

# Digital contact tracing for COVID: from initial theoretical evidence to evaluation

 Ferretti\* & Wymant\* ... Frasert, Science 2020

 Wymant\* & Ferretti\* ... Frasert, Nature 2021

 Ferretti\* & Wymant\* ... Frasert, in prep.

\*equal contribution, †corresponding author

[www.coronavirus-fraser-group.org](http://www.coronavirus-fraser-group.org)



**PANDEMIC SCIENCES  
INSTITUTE (PSI)**



 @ChrisWymant



Also: Marcos Charalambides, Matthew Ayres, Luke Milsom, Lele Zhao, Anel Nurtay, James Petrie Adam Fowler, Andrea Di Francia, Jasmina Panovska-Griffiths



# Renewal equation recap: basics

Basic idea:

Partition all new infections today according to their infector's age of infection (i.e. when their infector was infected)

Num infected today =  $\sum_{\tau}$  [num infected today by someone infected  $\tau$  days ago]

*Intuitive deterministic limit:*

Num infected today =  $\sum_{\tau}$  [num infected  $\tau$  days ago  $\times$  infectiousness at age-of-infection  $\tau$ ]

Continuous limit:

$$I(t) = \int_{\tau} I(t - \tau) \beta(\tau) d\tau$$

$I(t)$  = incidence at calendar time  $t$

$\beta(\tau)$  = infectiousness (instantaneous hazard for transmitting) at age-of-infection  $\tau$

c.f. demography:  $I(t)$  = birth rate,  
 $\beta(\tau)$  = fecundity rate

Factorise  $\beta(\tau) = R \omega(\tau)$ ,  
try ansatz:  $I(t) \propto e^{rt}$ , get

$$1/R = \int_{\tau} e^{-r\tau} \omega(\tau) d\tau$$

Wallinga & Lipsitch 2007

Fraser 2007

Grassly & Fraser 2008

# Renewal equation recap: extensions

As the basis for  $R(t)$  inference:

Fraser 2007, Cori et al 2013, Flaxman et al 2020, Abbott et al 2020, Gostic et al 2020

With  $\tau$ -dependent intervention, e.g. isolation upon symptoms, of effectiveness  $\varepsilon$ :

$$\beta(\tau) \rightarrow \beta(\tau) \times (1 - \varepsilon \text{ Prob(intervention by } \tau))$$

With constant heterogeneity between cases:

Scalar  $I(t)$ , scalar  $\beta(\tau) \rightarrow$  vector  $I(t)$ , matrix  $\beta(\tau)$   
or  $\rightarrow$  function  $I(t)$ , integral kernel  $\beta(\tau)$   
see Diekmann, Heesterbeek, Britton

As the expectation of a stochastic process:  
Pakkanen...Bhatt 2021, Penn...Bhatt 2022

With a second level of partitioning by  $\tau'$ : let  $\tau'$  be the age of infection of my infector's infector, at time of transmission:

Incidence as a double integral over  $\tau, \tau'$

Fraser & Riley et al 2004

Müller ????

Ferretti & Wymant et al 2020 - upcoming slides

Grassly et al 2020

Scarabel et al 2021

↑ *natural framework for modelling contact tracing*: my hazard for becoming infected depends on my infector's probability of having been contact traced, which depends on their infector's age of infection.

# 2020 February

Luca estimated the generation time distribution  $\omega(\tau)$  from 40 transmission pairs with enough data on timing. This,

- + Model for decomposing infectiousness  $\beta(\tau) \longrightarrow$
- + incubation period distribution  $s(\tau)$  (Lauer et al)
- + Proportion asymptomatic  $P_a = 40\%$  (Diamond Princess)
- + Relative infectiousness of asymptomatics  $x_a = 10\%$  (guess; few missing links in Singapore tracing)
- + Fraction of all transmission via fomites = 10% (ditto)
- + Model for timing of fomite transmission  $E(l)$  = box function
- +  $r = 0.14/\text{day}$ , doubling time = 5.0 days (Imperial Report 4)

Into the renewal equation,  $I(t) = \int_{\tau} I(t - \tau) \beta(\tau) d\tau$

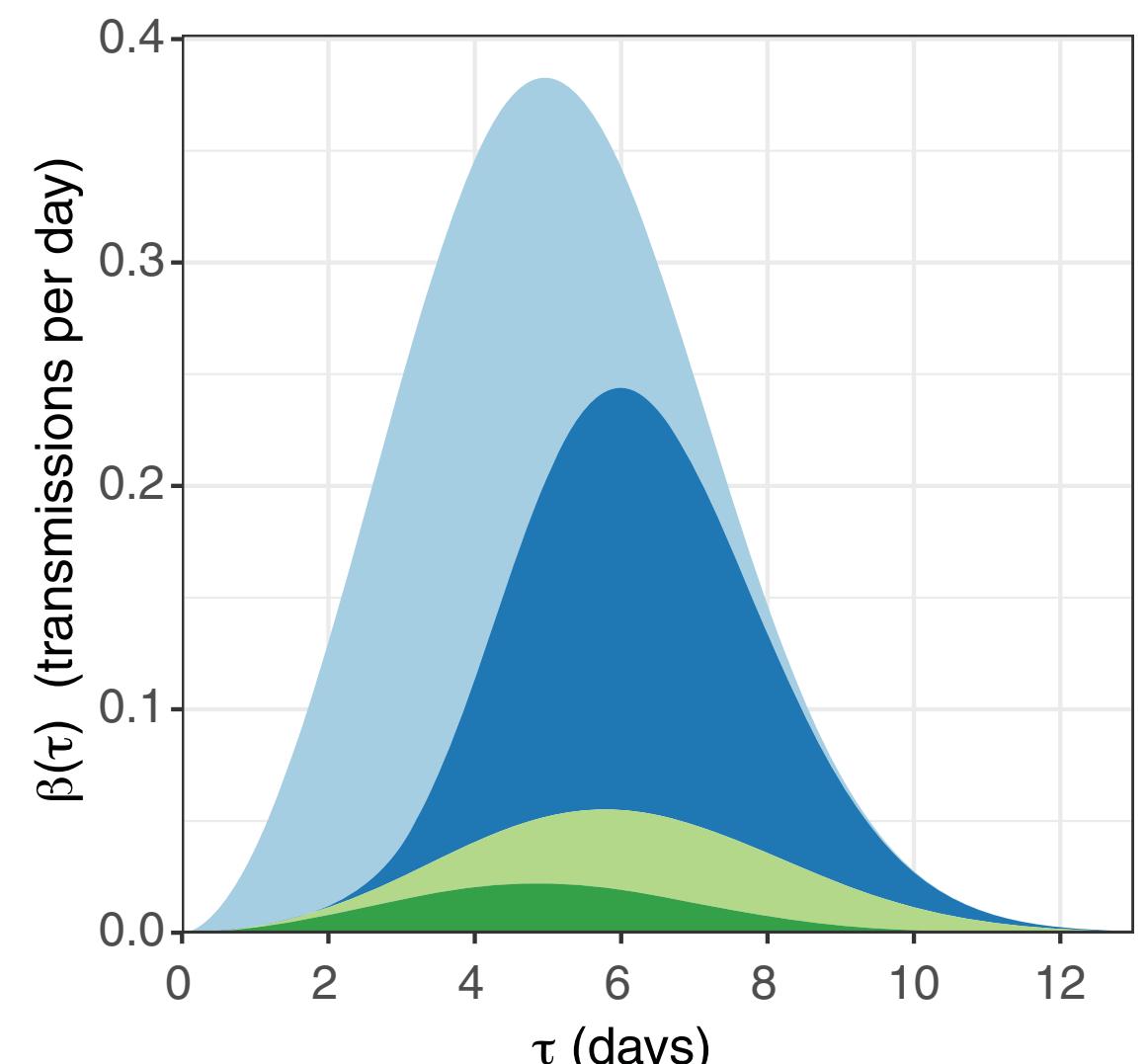
Gives transmission decomposed by route, timing, and magnitude

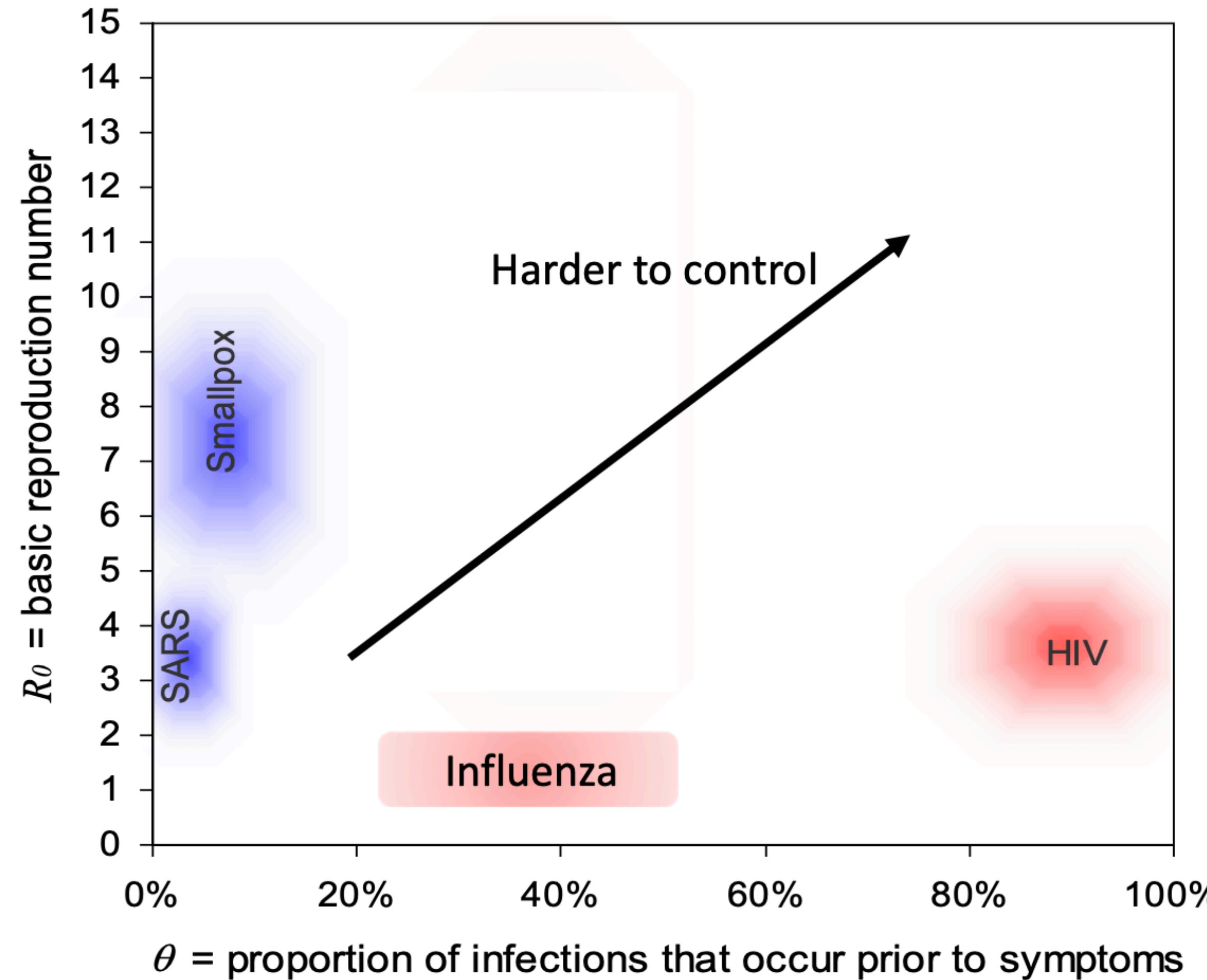
~half of transmission pre-symptomatic

$$\beta(\tau) = \underbrace{P_a x_a \beta_s(\tau)}_{\text{asymptomatic}} + \underbrace{(1 - P_a)[1 - s(\tau)]\beta_s(\tau)}_{\text{presymptomatic}} + \underbrace{(1 - P_a)s(\tau)\beta_s(\tau)}_{\text{symptomatic}} + \underbrace{\int_{l=0}^{\tau} \beta_s(\tau - l)E(l)dl}_{\text{environmental}}$$

$R_0 = 2.0:$

$R_p = 0.9$ from pre-symptomatic
$R_s = 0.8$ from symptomatic
$R_e = 0.2$ from environmental
$R_a = 0.1$ from asymptomatic

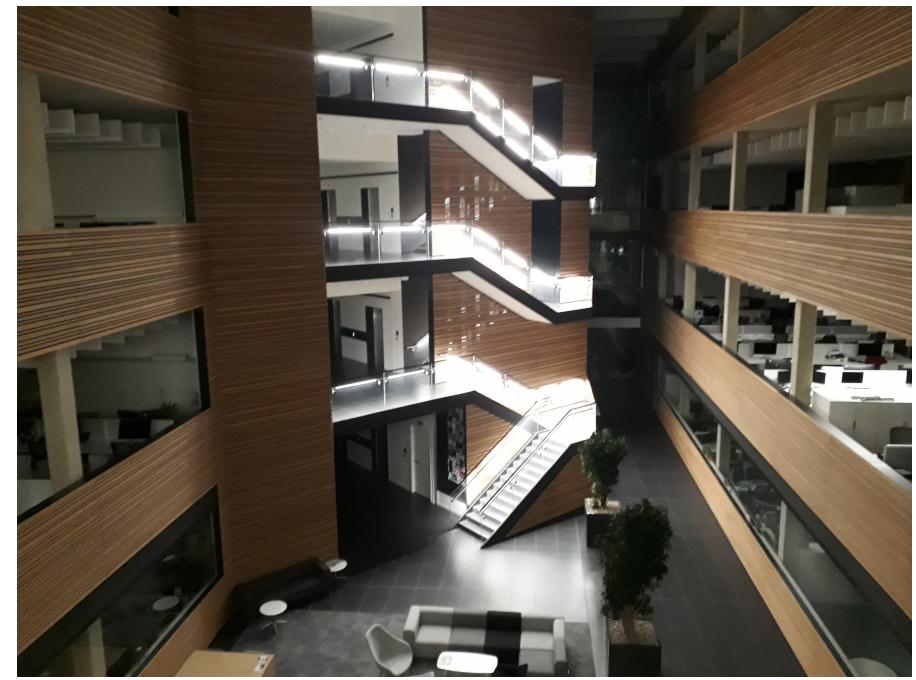




Symptoms (and screening) are important for reactive controls in an early stage epidemic – Fraser et al PNAS 2004  
\* n.b. influenza relocated

# 2020 March week 1

5 Mar, 04:44  
Big Data Institute



3 Mar: Christophe & David Bonsall independently reinvent idea of app-based contact tracing. Fast! Also automated, scales, anonymous, remembers.

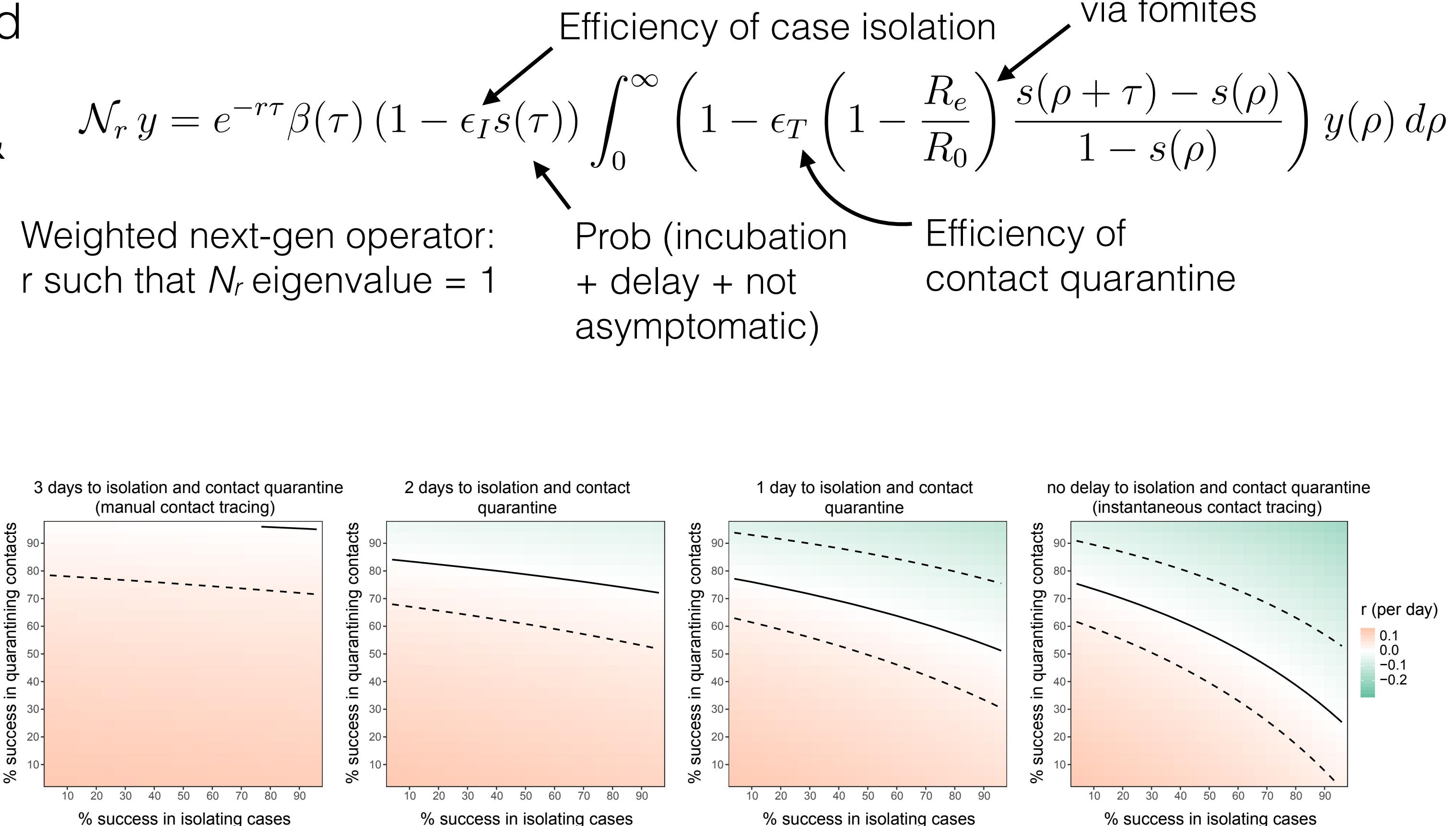
4 Mar 20:30, Christophe: idea encouraged by bioethicists. How fast does contact tracing need to be? Please solve Fraser & Riley 2004 with realistic distributions for  $\beta(\tau)$  and  $s(\tau)$  (both  $e^{-\tau}$  in original)

5 Mar ~03:00: Luca solves it  
numerical eigenfunction determination

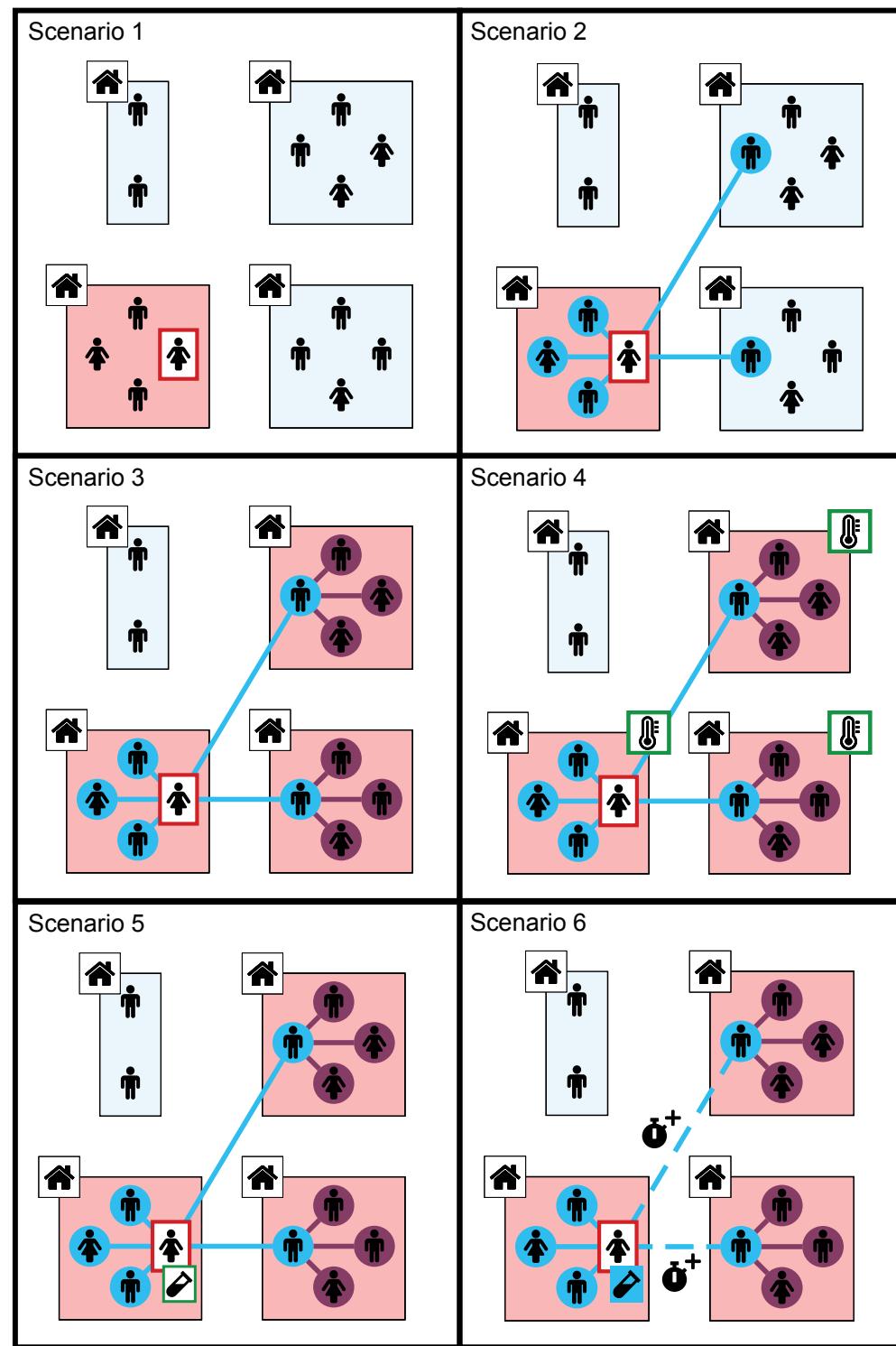
5 Mar: results presented to UK Chief Scientific Advisor + team

6 Mar: paper made public (GitHub)

7 Mar: start of work with NHSX



# 2020 Mar - Oct



Mar: started our agent-based model (Hinch & Probert et al 2021) →  
Fraser et al Risk algorithm.  
First meeting with Google.  
Ferretti & Wymant et al published.

← Apr: Hinch et al Report to NHSX

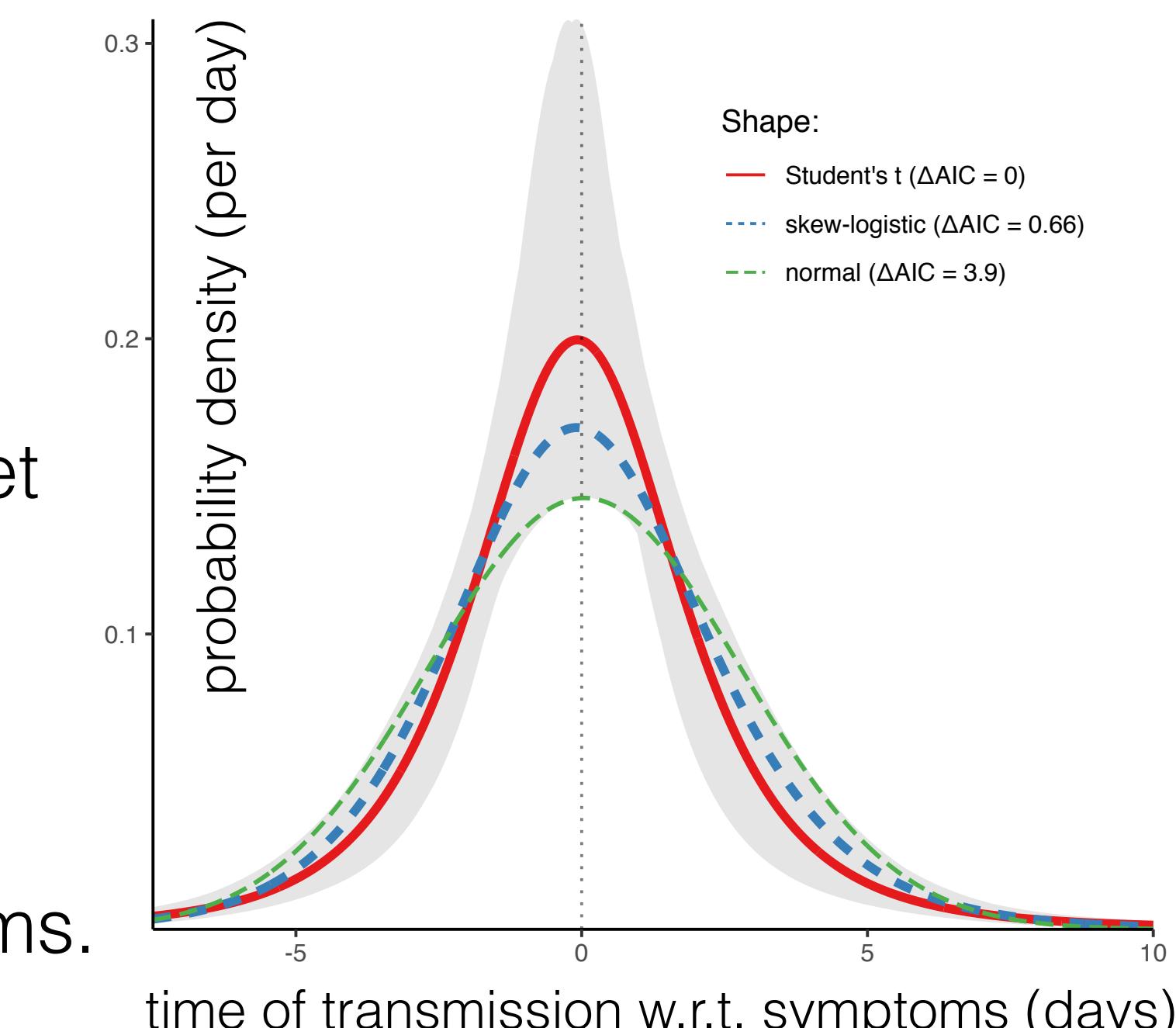
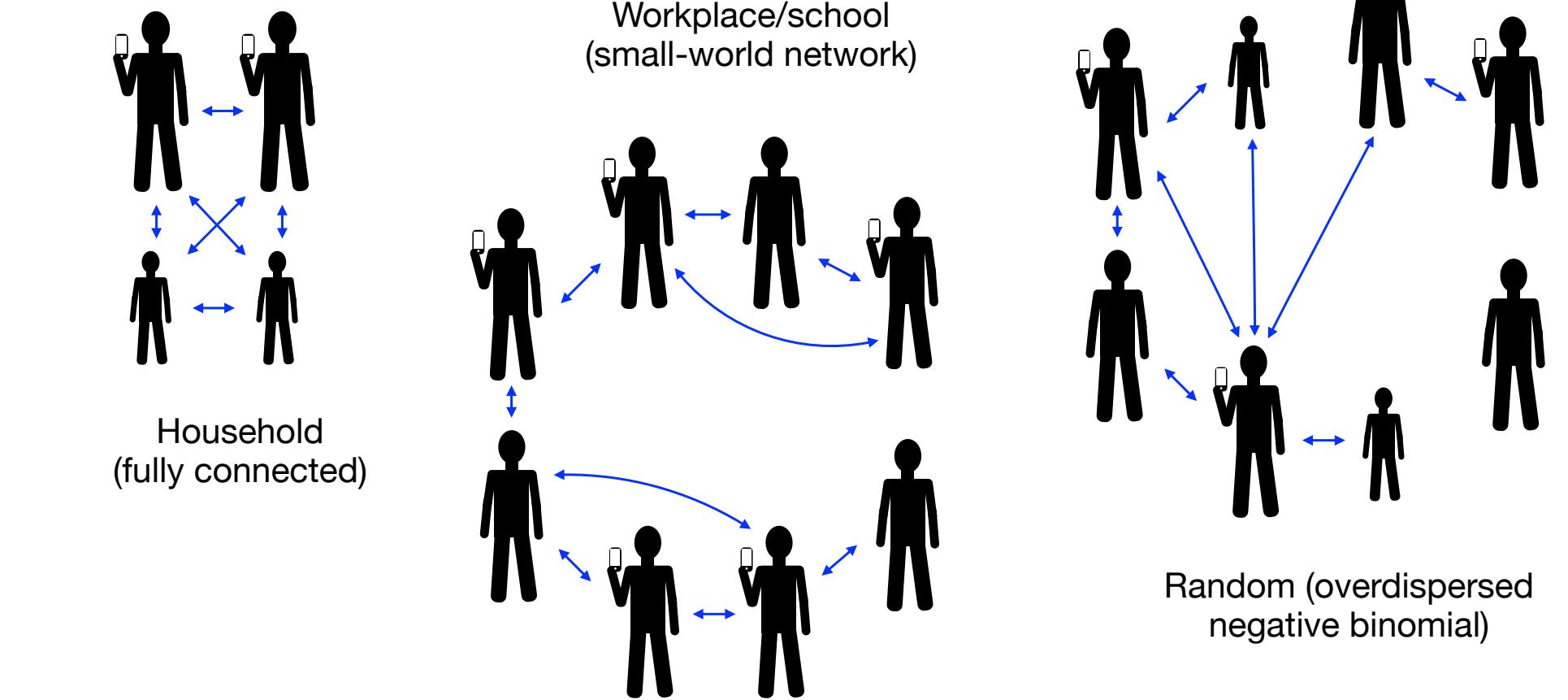
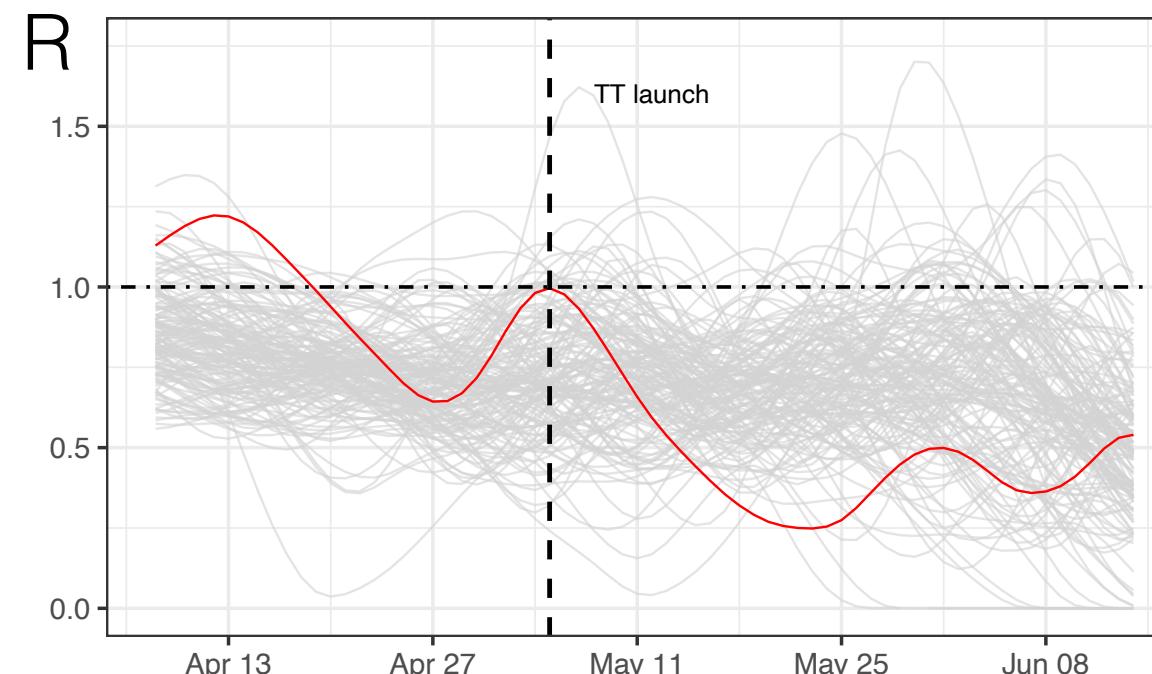
May: Pilot of NHS app v1 on Isle of Wight.  
Parker et al J. Med. Ethics.

Sep: Ferretti et al medrxiv →

Oct: Kendall et al Lancet DH

Other models: Kucharski et al (June) Kretzschmar et al (August) + ~10 preprints

Variety of national digital tracing programmes deployed (49+ countries) after Apple and Google embed functionality in their mobile operating systems.



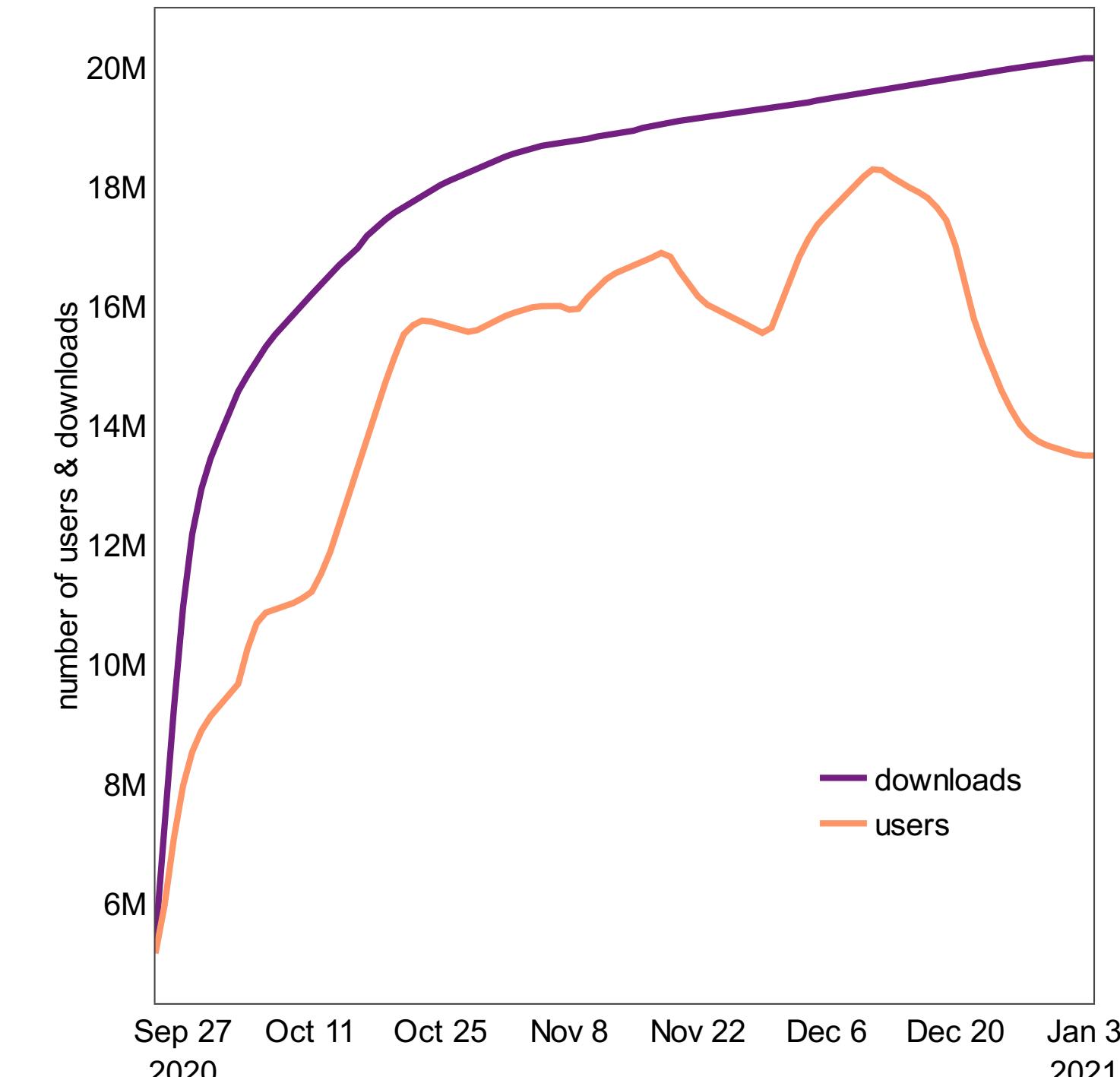
# 2020 Sep 24



Photo: @DavidMcClelland



Photo: NHS COVID-19 App



Counts (millions):  
Eng & Wales: 59m  
Aged 16+: 48m  
Eligible phone: 34m  
Downloads: 21m  
Active users: 16m

# 2020 Oct - Dec

Brief: estimate the epidemiological impact of the NHS app.

Observational study - a population-level intervention - no experiment.

Public data: cases etc. by day and lower-tier local authority (LTLA;  
338 in England and Wales).

Non-public data: app use ('uptake') by day and postcode district.

Idea 1: epidemia (Flaxman et al 2020).

Likelihood:

$P(R(t)) \sim \text{logistic-transformed random walk} + \text{effect of interventions}$

$I(t) = \text{renewal-equation-transformed } R(t)$

$P(\text{observations}) \sim \text{ascertainment} \times \text{NegBin}(\text{delay-transformed } I(t),$   
 $\text{overdispersion})$

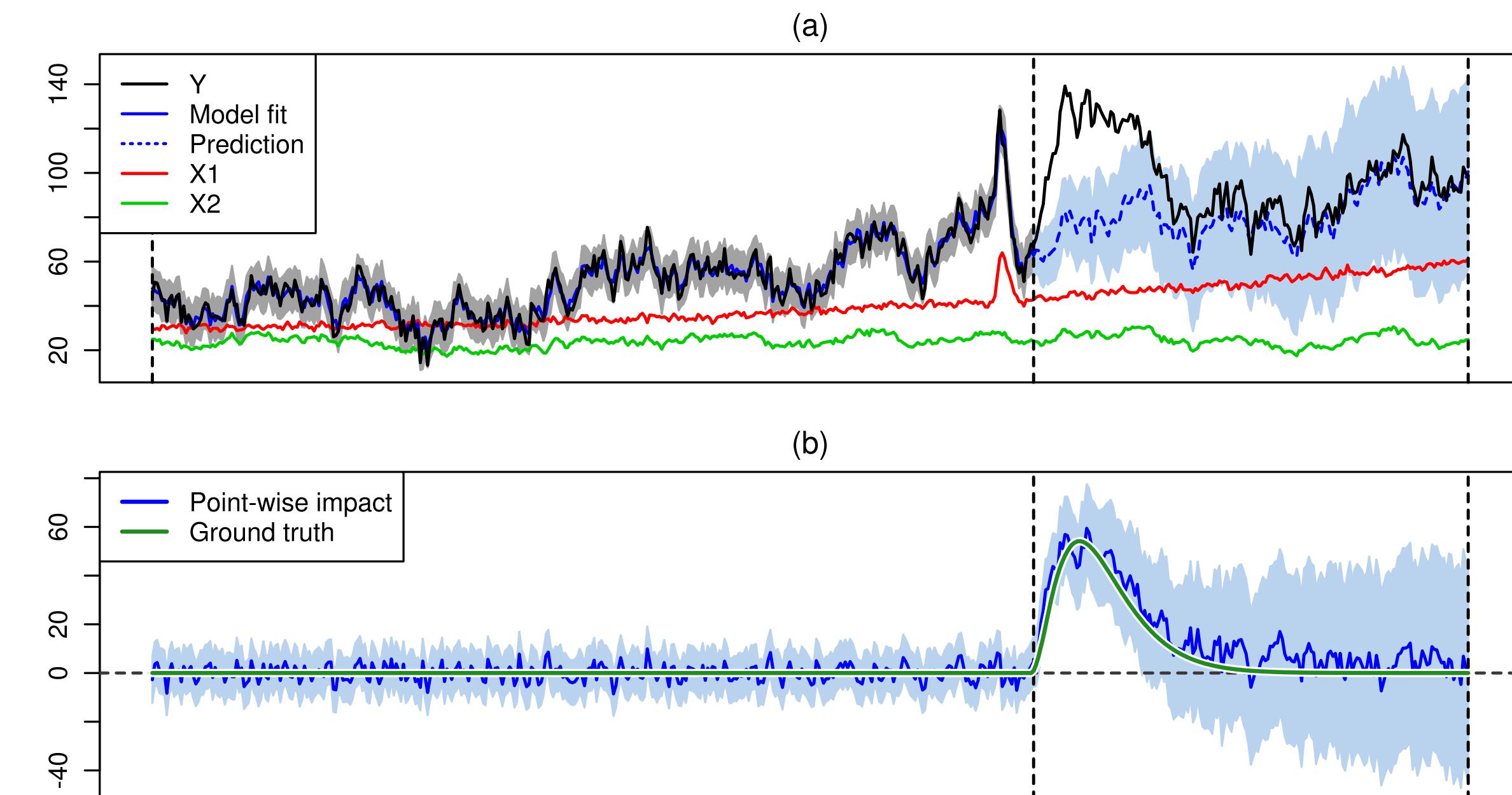
Wrapper around rstanarm.

Problem: not expected to scale to 338 LTLAs (linked by app).

(Epidemia generative code on request.)

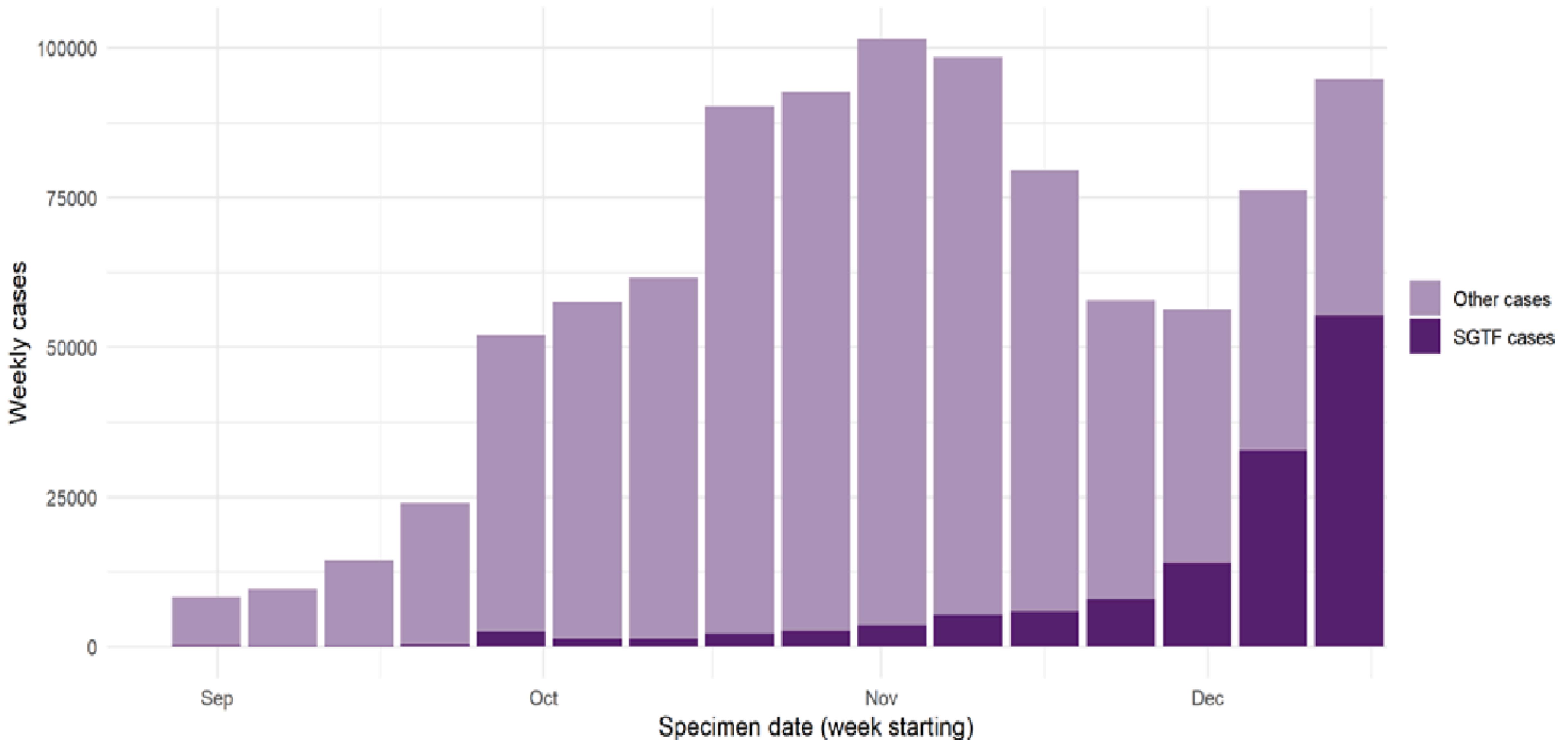
# 2020 Oct - Dec

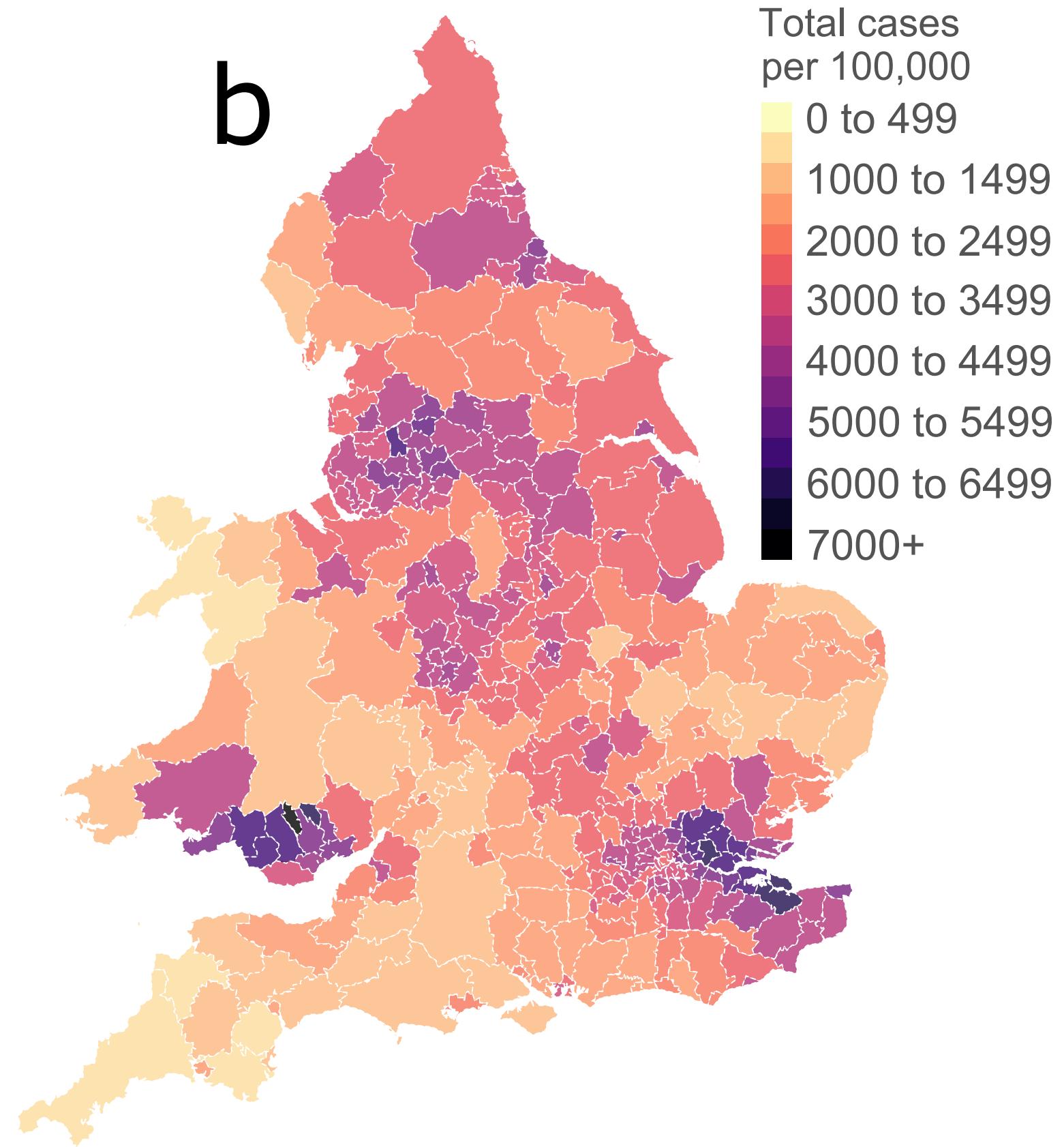
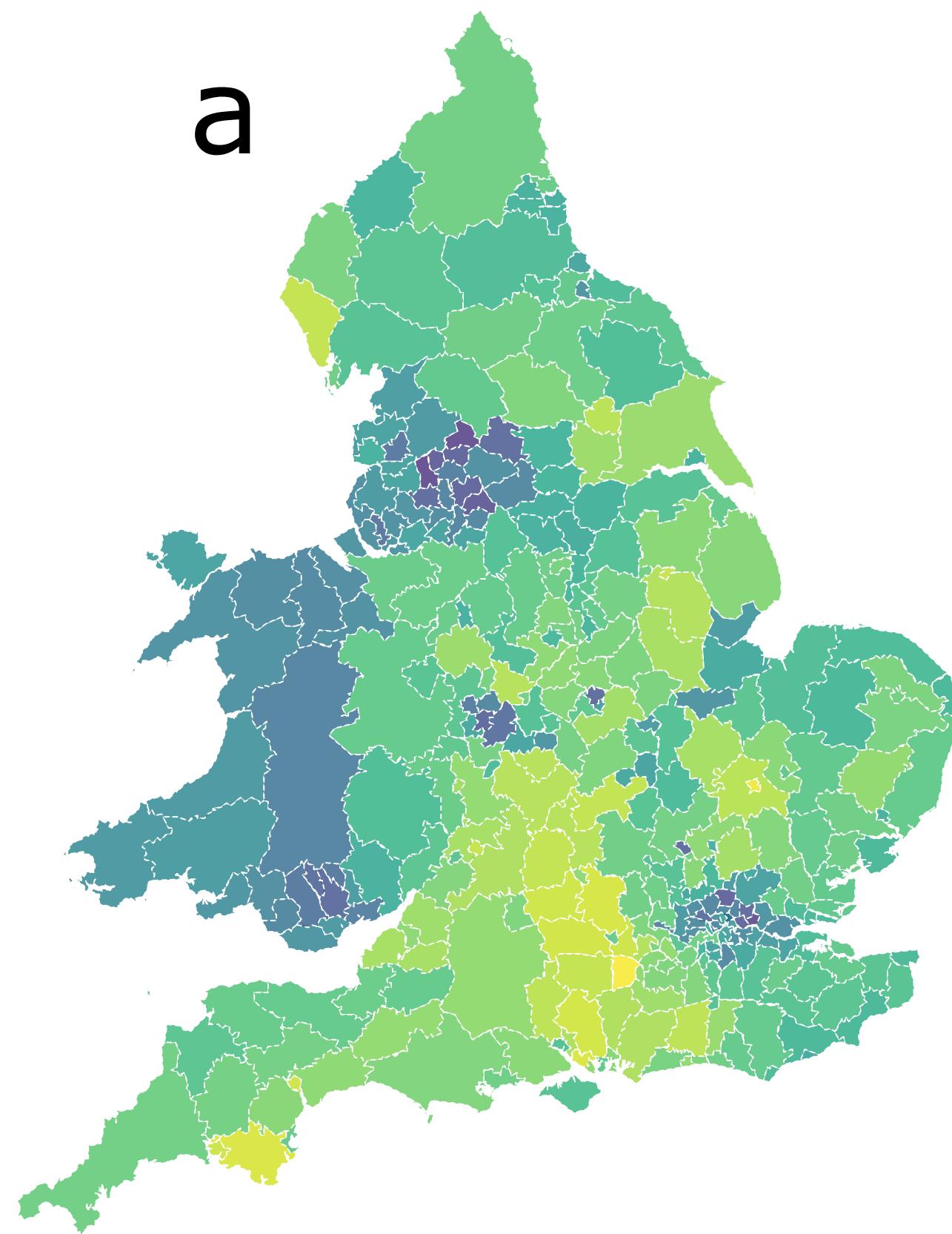
Idea 2: CausalImpact (Brodersen et al 2015) →  
Simple wrapper around bsts (Scott & Varian 2013):  
use Bayesian structural time series to predict a  
*treated* time series as a sum of other *untreated*  
time series in the counterfactual of no treatment.  
Factual - counterfactual = causal impact.  
Problem: all LTLAs were treated.



Idea 3: Synthetic controls, SCs (Abadie & Gardeazabal 2003, Abadie 2021)  
Similar, but predict using a weighted mean of untreated series, constraining  
weights  $w_i$  to sum to 1.  
Could define the SC's uptake, U, as  $\sum_i (w_i U_i)$   
Difference between actual U and the SC's U = treatment intensity  
Difference between actual epi curve and the SC's epi curve = outcome  
Outcome related to treatment intensity?  
Problem: found only one paper on SCs with continuous rather than binary  
treatment (Powell 2015), not appropriate here.

2020 Dec 13



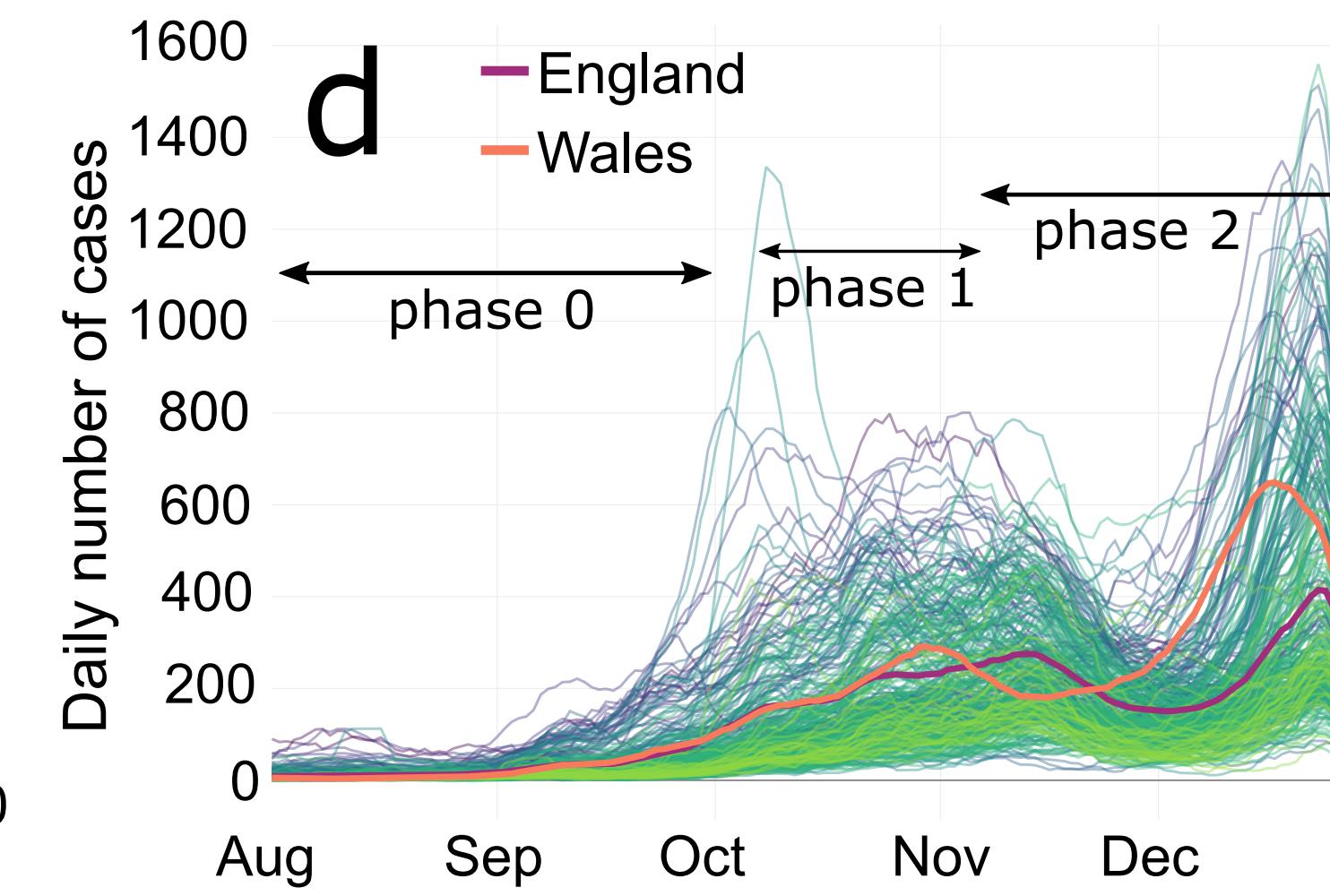
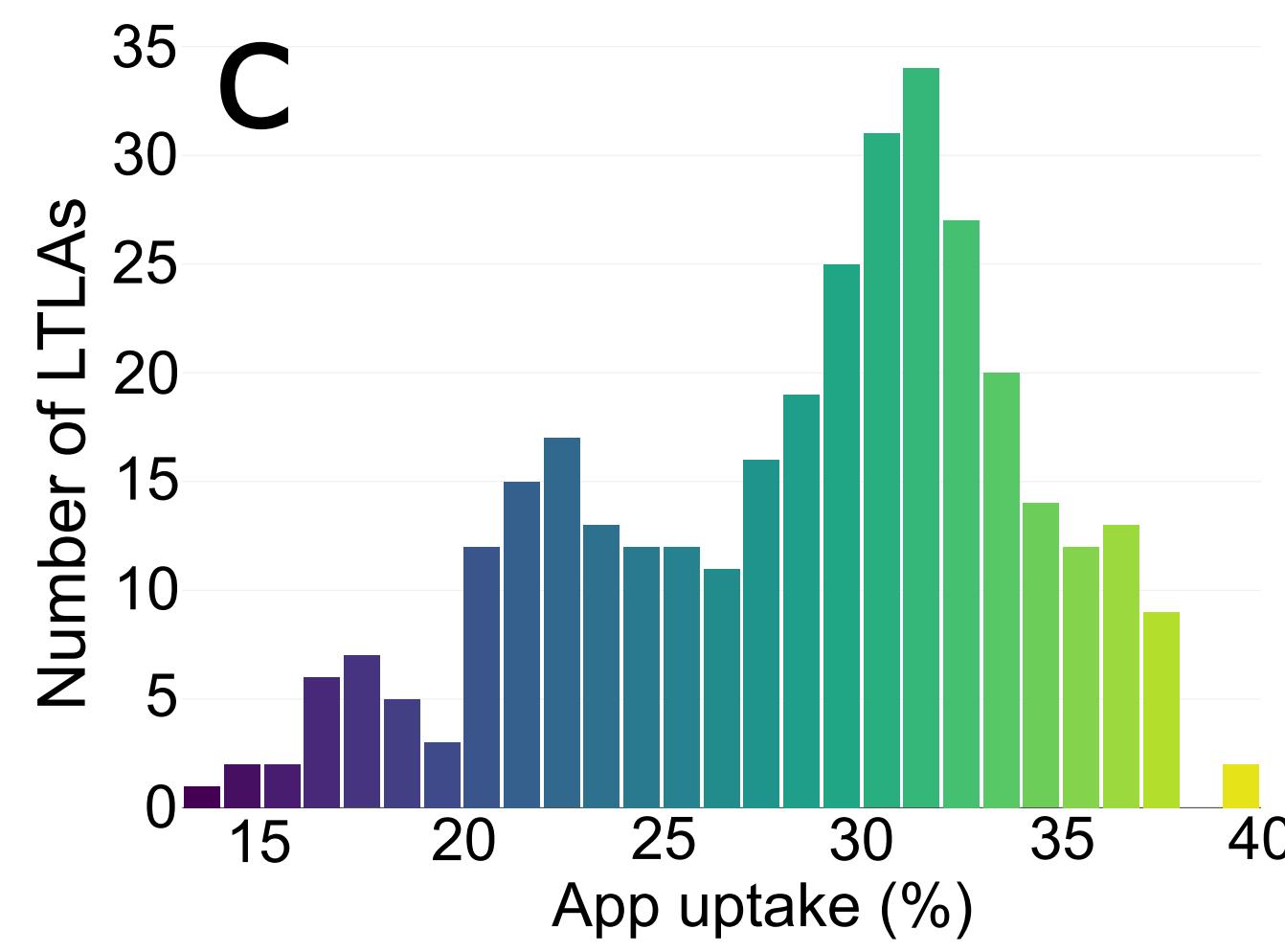
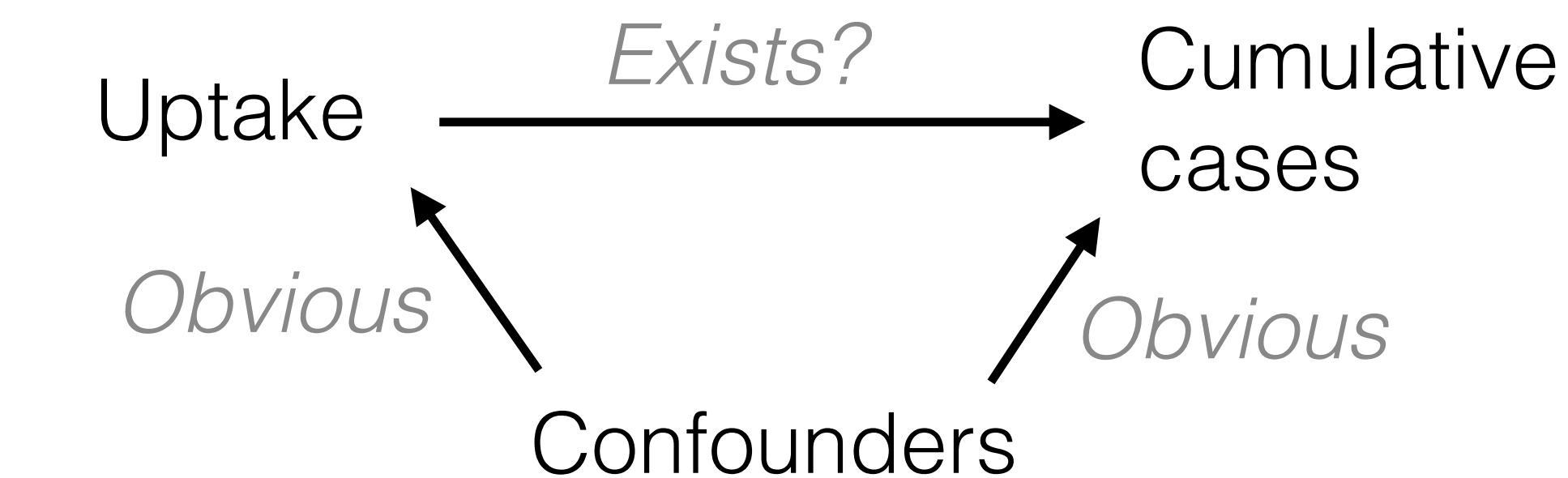


Simplify:  $\text{uptake}(\text{time}, \text{space}) \rightarrow \text{uptake}(\text{space})$

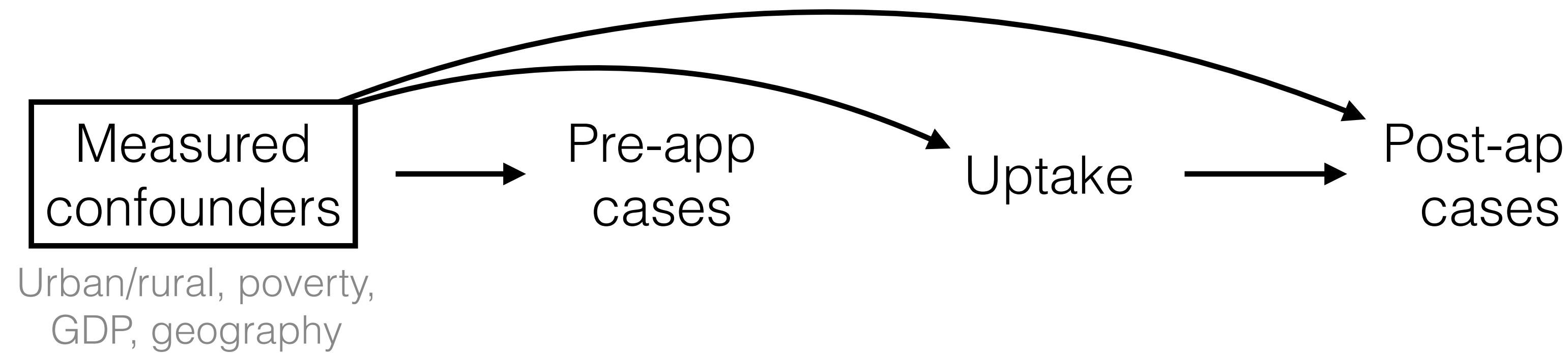
High-uptake LTAs typically have low (per-capita) incidence throughout

Simplify:  $\text{cases}(\text{time}, \text{space}) \rightarrow \text{cumulative-cases}(\text{space})$

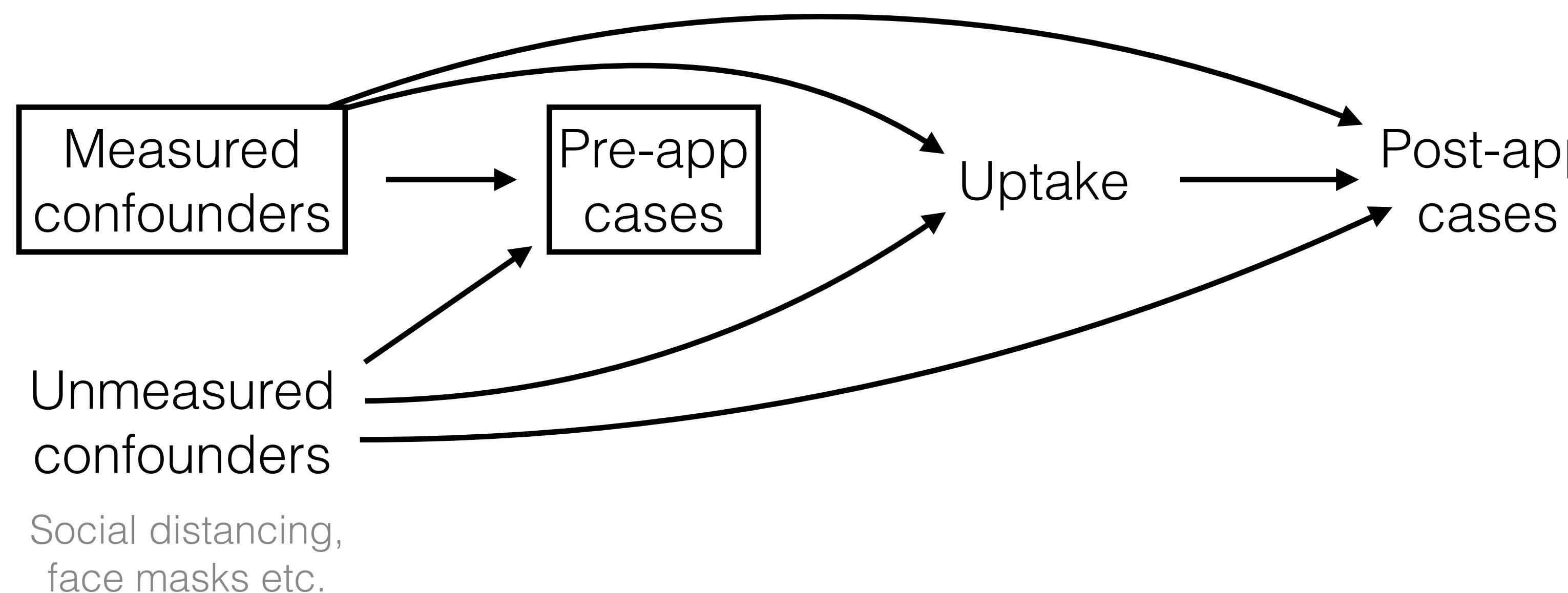
Causal influences:



# Placebo regression



Model 1:  
Pre-app cases  $\perp\!\!\!\perp$  Uptake |  
Measured confounders  $\times$



Model 2:  
Pre-app cases  $\perp\!\!\!\perp$  Uptake |  
Measured confounders  $\checkmark$

Time flows from left to right

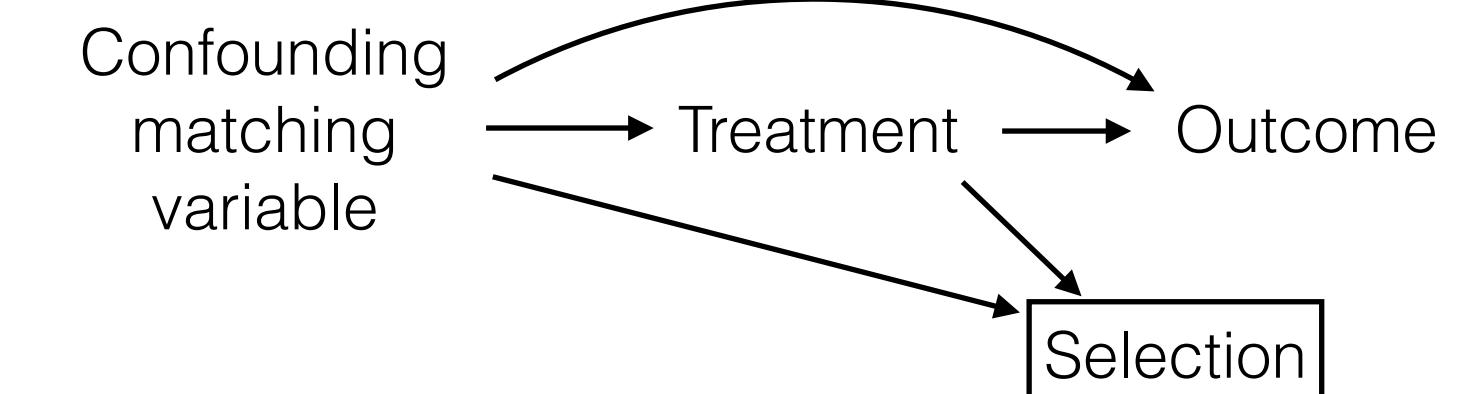
→ = causal influence

$\boxed{X}$  = variable X is adjusted for

" $X \perp\!\!\!\perp Y | Z$ " means X is conditionally independent of Y given Z

"Pre-app  $\rightarrow$  post-app cases" effect suppressed for clarity.

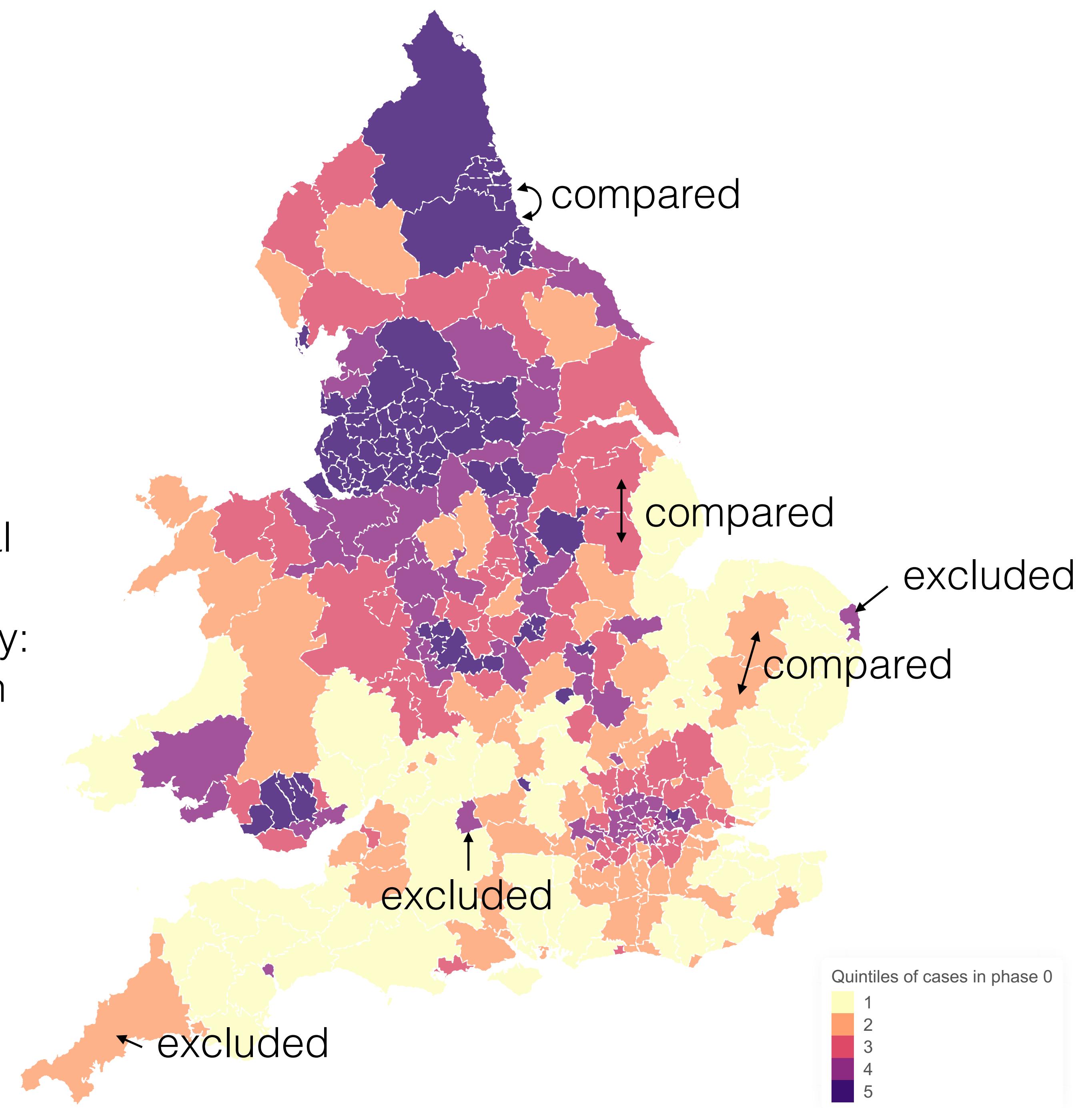
c.f. matched cohort study: boxed collider induces cancellation



# Adjusting for confounders

Urban/rural index, poverty index, local GDP:  
modelled as linear effects on  $\log(\text{cumulative cases})$

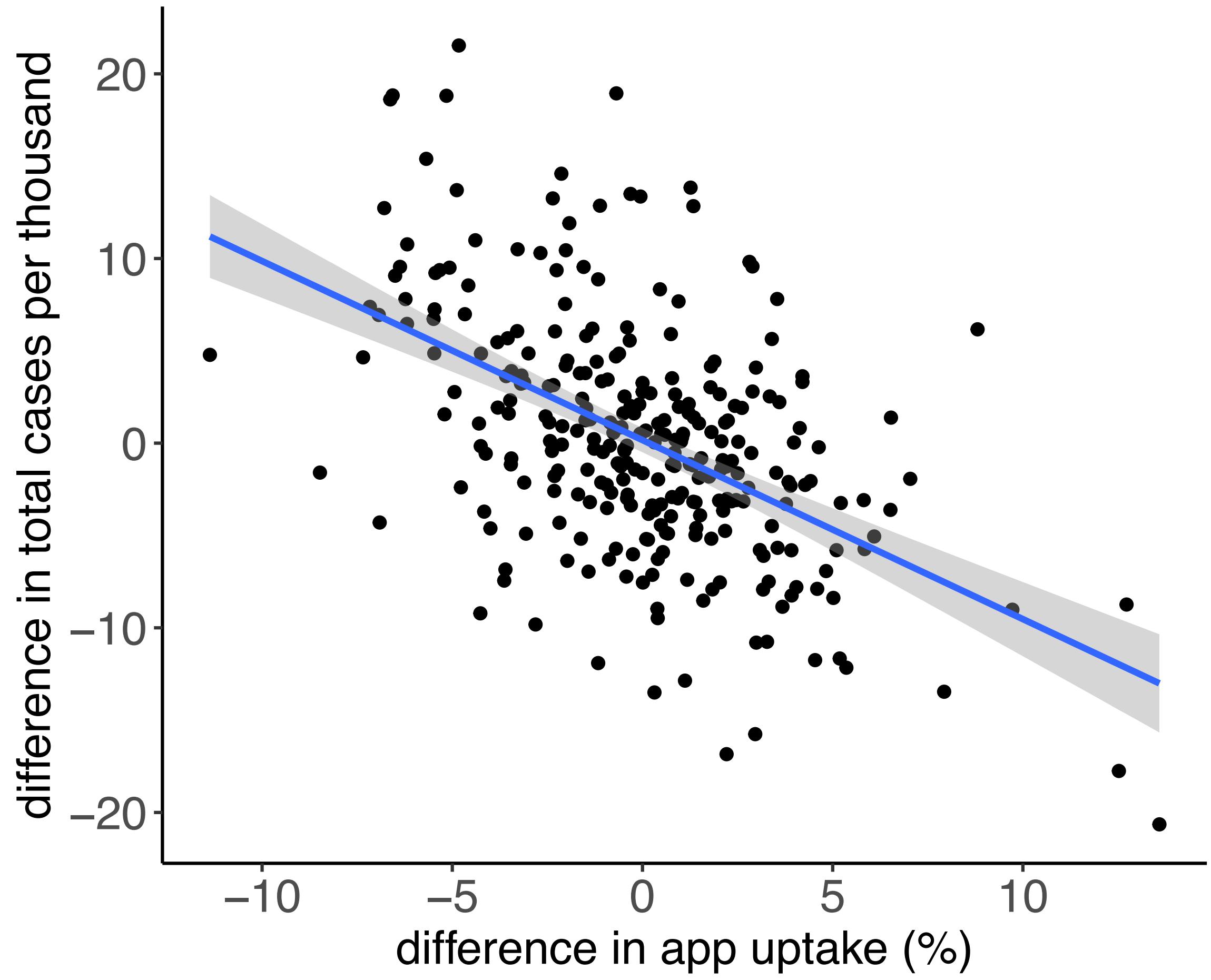
Geography, pre-app cumulative cases:  
stratification/matching c.f. Hernan & Robins  
2015 “Using Big Data to Emulate a Target Trial  
When a Randomized Trial Is Not Available”.  
Analyse subsets of the data with comparability:  
only pairs of LTLAs that are neighbours and in  
the same quintile for pre-app cumulative  
incidence.



$$\Delta \log(\text{cumulative cases}) = \\ \beta_{\text{rural}} \Delta \text{rural} + \beta_{\text{GDP}} \Delta \text{GDP} + \\ \beta_{\text{poverty}} \Delta \text{poverty} + \beta_{\text{uptake}} \Delta \text{uptake} + \varepsilon$$

$$\varepsilon \sim N(0, \sigma^2)$$

$\Delta$  denotes difference between focal LTLA & matched neighbours



(Unadjusted by rural, GDP, poverty)

# Modelling estimate: method

Cases averted = sum time t, LTLA x [product of:

1. number of individuals traced at t, x
2. fraction of those infected
3. fraction of R preventable by quarantine
4. quarantine effectiveness
5. size of transmission chain started at t, x]



(1) x (2) = number  
of infected  
individuals traced

(3) x (4) =  
fractional reduction  
in R for one traced  
infected individual

(3) x (4) x (5) = fractional  
reduction in the size of  
the whole transmission  
chain started by one  
traced infected individual

Assume no saturation, that LTLAs are independent, and that the counterfactual differs only in not having the app.

Let  $t_g$  = generation time in days. First assume all transmissions occur exactly at  $t_g$ . Number of cases resulting from one case at t:

- cases( $t + t_g$ ) / cases(t) in 1st generation,
- cases( $t + 2t_g$ ) / cases(t) in 2nd generation, etc.

Generations blur - they're not all exactly at  $t_g$  - therefore attribute

- cases( $t'$ ) / ( $t_g$  cases(t))
- cases at every later time  $t'$  to each case at t. Sum over  $t'$ .

# Modelling estimate: data

Cases averted = sum<sub>time t, LTLA x</sub> [product of:

1. number of individuals traced at t, x
2. fraction of those infected
3. fraction of R preventable by quarantine
4. quarantine effectiveness
5. size of transmission chain started at t, x]

Reported cases  
by LTLA by day

61% = mean of:  
Smith et al (11% perfect, 54% imperfect,  
35% no adherence to quarantine) and  
Fancourt et al (80%, 8%, 12%),  
assuming perfect quarantine is 100%  
effective and imperfect is 50%

1.7 million notifications total  
(From 410k cases triggering tracing: 4.2 traced  
contacts per index. c.f. manual tracing: 1.8, 1.2 of  
whom in the same household.)

6% “Secondary attack rate” SAR

Sub-analysis required

- similar to manual tracing, excluding ‘direct’ contacts, and to friends / school / work SAR
- Number infected likely twice, from case ascertainment

Estimated impact in first three months:

- several hundred thousand cases averted
  - statistical model estimate larger: probably contains residual confounding, but also indirect benefits
  - c.f. 410k index cases triggering tracing: ~1 case averted each (from whole onward chain, i.e. not  $R \rightarrow R-1$ ).
  - c.f. actual number of cases - 1.9 million - i.e. 15-25% averted.  
Abeug et al 2021: 20% averted from 30% uptake in 5.5 months.
- several thousand deaths averted (via CFR then: 1.5%).

SAR among individuals notified by the app		6%	
Cases and deaths averted in phases 1 and 2:		Cases	Deaths
From modelling of digital tracing		284,000 (108,000–450,000)	4,200 (1,600–6,600)
From matched-neighbours regression		594,000 (317,000–914,000)	8,700 (4,700–13,500)
Per cent reduction in cases for every percentage point increase in app use			
<b>Main analysis</b>		Phase 1	Phase 2
Modelling		0.33 (0.13–0.49)	0.93 (0.46–1.24)
Matched-neighbours regression		1.09 (0.04–2.14) (bootstrap: 0.15–2.16)	2.66 (1.75–3.56) (bootstrap: 0.80–4.71)
<b>Secondary analyses</b>		Phase 1	Phase 2
Stratified linear regression in clusters <sup>a</sup>		-1.05 (-2.08 to -0.04)	3.34 (2.53–4.14)
Matched pairs regression <sup>a</sup>		5.08 (1.77–8.40)	3.89 (1.05–6.74)
Matched-pairs regression adjusted for local efficiency of manual contact tracing <sup>a</sup>		4.49 (0.21–8.77)	3.11 (-0.14–6.35)
			3.67 (0.31–7.02)

The effect is measured as the per cent reduction in cases for every percentage point increase in app use, from different analyses. Ranges shown are 95% confidence intervals for regressions, and a sensitivity analysis exploring 2.5–97.5% of the distribution of outcomes for modelling.

1-2 % reduction in cumulative cases per percentage point increase in app uptake.

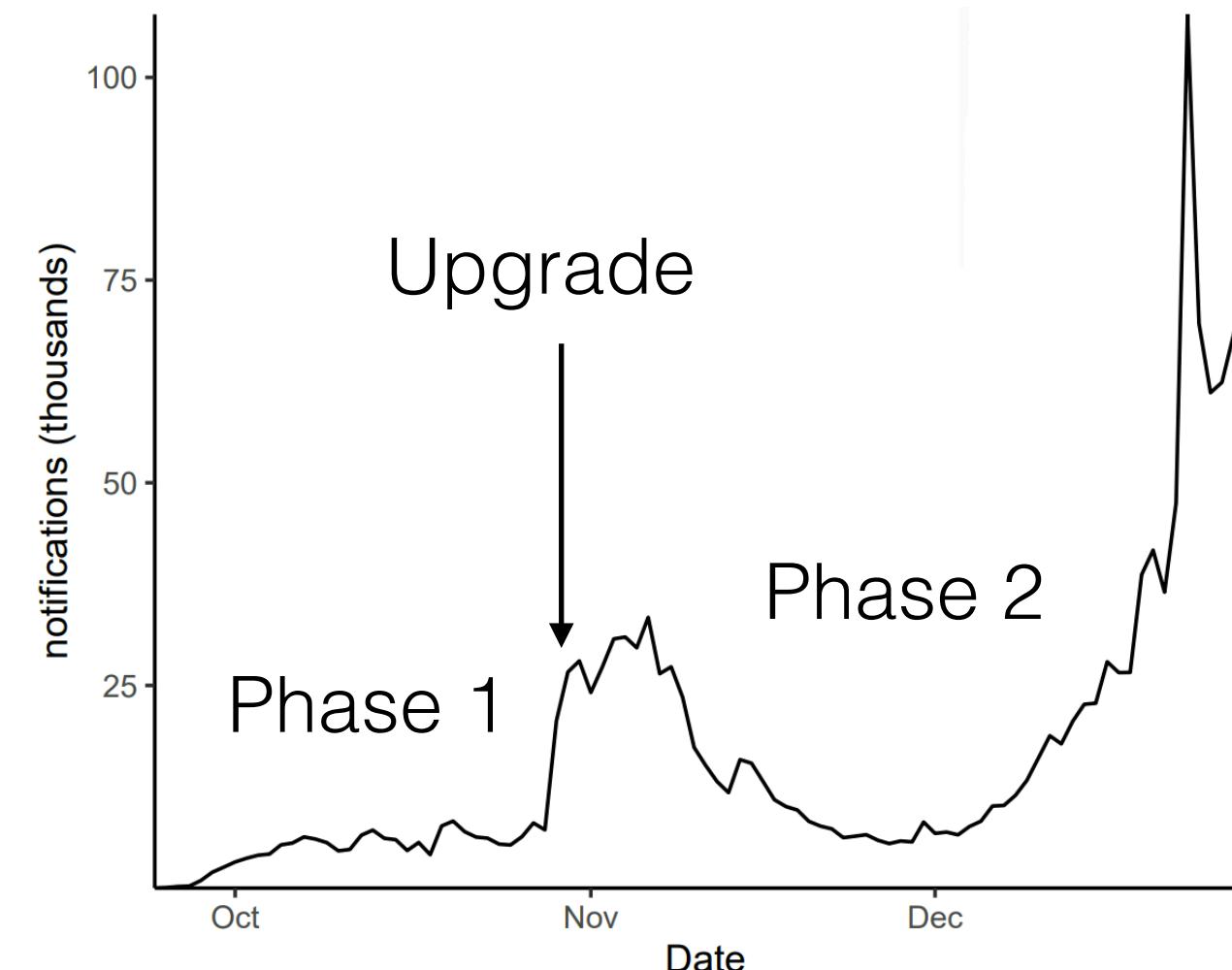
### Robustness checks:

Bootstrapping non-redundant LTLA differences (x vs y but not y vs x, two of three edges of a triangle)

Alternative matchings for comparability of LTLAs: in clusters, and in pairs with very similar pre-app cases (not just in same quintile).

<b>SAR among individuals notified by the app</b>			<b>6%</b>
<b>Cases and deaths averted in phases 1 and 2:</b>		<b>Cases</b>	<b>Deaths</b>
	<b>From modelling of digital tracing</b>	284,000 (108,000–450,000)	4,200 (1,600–6,600)
	<b>From matched-neighbours regression</b>	594,000 (317,000–914,000)	8,700 (4,700–13,500)
	<b>Per cent reduction in cases for every percentage point increase in app use</b>		
<b>Main analysis</b>	<b>Phase 1</b>	<b>Phase 2</b>	<b>Overall</b>
Modelling	0.33 (0.13–0.49)	0.93 (0.46–1.24)	0.79 (0.37–1.10)
Matched-neighbours regression	1.09 (0.04–2.14) (bootstrap: 0.15–2.16)	2.66 (1.75–3.56) (bootstrap: 0.80–4.71)	2.26 (1.50–3.00) (bootstrap: 1.60–3.19)
<b>Secondary analyses</b>	<b>Phase 1</b>	<b>Phase 2</b>	<b>Overall</b>
Stratified linear regression in clusters <sup>a</sup>	-1.05 (-2.08 to -0.04)	3.34 (2.53–4.14)	2.76 (2.16–3.35)
Matched pairs regression <sup>a</sup>	5.08 (1.77–8.40)	3.89 (1.05–6.74)	4.39 (1.70–7.08)
Matched-pairs regression adjusted for local efficiency of manual contact tracing <sup>a</sup>	4.49 (0.21–8.77)	3.11 (-0.14–6.35)	3.67 (0.31–7.02)

The effect is measured as the per cent reduction in cases for every percentage point increase in app use, from different analyses. Ranges shown are 95% confidence intervals for regressions, and a sensitivity analysis exploring 2.5–97.5% of the distribution of outcomes for modelling.



Separating phases 1 and 2: major upgrade to app's risk scoring algorithm (Bluetooth Low-Energy signal data → proximity with unscented Kalman smoother, Lovett et al 2020). Increased exposure notifications per index case by a factor of 2.5. Estimated app effect size increased a factor 2.4.

<b>SAR among individuals notified by the app</b>		6%	
<b>Cases and deaths averted in phases 1 and 2:</b>		<b>Cases</b>	<b>Deaths</b>
<b>From modelling of digital tracing</b>		284,000 (108,000–450,000)	4,200 (1,600–6,600)
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Press release

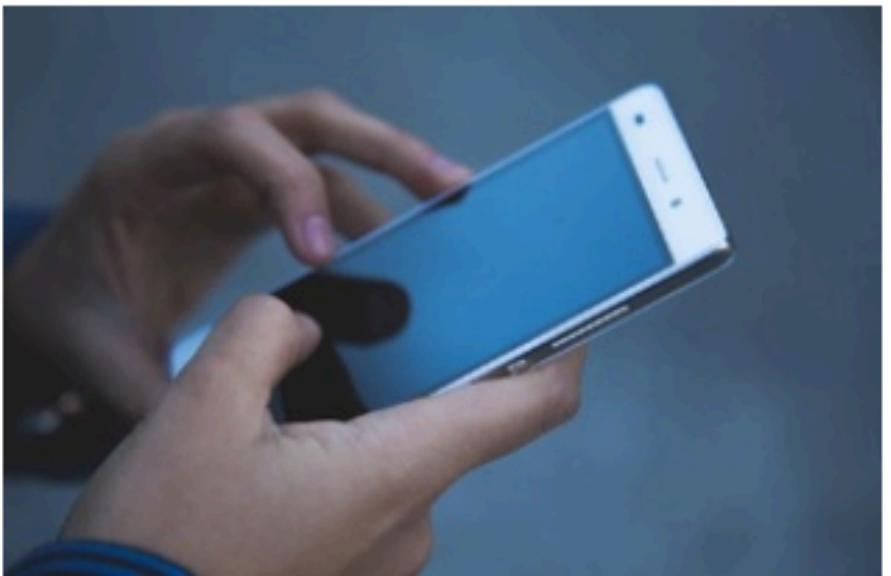
## NHS COVID-19 app alerts 1.7 million contacts to stop spread of COVID-19

Over 1.7 million users have been asked to isolate as a result of a close contact since launch, helping to break chains of transmission.

From: [Department of Health and Social Care](#)

Published 9 February 2021

Last updated 12 February 2021 — [See all updates](#)



- Analysis suggests approximately 600,000 cases have been prevented by the app since September
- The app notifies contacts as quickly as 15 minutes after a user inputs a positive test result

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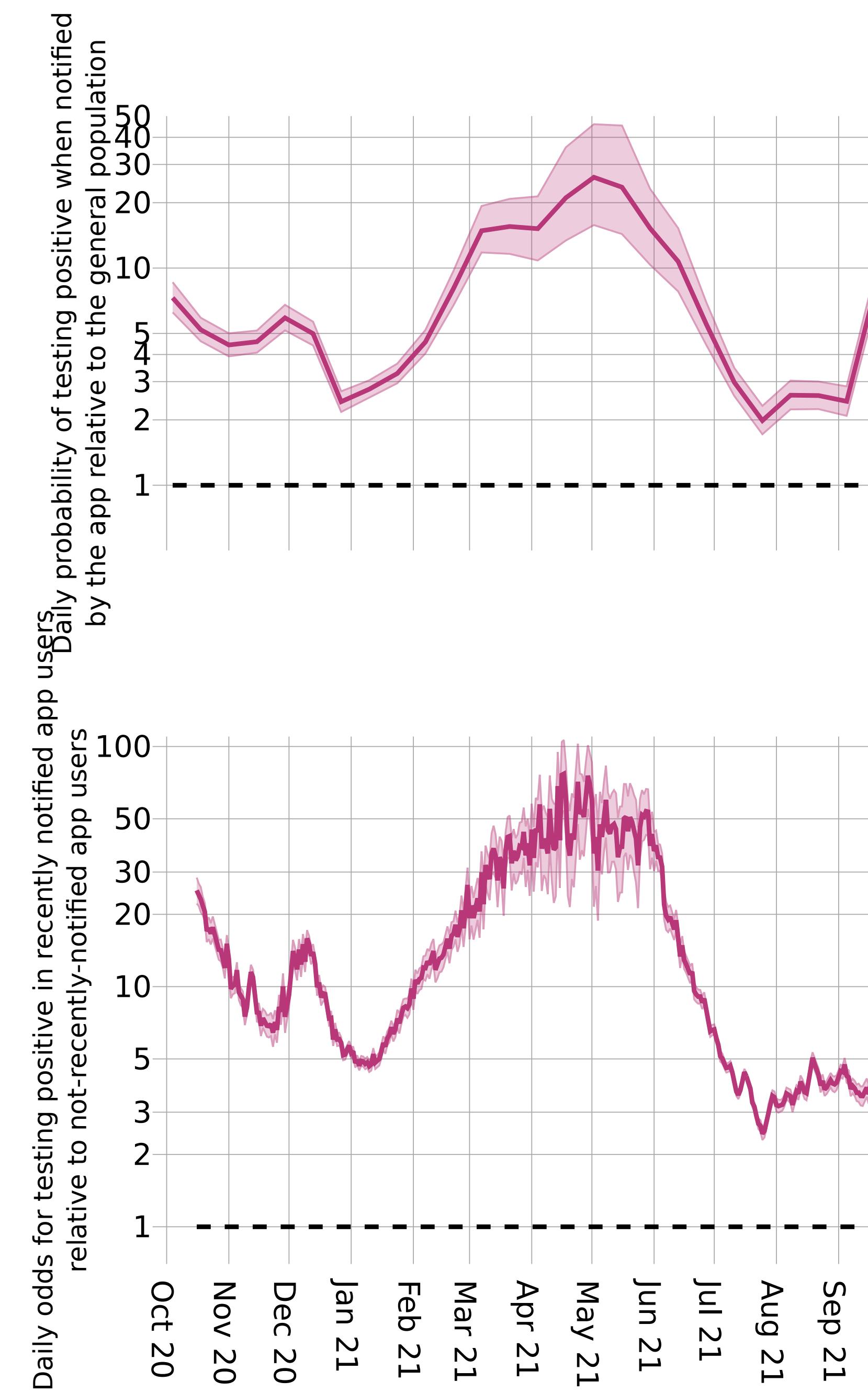
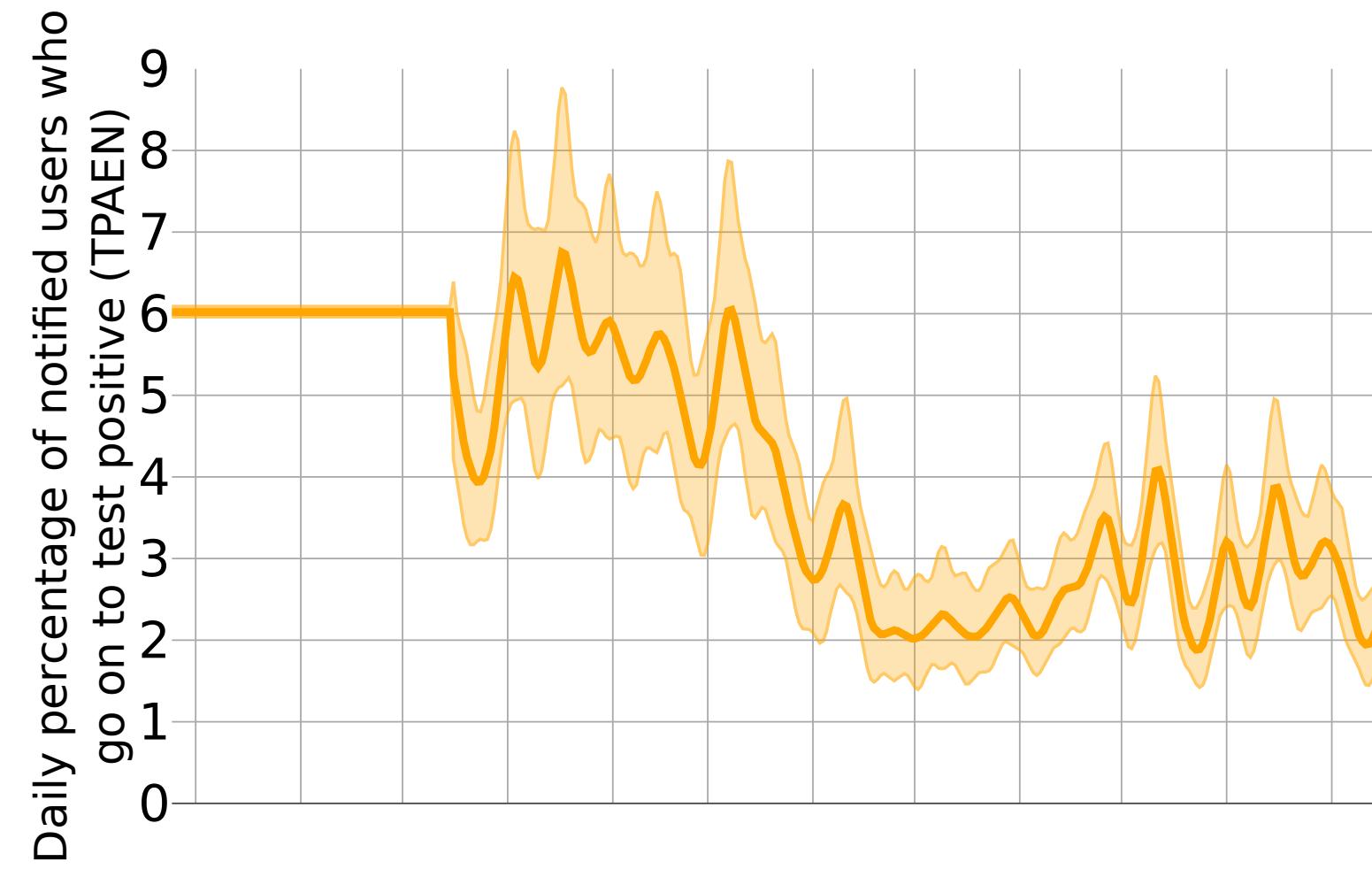
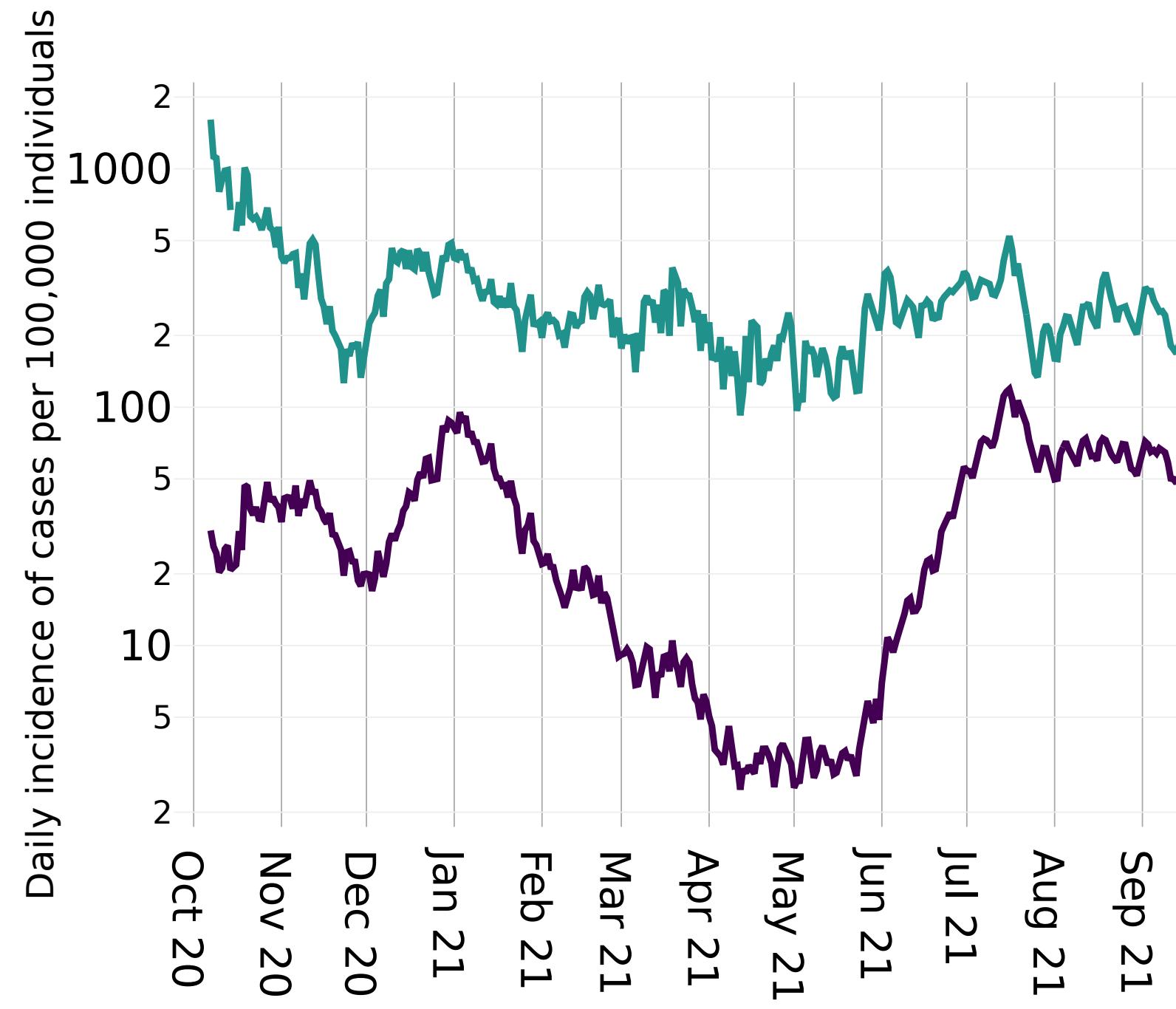
## smartphone app

### U.K. Study Shows Power of Digital Contact Tracing for COVID-19

Posted on May 25th, 2021 by Dr. Francis Collins



Credit: Adapted from Getty Image and Wymant C, Nature (2021)



Thanks to:  
app developers, app users,  
co-authors, funders, and you!

## Quantifying SARS-CoV-2 transmission suggests epidemic control with digital contact tracing

Luca Ferretti<sup>1\*</sup>, Chris Wymant<sup>1\*</sup>, Michelle Kendall<sup>1</sup>, Lele Zhao<sup>1</sup>, Anel Nurtay<sup>1</sup>, Lucie Abeler-Dörner<sup>1</sup>,  
Michael Parker<sup>2</sup>, David Bonsall<sup>1,3†</sup>, Christophe Fraser<sup>1,4‡</sup>

## The epidemiological impact of the NHS COVID-19 app

Chris Wymant<sup>1,7</sup>, Luca Ferretti<sup>1,7</sup>, Daphne Tsallis<sup>2</sup>, Marcos Charalambides<sup>3</sup>,  
Lucie Abeler-Dörner<sup>1</sup>, David Bonsall<sup>1</sup>, Robert Hinch<sup>1</sup>, Michelle Kendall<sup>1,4</sup>, Luke Milsom<sup>5</sup>,  
Matthew Ayres<sup>3</sup>, Chris Holmes<sup>1,3,6</sup>, Mark Briers<sup>3</sup> & Christophe Fraser<sup>1✉</sup>

In prep. authors



Department  
of Health &  
Social Care



[github.com/ChrisHIV/teaching](https://github.com/ChrisHIV/teaching)