xyzskaft • 9 days ago

The previous three comments made me curious. Looking around online I found this interesting and valid critique of the methodology:
https://www.medrxiv.org/con...

Some sort of disclosure: Both myself a and the authors of the work I found are Swedish. Biased or not, the methodology in the paper remains seriously flawed.

Stefan Homburg • 15 days ago

The article ASSUMES that the effective reproduction rate, Rt, is constant before and after interventions. Rt is allowed to change ONLY at the intervention points. This assumption drives the results and is at variance with the data. Using the same method, the authors could confirm a strong influence of sunspots on infections.

Jointly with Christoph Kuhbandner, I have written a formal comment that you can find on the Advance preprint server:

https://advance.sagepub.com...

Reply • Share ›

Ray Pawson • 15 days ago

Every modeler is aware of George Box's famous challenge: 'the question you need to ask is not "Is the model true?" (it never is) but "Is the model good enough for this particular application?"' The COVID-19 crisis has resulted in an epidemic of model building and the outpouring of simulations and extrapolations has had a profound effect on government policy the world over. Model building in all of its various guises relies on 'estimates' of infection parameters and it is the veracity of these assumptions that so vexed Box. Let us examine the two key assumptions built into Flaxman et al: i) 'changes in the reproduction number are an immediate response to interventions rather than gradual change in behaviour', ii) 'individual interventions have a similar impact in different countries and that the efficacy of those interventions remains constant over time'.

These assumptions are plainly wrong. People react to lockdowns in dozens of different ways – they comply, they resist, they grow weary, they change their minds, they seek exceptions. For example, lockdown adherence varies: i) according to whether it relies on policing or persuasion, ii) with the degree of political, media and popular support for the measure, iii) on externalities as when protesters enraged at the George Floyd killing ignore the rules on large social gatherings, iv) on national temperament, which varies from high compliance to deep division, and above all, v) over time with resolve weakening as lack of contact hits home.

The same fluctuations apply to all NPIs. Nudge intervention are notoriously short lived – how many people still wash hands to the double rendition of 'happy birthday'? Guidance is generally disputed even in the single community – reopening schools is interpreted either as an unwarranted risk (amongst those whose fear of infection persists), or a blessing (amongst those who perceive the threat has diminished). Finally, there is the 'free rider' problem, the disharmony provoked by people who believe their circumstances render them exceptions to the rules. These include government advisors and mathematical biologists.

1 Reply • Share ›

Nicholas Lewis • 16 days ago

I have posted a detailed critique of the paper at my website: https://www.nicholaslewis.o...

I have run the model both with the authors' base assumptions and after

making slight changes to them. I find that the attribution of the reduction in transmission between different intervention measures is highly sensitive to the

precise assumtions made, particularly as to the delay between infection and death.

Further, the Bayesian prior probability distributions used by the authors for the parameters representing the estimated effects of each intervention are extremely strongly biased in favour of finding that almost all the interventions have a negligible effect, with almost the entire reduction in transmission being due to one, possibly two, types of intervention.

Moreover, the authors make the highly unrealistic assumpption that each country's population is homogeneous as regards both susceptibility to COVID-19 and infectivity. Moreover, they ignore other non-intervention factors that are highly likely to have slowed transmission of the virus, such as unforced changes in individual behaviour in response to the epidemic and seasonal factors. In combination these result in their model being bound to estimate (a) that almost the entire reduction in transmission was due to government interventions and (b) that deaths would have been very high in the absence of the interventions.

As a result of these shortcomings, the findings in the paper are unsound and scientifically worthless.

Chris Horton ↗ Nicholas Lewis • 15 days ago • edited

Interesting as it is to pick over the dynamics of R, might that be both overcomplicating matters and looking in the wrong place? A circular argument must surely have but a single point of failure, from which everything else unravelled. It shouldn't be so hard to spot, nor too hard to repair.

Doubting the susceptibility and infectivity of populations, as per Lewis (2020), would seem to be getting closer, with its challenge to the unstated a-priori assumption that the Population Fatality Ratio must be the known Infection Fatality Ratio. A glance at Extended Data Fig 5 and perhaps we are there: assume a PFR, rather than the IFR, and re-fix the epidemiological parameters with a fatality ratio 10x lower (from distributions around 1% to around 0.1%). This step might re-align the counterfactual with Sweden, and relieve the model from the stress of its contradictions.

With the fatality rate re-fixed, it could no longer be assumed that R responds only to interventions. The model might instead assume that, with no intervention, R reflects a gradual approach to herd immunity. The model could then estimate the effect of each non-pharmaceutical

intervention, in each country, on holding infections short of that eventuality, and where continued intervention should be considered to avert a renewal of transmission.

Chris Horton • 17 days ago • edited

The article appears to present a somewhat circular argument. By first fixing a certain fatality ratio, then assuming that R changes only step-wise from an intervention, and also finding that lockdown is the only intervention having much effect on R, the model estimates that lockdowns drove R step-wise below 1 and averted a certain number of fatalities in each of the 11 European countries modelled. From this it is concluded that lockdowns had a large effect on R, and that continued intervention must be considered to keep R under control.

Nothing wrong with a circular argument, except there is little to stop doubts spreading from a single uncertainty until nothing is certain. For instance: in Extended Data Table 1, it is uncertain whether observed deaths in Sweden should fit the model or the counterfactual, when Fig 1 for Sweden models a step change

in R as if it had locked down, yet Extended Data Fig 4 confirms Sweden as the 1 country of 11 which did not lock down.

What effects might this have on the assumptions and/or conclusions?

Reply • Share ›

Giuseppe Rotondo • 21 days ago

Dear Authors
Death is such a variable parameter that cannot be used to build an universal model. Death regard a specific target of people i.e elderly with chronic pathologies. In Italy depended in elderly hospices contagions and so on..

Reply • Share ›

willem engel • 25 days ago

1. correlation vs causality, it assumes without proving
2. big title always suspicious
3. The DATE! look at the that 30 march but with data from May? HIGHLY suspicious

Reply • Share ›

Sebastian Rosemann • 25 days ago

Dear authors,

this study is an innovative approach using confirmed deaths as apparent manifestation of the pandemic.

But one major point is not clearly pointed out:
How valid are the modelled numbers compared to observations.
How does the non-uniform distribution of infections among age groups (that is observed in reality) affect the authors validation of their models?

Some details on this would be nice as otherwise all the numbers in this study are not provable by any real observation and therefore nothing more than imagined. The explanation of how the age specific attack rates are calculated is hardly explained and very intransparent in the appendix with a lot of links and papers hard to find (e.g. Walker, P., Whittaker, C. & Ghani, A. The Potential Impact of COVID-19 in Lower-and Middle- Income Countries.), i gave up to understand it. Same for usage and consequences for the rates in terms of the model.

As stated by the authors, the models are validated against results of e.g. antibody-studies in some countries:
„While there have still been few reliable national serological studies, initial small scale surveys in Austria and Denmark are closely aligned with our estimates.
A much larger study in Spain is very closely aligned with our estimates. These

initial results, to some extent, validate our choice of infection fatality rate.“

These studies show different infection rates between age groups (see below).
So when a fatality rate is observed, this is a rate distorted by the age distribution of infections.
A simple comparison against the overall fatality rate seems not correct in most cases.

How do the authors deal with the age-distributions of real infections?
What is the exact measure to validate their modelled numbers against those studies?
Is there something like an age distribution deriveable from the models?

Some remarks to age distributions:

Spain:
https://www.ciencia.gob.es/...
~ 6,9% seroprevalence in age group 70-74 vs. ~ 5% overall

Belgium:
https://www.medrxiv.org/con...
~15% seroprevalence in the age-group 90+ vs. ~ 6% overall

Denmark:
https://www.medrxiv.org/con...
Only ages 17-69,
~ 2.3% seroprevalence in the age group 60-69 vs. 1.8% overall (17-69 years)

Austria:
http://www.statistik.at/web...
states that 0.15% of the austrian population could have been infected.
The only value that would roughly match the 0.76% in the authors paper would be the prevalence of 0.7% in risk areas (which is probably not all of Austria).

GeorgeDance ➚ Sebastian Rosemann • 25 days ago

"How do the authors deal with the age-distributions of real infections?"
I'm still getting into the new IC paper, but I'm at that point now. Using each country's demographics, they come up with a different IFR for each one. It's admittedly a gloss, as it doesn't take into account regional variations; but it is something they try to account for, not something they ignore (though of course it has been ignored completely in the media accounts - outside the stuff on Lockdown Sceptics, I haven't seen any media discussing the paper's methodology).

Arturo López • a month ago

A simple SIR model with constant parameters through the whole outbreak nicely reproduces the daily death rate in several cases, such as Spain as a whole, Madrid or New York. The key words are "constant" and "daily death rate". Constant, because there is no need at all to modify the rate of contacts in coincidence with the social distancing measures to fit the data. (Occam's razor: if a simple model with constant parameters fits the data, why should one assume that the contact rate changed in between?) Daily death rate because this is the only reliable piece of data: number of infections or infection rate are science-fiction and any model based upon them is suspect to me. If confinement was so effective, just by inspection of the daily death curves one should see a change of tendency in coincidence with the most extreme confinement measures, but this does not happen anywhere. The outbreak stopped because "the virus run out of susceptible people" due to the rampant rate of contagion (R0 as big as 7 or 8). I conclude the evidence in favor of the IC views is weak. They should explain first why nowhere in the world, with or without distancing measures, the infected share is greater than 20%. They should also explain the results of de Diamond Princess (less than 20% infected), to begin with.

Dr Gareth Davies (Gruff) ↗ Arturo López • a month ago

I don't agree a simple model with fixed R and CFR fits the data. Not even for a single location like Spain. Do you have a link to such a model?

Arturo López ↗ Dr Gareth Davies (Gruff) • 25 days ago

It's my model. Find herebelow the parameters for Madrid. The only reliable data, in my opinion, are daily deaths. Infections or infections per day are just science-fiction and I disregarded them.

number of effective contacts per infected person and day (beta) = 3,5415
rate of recovery (gamma) = 0,0586 / day
Madrid population = 6,7 x 10E6
Initial number of infected (february 1st) = 2,26E-02
Initial fraction of susceptible people, So = 11,3%
Lethality rate = 1,207%
As a consequence, Ro = 6,82

The model is sensitive only to the product beta x So; it yields essentially the same results if one assumes a double lethality and half as many susceptible people. I do not see any reason why 100% of the population must be allocated to the susceptible box. It seems

to be so nowhere in the world.

For Spain as a whole:
number of effective contacts per infected person and day (beta) = 7,416
rate of recovery (gamma) = 0,043 / day
Spain population = 4,5 x 10E7
Initial number of infected (february 1st) = 8,18E-02
Initial fraction of susceptible people, So = 5,0%
Lethality rate = 1,367%
As a consequence, Ro = 8,62

I made a fit to daily deaths rate from feb 1st until mid may. Free parameters are beta, gamma, lethality rate and initial number of infected.

Check by yourself.

1 ▲ | ▼ • Reply • Share ›

Dr Gareth Davies (Gruff) ➚ Arturo López • 25 days ago

Also if you Google "ESI Epidemic Severity Index" you'll see another technical paper on the technique I devised for this. I'd be interested to hear your views.

▲ | ▼ • Reply • Share ›

Dr Gareth Davies (Gruff) ➚ Arturo López • 25 days ago

Thanks. I'll take a look. I agree detected infection numbers are meaningless. I've only modelled with fatalities and reported recoveries. The latter whilst less reliable than fatalities is still a good metric in early days since these come from hospital discharges.
You may find our study interesting (see link in my comment below).

▲ | ▼ • Reply • Share ›

Dr Gareth Davies (Gruff) • a month ago • edited

From the paper:
"Our model relies on fixed estimates of some epidemiological parameters such as the [1] onset to death distribution, [2] the infection fatality rate..."

(Assumption numbering mine)
1) onset to death is not a constant - there's plenty of data to trivially demonstrate this - this assumption is invalid.
2) IFR is certainly not a constant and varies dramatically by location and most

crucially by latitude as we have shown. This is also trivially demonstrated by considering the ratio of fatalities to recoveries which is an effective proxy for Hospital FR which is related to IFR by a fixed factor. More precise hospital data can be used to confirm this.

The two most fundamental assumptions underlying this model are demonstrably false.

Reply • Share ›

Joerg Stoye • a month ago

For Germany, the paper gives a counterfactual *population* death rate of .68% (credible set up to .93%) by 5/4 and therefore a cumulative prevalence of 68%, and possibly 93%, by 4/20 or so. Does it sound plausible that Germany would have reached herd immunity and possibly asymptotic cumulative burden in that time frame? I tried but failed to understand the counterfactual's exact computation.

Reply • Share ›

Pragmatist_in_another_guise • a month ago • edited

Did each of the many authors truly contribute to the analysis and its writing up? Given timescale for production of this work I doubt that practicable.

It would be far better, indeed less misleading, to distinguish the names of those doing the donkey work from others whose contribution was providing data and/or glancing over the penultimate draft.

Of course, the press and general public will be impressed by the authorship because to them it's obvious the more collaborators there be the greater the verity of that written (an extension of the fallacy of 'democracy').

A nit to pick is: when did mathematics and computation become 'science'? Perhaps the authors don't make claim to being scientists but the press and government are under that illusion.

This is not intended to demean what they have done, there are plenty other grounds for that. Rather, it's a question of epistemological accuracy.

'Science' proceeds via the logic of refutation: predictions not borne out send a chain of (potential) falsification upwards to the originating theory/hypothesis. Mathematics is wholly deductive; its validation rests with peers in the field agreeing that the premises are not self-contradictory, and with the validity of each step in deduction; conformity with some external reality is irrelevant.

If the authors are engaging in 'science' then they have completed only the first stage of the process. Their model(s) loosely equate to theories/hypotheses. At best, they have attempted partial validation of their findings by reference to very recent measurable (with caveats) outcomes. Some of their predictions were notably off-track but this doesn't get mention.

To proceed with 'science' they could do the following. Refine their models (and assumptions) in light of what's recorded to have happened a few months down the line. Then re-run the model(s) and compare outcomes with observations. Keep adjusting the models (in somewhat atheoretic manner) until best possible predictions against current data are obtained. The next step is where scientific method emerges: test the model(s) in context of the next pandemic of comparable nature.

The upshot from the above is that everything produced by this team is currently speculative. It may be of great interest but nobody in their right mind would place these investigators 'at point' in leading a public health response. They may have valuable qualitative insights to offer but those ought be filtered through field-epidemiologists and public health professionals before being put in front of politicians. Seemingly, the Department of Health, and considerable expertise nationally and internationally the department can call upon, was sidelined when snake-oil was on offer. Perhaps somebody from the Department would care to tell me why I am under a misapprehension?

2 ^ | v • Reply • Share ›

Andrew Shardlow • a month ago

Why bother with R?. Rather like a broken clock, we only really know it when it is 1. We don't have enough information to squeeze in time. A sensible approach is to focus on daily growth. Daily growth was tumbling down early in the course of the pandemic in each country studied and this fall pre-dates any NPI. There is no clear NPI impact on this, though there are two significant and curious yet consistent discontinuities seen in all daily growth factor vs. normalized mortality plots, including that from Sweden where there has been no lockdown. Sweden has a lower mortality per million than Spain, Italy, the UK and France. Your findings seem rather fanciful and there is insufficient detail in your paper to test the validity of a complex mathematical model in software in which there are probably many assumptions. The near straight lines segments in daily growth vs. death mean the time series data can be very precisely modelled simply by two logistic segments. View – uploads.disquscdn.com The lockdowns may even have increased mortality.

View – uploads.disquscdn.com View – uploads.disquscdn.com

2 ^ | v • Reply • Share ›

claude • a month ago

Maybe of interest, since these publications show modeling of nonpharmaceutical measures, too:
https://science.sciencemag....
https://www.medrxiv.org/con...

Reply • Share ›

Jeremy Hooper • a month ago

The modelling in this paper seems to stretch the bounds of reality and is not in line with previous modelling. The modelling undertaken by Imperial under the leadership of Prof. Fergusson suggested there were be around 550,000 deaths in the UK over a 2 year period if there was no mitigation strategies. This modelling assumed a death rate for Covid19 infections of around 1%, which is borne out by the current numbers of deaths and ONS estimates of actual infections.
The authors of this modelling suggest that there would have been around 500,000 deaths in the UK by the middle of May this year with the upper confidence interval suggesting it could have been as high as 600,000. This would suggest either a very much higher death rate than expected or that the entire population would have been infected in around a month. Neither seem likely in reality.
Further the modelling does not take account of the excess deaths which are non Covid-19 related which currently is at least 14,000. With more to come in the years ahead as the NHS attempts to get back to normal.
Its a shame the authors chose a sensationalist approach rather than looking to develop a balanced approach looking at the impact of Covid-19 infections and the measures taken to reduce the spread of the virus. Looking at Years of Life Lost approaches rather than number of deaths would be better and more balanced in its approach.

1 Reply • Share ›

Pragmatist_in_another_guise ↗ Jeremy Hooper • a month ago

Spot on, just considering deaths attributable to Covid-19 takes no account of morbidity and mortality arising from control measures like lock-down and from subsequent economic malaise.

Indeed, years of life lost analyses would shed valuable light.
Unfortunately, mishandling by the government and gleeful talking up of disaster by news media has led to an emotional approach wherein each death is an equal and, supposedly, avoidable tragedy.

Reply • Share ›

Maurizio Rainisio • a month ago

If the lockdown would have been as afficacious as claimed, lifting it (in Italy now

for longer than 4 weeks) would have caused some kind of visible effect.
The Generalized Logistic that I fit to the Lombardy data, did not change a bit after early May. R2=0.999. View — uploads.disquscdn.com

1 • Reply • Share ›

Pragmatist_in_another_guise ↗ Maurizio Rainisio • a month ago

That is a chart wondrous to behold.

They key point is of simple statistical summary of the data revealing much the same as complicated 'black box' and 'take on trust' attempts mechanistically to model disease progression.

1 • Reply • Share ›

JeiPi. • a month ago

The authors wrote that "Most interventions were implemented in rapid succession in many countries, and as such it is difficult to disentangle individual effect sizes of each intervention. " jet they claim that only lockdown has an effect.

3 • Reply • Share ›

Pragmatist_in_another_guise ↗ JeiPi. • a month ago

Boris Johnson picked his snake-oil salesmen and now we all have to live with the consequences.

• Reply • Share ›

Nicholas Lewis • a month ago

A major issue with the authors' approach is that other factors than non-pharmaceutical interventions (NPI) may account for most of the reductions in Rt. Obvious such factors are: unforced changes in behaviour, growth of herd immunity (particularly in large cities and other locations with a high Rt), and seasonal factors. Many NPI were adopted by all the countries studied. However, Lockdown - which the authors estimated to be far and away the most effective NPI (Figure 2) and the only individual NPI with identifiable effects - was not implemented in one country, Sweden. Therefore, Sweden is the only country studied for which the confounding effects of other non-NPI factors can, at least to some extent, be avoided.

In the original version of this study (Imperial College COVID-19 Response Team - Report 13), published on 30 March, the same day that the authors' manuscript was submitted to Nature, the date on which public events were banned on Sweden (12 March 2020) was correctly reflected in their plotted results, and after the final NPI of school closures some days later their central estimate for Rt was 2.6, with a 2.5% lower bound of 1.5. In the study's model, interventions have an immediate, step effect on Rt, so no subsequent further change in Rt was

projected. The implication of this is that deaths in Sweden would continue to rise strongly, as they were projected to do in the short (early April) projection period of Report 13, until the epidemic engulfed most of the population. As we now know, that didn't happen; after mid-April Swedish COVID-19 deaths ceased to increase.

In the revised version of this study published in Nature, Rt for Sweden (Extended Data Fig. 1) is estimated to behave very differently, falling from 2.6 to 0.7 on 29 March 2020, with the result that subsequent recorded deaths are reasonably matched by the NPI-based model projections.

What caused this major reduction in estimated Rt on 29 March? Both Extended Data Fig. 1 and 4 show an intervention in Sweden on 29 March, being "Public events banned". But this appears to be just an invention by the Imperial College authors. Public events were actually banned from 12 March, as correctly stated in Supplementary Table 2 and the reference cited therein, and in the original (Report 13) version of the study.

As a result of inventing an imaginary Swedish NPI on 29 March, the authors' model projects a time profile of Swedish COVID-19 deaths that matches quite well the incidence of actual deaths (Extended Data Fig. 1). If the ban on public events were moved back to its true date, 12 March, the match between the time-profiles of projected and recorded deaths would be much poorer.

Even if the authors instead attributed the large modelled reduction in Rt to the previous NPI reflected in Extended Data Fig. 1, being 'Social distancing encouraged' on 16 March (the unmarked 'School closure ordered' NPI on 18 March had no evident effect) , would not solve this problem.

Altering the date of an NPI from its previous, correct, value with the result that the model projections well match recorded deaths in Sweden in itself destroys the credibility of the study. There are other very serious flaws in this paper, but the alteration of data casts doubt over the validity of the whole study. If Nature values its reputation, it would in my view be wise to insist on a retraction of this paper.

1 ^ | v • Reply • Share ›

Nicholas Lewis ➚ Nicholas Lewis • 16 days ago

Since posting the above comment I have realised that although the Timeline of Interventions (Supplementary Table 2) for this paper shows the date of "Public events banned" for Sweden as 12 March 2020, with events of >500 people banned, which is correct, on 29 March 2020 Sweden reduced the size limit of permitted events to 50 people. So there is a partial justification for the authors having treated the public events ban for Sweden as occurring of 29 March even though their own Timeline of

Interventions table only gives the 12 March date and does not mention the modification of the ban made on 29 March.

Reply • Share ›

Arun Thirunavukarasu • a month ago

There are massive issues with this study, and I predict it will be retracted within the year:
a) Assuming that *only* lockdown and no other public health measures affect R is a fallacy.
b) Simulation data is used to argue against real evidence infection rate fell before lockdown.
c) Examples where identical results occurred without lockdown (e.g. Sweden) are ignored.

I'm disappointed that it didn't occur to the researchers or the reviewers how absurd the curves are - do they really believe that infections halted upon lockdown with no prior decline?

4 Reply • Share ›

mosmosmos ↗ Arun Thirunavukarasu • a month ago • edited

Call me cynical, but given that this publication has been across the world media as 'lockdown has saved 3m lives', with very little critical analysis, it almost seems this was designed to get out ahead of anything which would show what an unnecessary action a 'full lockdown' was.

By the time it has been reviewed and retracted, it will barely be mentioned outside of the scientific community.

The data laid out in front of you in the paper just plain contradicts the conclusions drawn.

2 Reply • Share ›

claude ↗ Arun Thirunavukarasu • a month ago

Sweden is a bad example, since of its low testing rate (severely affected patients tested only) and because of its higher excess mortality (see: https://www.euromomo.eu/gra... in comparison to all other sparsely populated Northern European countries such as Norway, Denmark and Finland.

2 Reply • Share ›

Arun Thirunavukarasu ↗ claude • a month ago

Your selective comparison to other nordic countries and not for example, Spain or Italy, is telling - it seems you accept that factors

other than lockdown are more important in the epidemiological trajectory!

1 ^ | v • Reply • Share ›

claude ➚ Arun Thirunavukarasu • a month ago • edited

I'm sorry, I've been too quick to answer first. Yes, I agree, that the "complete lockdown" doesn't seem to be most important measure. Population density probably seems to be one essential parameter as well among banning big events and others. The impression of the Swedish infection process, however, to comparable neighbouring countries seems to hint on less efficient measures being taken. Still, a complete lockdown seems to be one brick in controlling the pandemic outbreak.

^ | v • Reply • Share ›

Arun Thirunavukarasu ➚ claude • a month ago

This study attempts to claim that complete lockdown was the only significant measure reducing infection rate. Since infection rate peaked before lockdown measures in many countries, and Sweden experiences an almost identical rise and fall to other countries, it is obvious to me that these claims are absurd. Just look at the figures in the paper; they look absolutely nothing like reality - do these people truly believe R remained very high until (and only until!) lockdown was announced? Completely ridiculous - I hope this paper will be rescinded soon.

^ | v • Reply • Share ›

Josh Samuel • a month ago

The authors have taken a very interesting approach to an important problem, but is there a risk that the choice of source data and use of piecewise constant modelling for Rt, combine to seriously inflate the impact attributed to full lockdown?

1. Choice of data - consistency across countries: I understand the authors preference for one central source of data, but I'm afraid that ECDC data, whilst held in one place, is not necessarily consistent. There is great variation in whether and when countries have included non-hospital deaths in the reporting, and whether all probable covid deaths are included, or just those with a positive test.

2. Choice of data - reporting delays: More importantly the ECDC data is based on date of report, not date of death. There are therefore significant delays between the date of the actual deaths and the date they are attributed to in the data. For the UK, for example, comparing date of death data to ECDC data, suggests

around a 7 day delay in the ECDC data. It is not clear that the authors have accounted for this. It is also likely that this will vary by country. This will tend to push changes in R to seem later than they were, and thus push them towards the last intervention (usually full lockdown). Have the authors looked at modelling day of death data for hospital setting only? This would seem the "cleanest" data

see more

Reply • Share ›

Marcel Bouvrie • a month ago

Hi, unfortunately I am unable to open the additional links to this study to check the conclusions that are drawn. To my humble opinion it must be a highly speculative outcome, because there are too many (unknown) factors. If herd-immunity could have ran it's natural course, the pandemic possibly would have been halted weeks ago. Now the virus spread is only slowed down. Vulnerable groups are still not safe. My guesses are if we had done a lockdown only on these groups, we would have been much better off. Now we face economic and social damage on a hughe scale, with doubtless more deaths and financial and social psychological problems upfront.

Based on figueres in Brazil, where there is no lockdown present, the current deaths are around 37.000 (09/06/2020) on a population of more than 212million inhabitants with very poor healthcare regarding to Europe. Deaths in Europe are 2 to 3 times higher with a lockdown. (probably due to unhealthier live styles and a more aged population). Or maybe all these figures are also based on specutalive guessing. Counted deaths having covid-19 in their bloodstream does not mean they have died from it, as we have many traces of virusses in our bodies all of the time. It is obvious the virus unmasks underlying diseases to these patients, who probably would have died next flew season in september or of something else.

Where can I find the complete documentation?

2 Reply • Share ›

bert • a month ago

The comment marked as spam, actually put forward, according to me, the valid argument (although rudely stated) that the accuracy of the output (conclusions) of any model depends on the model, but also very much on the accuracy of the data put into the model. I believe all scientists would agree with this?
I have some other arguments that would imply that the conclusions of the model used are far from accurate because the data put into the model are inaccurate:
1. Nobody knows how many people are actually infected by the new corona virus.This because relatively few people have been tested because they had none or little symptoms of the disease. This implies that the relation between corona virus infected people and infection fatality rate is just inaccurate. It could be very

inaccurate!
2. Nobody knows which "lock-down measure" influenced infection rate.
3. In Sweden, they didn't do the corona measures. People dying because of the virus in Sweden is the same as in other countries.
Pls don't say this is a SPAM

2 ^ | v • Reply • Share ›

Pragmatist_in_another_guise ➚ bert • a month ago

In the UK, Sweden, and perhaps elsewhere, there is need to factor in the impact upon death totals of failure to take measures for effective isolation of high risk populations as in care homes. In the UK elderly patients, some very likely to be infectious were dumped back from hospitals into care homes in order to make room for the deluge of people predicted by 'science'.

^ | v • Reply • Share ›

claude ➚ bert • a month ago • edited

In Sweden, a country with excellent health care, excess mortality has been clearly higher in the past 3 months than to its neighbouring countries (Norway, Denmark, Finland): https://www.euromomo.eu/gra...

^ | v • Reply • Share ›

bert ➚ claude • a month ago

It may be true Claude, more people relatively may have died because of corona in Sweden in the last 3 months. But not that many. But the 'curve' (so the number of deaths), shows the same pattern. how is that possible?

1 ^ | v • Reply • Share ›

claude ➚ bert • a month ago

Bert, as far as I know, in Sweden public gatherings >50 people are forbidden, senior homes are closed for visitors, university and schools for teens >~15 do home schooling, travel ban is announced. So maybe these measures take effects? This would argue for several parameters instead of a complete lockdown solely though.

^ | v • Reply • Share ›

mosmosmos ➚ claude • a month ago • edited

ICL ran the headline on the release of their paper as 'Lockdown and School Closures may have saved 3m lives. They even showed in fig2 that only lockdown had a

significant impact of transmissability which doesn't seem to match the outcomes.

The justification for this figure was comparing actual deaths to the counterfactual.

Sweden neither closed most schools, nor locked down, yet while higher than other countries who put in more stringent rules, only saw a death toll of up to 10% of the counterfactual, even if you consider excess mortality.

How the paper arrives at the conclusion that 'lockdown in particular' slowed the transmission when the only country in the paper which didn't lockdown had a similar outcome is beyond me, it actually appears that NPI's **up to** lockdown saved the overwhelming majority of the lives (against the counterfactual) and lockdown itself had small supplementary impact.

M. Lehmann • a month ago

A couple of comments and questions:
1. Estimated infection fatality rates (IFR) and the underlying methodologies, assumptions, and data sources are central for the results of the model. Why is this critical information only presented in the “supplementary information” section? As a result, the main source (Verity et al, Lancet Infect Dis 2020:20) does not even appear in the literature list of the main article.
2. For estimating IFR, the article uses the approach and numbers presented in Verity et al., correcting for non-uniform attack rates. Verity et al use very early data and analyses from China, from January and February 2020. Specifically, in order to calculate IFR, Verity et al match incidence reported in Wuhan in January 2020 with data on expatriate prevalence from six international repatriating flights on 30 January and 1 February 2020 (!). Do we really not have more recent and better data by now?
3. In a (very brief) passage on page 2, the authors note the role of serological surveys and, in referring to a few of them (from Austria, Denmark, and Spain), note that “these initial results, to some extent (sic!), validate [the] choice of the IFR.” This is remarkably vague -- what does “to some extent” mean? And to what “extent” does it not validate?
4. The choice of the serological studies that are referenced is not explained and seems to be selective. Key studies are missing, including from the countries under investigation, despite them having been widely discussed; for instance Streek at al. (https://www.medrxiv.org/con...). Streeck et al report an estimated IFR of 0.36%, which does _not_ seem to validate the choice of the IFR in the current

paper. The authors may not agree with this and similar findings, but should such findings not at least be mentioned and discussed?
5. The authors also note the limited reliability of most serological surveys, referring to a recent metastudy of serological surveys by Bobrovitz et al. However, the authors do not make an attempt to compare such limitations with any methodological limitations in Verity et al.
6. The choice of the metastudy of serological surveys is again selective; other recent work is not reflected (see https://www.medrxiv.org/con...). Based on currently available evidence, Oxford University's Center for Evidence Based Medicine (CEBM), as of 3rd June 2020, estimates COVID-19 IFR somewhere between 0.1% and 0.41 %, which is _not_ in line with the authors' IFR. Again: the authors may not agree with the results presented by others, but should these results not at least be mentioned and discussed?

John Smith ↗ M. Lehmann • a month ago

https://www.medrxiv.org/con... - has that ever been peer reviewed ? According to Carl Bergstrom, renowned epidemiological modeller, the author picked the surveys with lowest estimates of IFR available and ignored the rest in that meta study ...

Pragmatist_in_another_guise ↗ John Smith • a month ago

Ah, meta-analysis - means of constructing silk purses from sows' ears.