19 Vaccines and Children: A Scientist's Guide for Parents" to Dr. Fisman, referred to at paragraph 92 of the Statement of Claim;

(r) The social media posts from June 21, 2021, referred to at paragraph 96(a) of the Statement of Claim;

(s) The social media posts by Dr. Fisman referred to in paragraph 106 of the Statement of Claim that allegedly "reached members of the University, as well as members of the general public through the world wide web";

(t) Social media posts, or other documents or emails, in which Dr. Fisman allegedly "circulated widely on the internet" the July 6, 2021, open letter from University of Guelph faculty members, referred to at paragraphs 111 and 112 of the Statement of Claim;

(u) Documents, social media posts, or emails in which scientists request "that … the co-conspirator Defendants, cease their conduct intended to silence the Plaintiff, cause him harm, and push to remove him from campus" referred to at paragraph 114 of the Statement of Claim;

(v) The social media post from November 10, 2021, in which the Defendant Weese allegedly re-posts an old post from the Defendant Fisman "inviting the public to link 'anti-vaxxers,' to neo-Nazi White Supremacists", as well as the original "old

post from the Defendant Fisman inviting the public to link 'anti-vaxxers,' to neo-Nazi White Supremacists" referred to at paragraph 151 of the Statement of Claim;

(w) The social media post, or other document or email, in which Dr. Fisman allegedly labels the Plaintiff as an "'anti-vaxxer,' white 'Neo-Nazi'" as referred to at paragraph 214 of the Statement of Claim.

March 10, 2023

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DR. BYRAM BRIDLE
Plaintiff

-and-

UNIVERSITY OF GUELPH et al.
Defendants

Court File No. CV-22-00691880-000

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REQUEST TO INSPECT DOCUMENTS

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This is Exhibit "Z" referred to in the Affidavit of Dr. David Fisman sworn by Dr. David Fisman at the City of Toronto, in the Province of Ontario, before me on May 26, 2023 in accordance with O. Reg. 431/20, Administering Oath or Declaration Remotely.

Commissioner for Taking Affidavits (or as may be)

KATHERINE R. COSTIN

ROCCO GALATI LAW FIRM
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May 3, 2023

Via E-mail: jlilles@litigate.com

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RE: **Bridle v. University of Guelph et al.; CV-22-00691880-0000**

Dear Jaan:

This is further to your request to inspect documents.

I apologize for my delay in responding which was caused by a 3-week illness. My client is currently in Europe, addressing the European Parliament on invitation.

With respect to your request I have two preliminary observations:

(a) Your request lies outside of the scope of **Rule 30.01** in that you have yet not pled; and
(b) Regardless of (a) above, the vast majority of documents you seek originate from your client and we will not search nor produce them because they originate from your client. Rule 30.01, which applies to Rule 30.02 to 30.11 reads:

> 30.01 (1) In Rules 30.02 to 30.11
>
> (a) ….
> (b) A document shall be deemed to be **in a party's power if** that party is entitled to obtain the **original document** or a copy of it and **the party seeking** it is not so entitled.

The documents originally from Dr. Fisman that you seek are in the control of Dr. Fisman and originate from him, not in my client's control. Your client is entitled to obtain the original or a copy.

With respect to the other documents that you seek, namely in paragraph 1(m), (p), (q), while it is my view that these would be available upon their inclusion in any affidavit of documents, and

not at this stage, upon consultation with my client, and his access to his originals, I will likely provide them. These documents I believe may have attached copies, as part of these three documents, other documents I say Dr. Fisman needs to fetch himself.

I will send these three documents very shortly.

On the issue of pleading, if a statement of defence is not served and filed within twenty-one (21) days, we will move to note your client in default.

We wish to move this matter along and inspection and/or production of the documents you seek are not relevant nor necessary to your client's pleading. Enough time has lapsed.

Since your client is the author and owner of these documents you seek, this is a dilatory request. If your client wishes to deny the existence of these documents, your client can simply deny them, and the facts pleaded with respect to them.

Yours very truly,
ROCCO GALATI LAW FIRM PROFESSIONAL CORPORATION
Per:

Rocco Galati, B.A., LL.B, LL.M.

Dr. BYRAM BRIDLE
Plaintiff

-and-

UNIVERSITY OF GUELPH et al.
Defendants

Court File No. CV-22-00691880-000

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Dr. BYRAM BRIDLE
Plaintiff

-and-

UNIVERSITY OF GUELPH et al.
Defendants

Court File No. CV-22-00691880-000

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MOTION RECORD OF DEFENDANT DAVID FISMAN
(Returnable November 19, 2024)

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RCP-F 4C (September 1, 202