Why Similar Policies Resulted in Different COVID-19 Outcomes: How Responsiveness and Culture Influenced Mortality Rates

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Abstract:

In the first two years of COVID-19, per-capita mortality varied over a hundredfold across countries, despite most implementing similar non-pharmaceutical interventions (NPIs). Factors like policy stringency, GDP, and age distribution only explain a small fraction of mortality variation. To address this puzzle, we build on a previously validated pandemic model in which perceived risk alters societal responses impacting transmission. Using data from over 100 countries, we show that a key factor explaining heterogeneous death rates was not policy responses themselves but rather variation in *responsiveness*. Responsiveness measures how sensitive communities are to evolving mortality risks and how readily they adopt NPIs in response to curb transmission. We further find that responsiveness correlates with two cultural constructs across countries: openness to novelty and power distance. Our findings show that more responsive adoption of similar policies saves many lives, with important implications for the design and implementation of future outbreak responses.

Keyword: COVID-19, Pandemic Policy, System Dynamics, Mortality, Policy Responsiveness

1. Introduction

The COVID-19 pandemic has caused millions of deaths and major health, economic, and social disruptions around the world. But the mortality burden was not distributed evenly. In the first two years of the pandemic the same SARS-CoV-2 virus (and its variants) led to per capita death rates that varied by more than two orders of magnitude across countries.[1] Importantly, differences in many usual explanatory factors such as demographics, per capita income, pandemic preparedness, or healthcare capacity do not explain these vastly divergent outcomes, hinting that the differences in fatalities may instead be due to divergent responses of governments and individuals.[2-4]

The pandemic elicited major responses both from governments and from affected communities globally. Governmental policy responses included imposing a range of non-pharmaceutical interventions (NPIs), such as lockdowns, activity closures, mask mandates, and limits on social gatherings and mobility; as well as pharmaceutical measures, such as novel treatments and vaccination, which started being deployed at scale after the first year and a half.[5, 6] Responses from individuals and communities, from voluntary NPI adoption to adherence to various government mandates, further moderated the spread of the disease.

While studies focused on the short-term impacts (days to weeks) of specific NPIs identify some benefits,[7-9] examining the data over longer time horizons points to three unexpected regularities. First, while specific combinations of NPIs vary, the average stringency of governmental policies was rather similar across countries.[10] Second, important outcomes (such as mortality) were substantially different in various regions,[11, 12] a feature robust to controlling for undercount.[4] Finally, over the horizon of months there is little correlation between stringency of policies and mortality outcomes.[13-15] Appendix S4a provides a simple demonstration of these regularities across 231 countries and regions.[16] The latter observation extends not just to measures of policy but to individual and community responses, such as reductions in mobility, which are likewise not correlated with longer term mortality outcomes(e.g. see Appendix S4b[16]). This policy outcome variation presents a puzzle: How have different countries achieved such vastly different mortality outcomes despite relative similarity in the stringency of their policies and the magnitude of community responses? This variation is especially intriguing as it suggests that more stringent responses are not necessarily required to achieve significantly better outcomes.

In this paper, we offer a novel explanation for the policy outcome variation puzzle, one with important policy implications. We start with the observation that past analyses have not accounted for the feedback loop between health outcomes and implemented policies (with some exceptions [17-19]). In most policy analyses, policies are treated as independent variables affecting the dependent variable of health outcomes. Less appreciated is the other pathway in the feedback loop: that both government policies and public compliance

also change in response to the perceived risk of the disease, as inferred from, e.g., recent deaths. This feedback perspective refocuses the analysis on societal sensitivity to a continuously evolving risk situation. In contrast to thinking about the effectiveness of specific policy responses, one needs to consider 'collective governmental and societal responsiveness' to risk (for brevity 'collective responsiveness' or 'responsiveness' from here on). Greater responsiveness indicates a community's willingness to adopt and adhere to various NPIs even at lower levels of perceived risk. As such, collective responsiveness is a social and cultural construct likely related to risk perception, government priorities and agility, and societal preferences for health outcomes, economic performance, and personal freedoms among others.[20] Explicitly accounting for the feedback loop between health outcomes and societal responses, we first estimate responsiveness for 136 countries around the world and show that this single measure can predict a significant proportion of variation in future mortality rates. We then explore some of the cultural constructs that may explain the observed variations in responsiveness across nations.

1.1. Risk-response feedback and the puzzle of policy outcome variation

The policy outcome variation puzzle asks why responses to COVID-19 had modest variation across nations and barely correlated with the large variations in the COVID-19 outcomes (notably mortality). In response we first observe that while more stringent policies can reduce deaths, the causality can also operate in the opposite direction: more stringent policies are potentially adopted in response to increases in perceived risk due to recent deaths. Such a bidirectional relationship constitutes a risk-response feedback loop where responses reduce deaths, and deaths increase responses. To explore the second part of the relationship further, we correlate, within each country, the weekly policy stringency as a function of recent deaths. A positive correlation emerges where deaths over the previous three weeks predict current-week stringency (average correlation across all countries/regions in our sample is 0.24, SD=0.36; also see Appendix, figure S2[16]). The idea that risk perception and change in responses should be incorporated in epidemic modeling is well recognized.[20] However, its full implications only emerge when the mechanism is modeled as an endogenous feedback process in which epidemic and societal behaviors co-evolve.[10] With a few exceptions,[19, 21, 22] this endogenous feedback mechanism is missing from current models. For example, a recent review of models in CDC's COVID-19 forecast hub finds only 1 of 61 models captures this feedback mechanism.[21]

Transmission reductions in this risk-response feedback result from a combination of official policies and individual behavioral changes, including adherence to those policies; for simplicity, we combine these factors into a single construct of overall response. The feedback from risk levels to this overall response implies that long-term COVID-19 risks (and thus death rates) in each country converge to a threshold that triggers just enough of a response to contain transmission. If perceived COVID-19 risks are below this

threshold, responses remain insufficient to contain transmission, allowing continued disease spread and thus, with some lag, increasing perceived risks. If perceived COVID-19 risks are above that threshold, they trigger responses that bring down transmission and ultimately reduce perceived risk. This feedback framing raises the question of what risk threshold prompts a sufficient response - in other words, how responsive are governments and societies to perceived risks?

This study's central hypothesis is that such collective responsiveness to risk varies across countries, and this variability accounts for a large part of the differences observed in policy outcomes. The subsequent sections of this paper elaborate on this hypothesis, providing a formal estimation of responsiveness to COVID-19 risk across nations and its impact on mortality outcomes. Acknowledging that responsiveness is influenced by social and cultural factors, we further delve into the potential for predicting responsiveness by analyzing specific cultural traits across nations. Understanding the role of responsiveness, and the societal factors that shape responsiveness, is key to better adapting policies to mitigate disease transmission.

2. Study Data and Methods

We use a previously validated model of pandemic dynamics in which governmental policies and behavioral change are a function of the state of the pandemic, operationalized as a response to recent death rates.[21] We first use the model to estimate collective responsiveness across 136 countries and regions by quantifying how recent perceived risk levels drive the societal responses that change transmission rates. We then examine whether the estimated responsiveness measures predict future (out of sample) death rates, and thus policy outcomes, over long-time horizons. We conclude our analysis by exploring the cultural features that predict responsiveness, and thus death rates, across countries.

2.1. Study data

Our estimates of country-level parameters include all 136 countries for which sufficient data are available, covering 7.5 billion people. For simplicity, we limit the estimation period to 1 May 2020 to 31 Mar 2021. We exclude the first 4 months of 2020 to avoid conflating the rise of the first wave of the pandemic with the longer-term dynamics (e.g. over multiple waves in the first two years of the pandemic; see Appendix S4f and S4h for robustness[16]). To reduce model complexity, we choose an end date that largely excludes vaccination effects (only 5 countries exceeded 10% vaccination by that date[1]) and Delta and Omicron variants. For death and case data we use 7-day rolling averages.[1] Unless noted, data for the study come from the OurWorldInData (OWID) global COVID-19 database,[1] which draws on different sources, e.g., the Johns Hopkins University CSSE COVID dashboard for cases and deaths.[23] Recognizing significant death under-reporting in many countries, in sections S4g and S4h we report robustness to using estimates of true infection and death rates from IHME.[4] Other data we use include GDP per capita, population, age

distribution (to calculate country-level Age Multipliers of Mortality), hospital beds per capita, Oxford University government response stringency[10] and independent estimates of (maximum) effective reproduction number R_e (number of secondary cases from an index infection)[24]. In addition, we use Hofstede's cultural dimensions to examine associations between collective responsiveness and cultural constructs [25], available from Hofstede's database.[26]

2.2. Estimating responsiveness

We build on a previously validated epidemic model, the SEIRb model, which incorporates the feedback loop between mortality and societal responses.[21] This model is intentionally simple to aid transparency and generalizability of insights. Nevertheless, it has outperformed many more complex alternatives in forecasting mortality on an extensive dataset of predictions.[21] The model is structurally similar to the classical SEIR (Susceptible, Exposed, Infectious, Removed) compartmental model and incorporates a behavioral risk-response mechanism (thus the b in SEIRb), where transmission intensity declines (increases) as recent death rates increase (decline). 'Responsiveness' represents the strength of this behavioral response mechanism. Formally, rather than being a constant, transmission intensity, β, is a decreasing function of perceived risk of death (f'). f' is operationalized as lagged (per capita) mortality rates. The lag reflects the time it takes for governments and people to perceive and respond to changing risks and thus could vary across communities. As perceived risk of death increases, all else equal, the overall transmission intensity β declines with a 'response' multiplier, k, which captures the impact of various governmental and societal risk-driven responses on transmission. We formulate this multiplier to be proportional to $1/(1+\alpha f')$. Parameter α represents collective responsiveness of the government and society to changing perceived risks. With higher values of responsiveness α, transmission intensity β will be more sensitive to changes in perceived risk. In short, the model separates collective responsiveness (α; a country-specific trait) from changing responses (k), allowing us to estimate responsiveness.

Using the SEIRb model, for each country, we estimate the value for α(as well as lags in risk perception and response adjustment) that offers the best fit between simulated and observed cases and deaths.

2.3. Estimating Contributors to Long-term Deaths Rates

Having estimated country-level collective responsiveness (α), we assess its predictive value in explaining future COVID-19 death rates across nations. We use linear regressions to explain (log10) deaths as a function of (log10) responsiveness. We predict deaths for the period 01 Apr 2021 to 30 Sep 2021, which is excluded from the estimation data. We exclude countries where responsiveness is not reliably identified (is not distinguishable from zero). Moreover, to account for death undercounts we limit the analysis to countries where cumulative excess mortality by 30 Sep 2021 (based on

The Economist's estimates[27]) does not exceed the official COVID-19 deaths by more than 100%. In the Appendix (sections S4f-S4h)[16] we assess robustness to other inclusion thresholds (25% and 50%), exclusion of countries with significant early vaccination, and use of estimates for actual (instead of reported) cases and deaths from IHME.

To put into perspective the predictive value of responsiveness for understanding mortality, we control for a few other explanatory mechanisms including the impact of age distribution on COVID-19 mortality, GDP per capita, maximum reproduction number, healthcare capacity, and average government policy stringency.

2.4. Explaining responsiveness through cultural constructs

Finally, we explore potential correlates of collective responsiveness (α) across countries. Conceptually, responsiveness relates to distinct social, governance, and cultural factors. For example, sensitivity to risk may be related to the community's tolerance for uncertainty and its emphasis on short- vs. long-term outcomes. Hofstede's cultural dimensions offer a common set of measures that inform the hypothesized correlates of responsiveness.[25] These cultural dimensions include individualism, uncertainty avoidance, power distance, masculinity, long-term orientation, and indulgence. They have been estimated for many countries through representative national surveys, and are available for 46 countries in our sample. Similar to above, we exclude countries with too much excess mortality compared to reported COVID-19 deaths. The remaining sample includes 33 countries with all Hofstede's measures (an additional 3 include individualism, uncertainty avoidance, and power distance). We use these cultural factors to predict (log10) responsiveness, and also as separate predictors of (log10) death rates using linear regressions.

2.5. Technical documentation

We follow replicability best practices for model-based analyses,[28] and report full documentation of our data, model, estimation methods, and supplementary analyses in the Appendix and online repository.[16]

2.6. Study Limitations

The current study focuses on three main points: establishing the COVID-19 policy outcome variation puzzle, providing a plausible resolution based on risk-response feedback and variation in collective responsiveness, and exploring cultural determinants of responsiveness. As such, we make many simplifications that should be noted in interpreting the results. First, by estimating a single 'collective' responsiveness measure we combine NPIs, government mandates, and individual behaviors (from adherence to NPIs to hygiene and social distancing) together. Thus we cannot separate effects of distinct behaviors or offer recommendations for specific NPIs; more complex models would be needed for those purposes. Second, to keep the analysis simple we exclude many relevant factors

such as variants, vaccination, adherence fatigue, and loss of immunity. These simplifications increase model transparency and build intuition, but limit its predictive power and realism. Third, we focus on the role of collective responsiveness in predicting mortality, rather than offering a comprehensive explanation of country-level mortality variation, thus missing potential determinants such as comorbidities. Fourth, our preliminary exploration of determinants of responsiveness misses plausible factors such as recent experience of other epidemics or ideological leaning of governments during the pandemic. Fifth, we assume responsiveness is constant, but it likely changes over time due to factors such as adherence fatigue. Finally, the data we use in the primary analysis is based on reported cases/deaths. Those may significantly undercount true incidences and thus we assess the use of alternative data for cases/deaths, as well as including only regions with limited undercount, in our robustness checks. With these simplifications, our analysis provides an illustration of, and a lower bound for, the value of incorporating risk-response feedback in understanding pandemic outcomes and designing more effective policies.[29]

3. Study Results

3.1. Estimating Collective Responsiveness

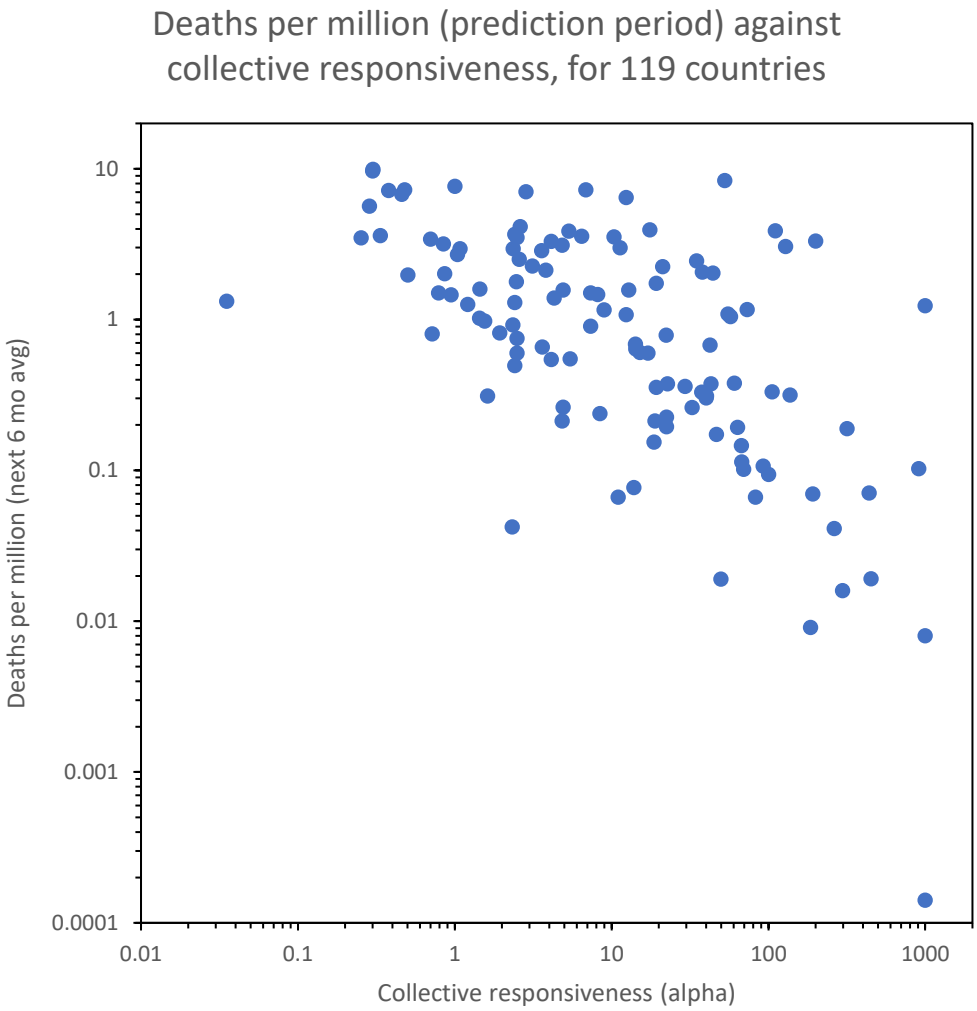
Estimated responsiveness values vary widely across nations and indicate robust effects of risk perception on changing transmission intensity through adoption of NPIs and behavioral changes. For example, at median responsiveness, the number of daily deaths per million that triggers sufficient responses to reduce transmission intensity (β) by 50% is 0.09, with substantial between-country variation (90% range: 0.003-2.67). Appendix table S4 provides estimated responsiveness levels for the sample informing baseline regressions. Moreover, estimated responses (k values) correlate positively with the Oxford University measures of policy stringency (mean/median correlation is 0.35/0.37 across nations).[16] This provides evidence that our estimates of changes in responses over time relate to measures of policy not used in our estimation. Note that we do not expect the correlation to be very strong because k values include population adherence and behavioral change beyond formal policy stringency.

3.2. Collective responsiveness as a predictor of deaths

Exhibit 1 shows that (log10) responsiveness estimated from 01 May 2020 to 31 Mar 2021 is strongly and negatively correlated with (log10) death rates (R=-0.625) averaged over the subsequent 6 months (01 Apr 2021 to 30 Sep 2021).

EXHIBIT 1.

Reported daily deaths per million people (averaged over prediction period, 01 Apr-30 Sep 2021) against estimated collective responsiveness α (estimated from 01 May 2020-31 Mar 2021), with selected larger countries labeled.



Source: Authors' analysis of data on daily confirmed cases and deaths come from the OWID global COVID-19 database. **Note:** Graph includes 119 countries as we exclude countries where responsiveness was too small to be reliability estimated. The correlation in the graph is -0.625.

Regressing the 6-month averaged death rates against responsiveness and several other predictors (Exhibit 2) provides a comparison of these factors in explaining death rates. Responsiveness (log10 α) is the most important driver of variation in death rates (t=-4.1, p=1.9E-4), enhancing the model's fit (adj. R^2) by 0.28 (from 0.00 to 0.28); increasing responsiveness by one standard deviation reduces death rates by a factor of about three (0.35 (0.15-0.82)). In comparison, we find no evidence that initial local transmission intensity, hospital capacity, GDP, or policy

stringency are significant predictors of deaths. Even Age Multiplier of Mortality (a variable calculating expected fatality rates in each country due to age distribution), which is a statistically significant correlate of deaths during the estimation period [30], loses its predictive power for cumulative deaths later in the prediction period (April-September 2021).

EXHIBIT 2.

Predictors of cross-country variation in mortality rates per capita.

	Coefficient(± std. err.)	p-value	Marginal adj. R^2 [a]	Effect Size[b]	95% CI of effect size
Collective responsiveness (log10)	-0.546±0.227	0.000	0.28	0.35	(0.15-0.82)
Age Multiplier of Mortality (log10)	-0.357±0.361	0.474	-0.008	0.84	(0.59-1.19)
GDP per capita (log10)	-0.240±0.226	0.266	0.005	0.83	(0.59-1.17)
Hospital beds per thousand	0.018±0.024	0.685	-0.015	1.12	(0.83-1.50)
Initial reproduction number	-0.142±0.152	0.299	0.002	0.74	(0.39-1.40)
Policy stringency (6 mo. avg.)	0.016±0.009	0.21	0.011	1.44	(0.98-2.11)

Source: Authors' analysis of data from the OWID global COVID-19 database. Note: Dependent variable: per capita mortality (reported daily deaths per million), averaged over the 180 days from 01 Apr 2021 onward. a: Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor; b: Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor; n (number of countries) = 46; Adj. R^2=0.277; F (p-value) = 3.9 (0.003).

We assessed the robustness of these results to various assumptions such as including the early pandemic period, excluding countries with early vaccination, excluding countries with less reliable death data, and using IHME estimates of cases and deaths. Those analyses are detailed in Appendix s4f-s4h and show none of those assumptions change any of the results qualitatively: in all responsiveness remains statistically significant and is the primary driver of variation in death rates.[16] Overall, the results support the hypothesis that responsiveness to risk is a better predictor of mortality variation across countries than many commonly considered factors, from policy stringency, to demographics, healthcare capacity, and transmission potential.

3.3. Cultural measures associated with responsiveness and death rates

Exhibit 3 reports the regression results of collective responsiveness and death outcomes against Hofstede's cultural dimensions. Regression columns M1-M3 with the dependent variable of responsiveness show that 'uncertainty avoidance' and 'power distance' are important predictors of responsiveness. In Model M1 these two cultural constructs explain about 28 percent of variation in responsiveness, and the results are robust after adding other cultural dimensions to the regression in Models M2 and M3.

EXHIBIT 3.

Cultural constructs as explanatory factors for responsiveness and death outcomes for the prediction period

	Collective responsiveness (log)			**Cumulative death (log)**		
	M1	**M2**	**M3**	**M4**	**M5**	**M6**
Intercept	1.887*	-0.363	1.478	0.654	-5.148*	-4.427*
Uncertainty avoidance	-0.035***	-0.030**	-0.030**		0.047****	0.032**
Power distance	0.035**	0.051***	0.048**		0.015	0.038*
Individualism		0.022	0.019		0.016	0.025
Masculinity			-0.001		-0.002	-0.003
Long-term orientation			-0.001		-0.026*	-0.027*
Indulgence			-0.025		0.031	0.019
Collective **responsiveness (log)**				-0.554***		-0.488***
n	36	36	33	33	33	33
R^2	0.28	0.33	0.34	0.27	0.48	0.62
Adjusted R^2	0.24	0.26	0.18	0.24	0.36	0.51
F-statistics	6.42***	5.17***	2.20*	11.16***	3.98***	5.71****

Source: Authors' analysis of data from the OWID global COVID-19 database and Hofstede's cultural measures from Geert Hofstede's database. **Note:** * p < .1 ** p < .05 *** p < .01 **** p < .001; Blank cells are for variables not included in the regression model of the column; M: Regression model; Dependent variables: collective responsiveness (estimated from 01 May 2020-31 Mar 2021) and per capita mortality (reported daily deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

Models M4-M6, with the dependent variable of (log10 of average daily) deaths during the prediction period show that these cultural

dimensions can partially explain differences in mortality outcomes. Model M4 shows the predictive value of responsiveness alone for the subset of countries for which cultural measures are available. In M5 we show that the association between risk avoidance (and, to a lesser extent, long-term orientation) with deaths is statistically significant, and the cultural constructs alone explain about 48 percent of variation in deaths across different countries. Model M6 adds responsiveness to the predictors and shows improved predictive power against Model M5. As in model M5 long-term orientation is negatively associated with deaths. Interestingly, controlling for responsiveness, power distance also becomes marginally predictive of deaths. In summary, we note that 1) two cultural constructs, uncertainty avoidance and power distance, partially predict the variation in responsiveness, 2) they are also associated with the variation in mortality outcomes; and 3) cultural constructs partly explain the association between responsiveness and deaths, yet the impact of responsiveness is not limited to the pathways overlapping with cultural precepts.

4. Discussion

Examining COVID-19 mortality globally points to a puzzling variation in policy outcomes. Specifically, during the acute phase of the pandemic, the stringency of government and societal responses was similar across most countries, yet mortality outcomes varied by more than a hundred-fold. While some studies have shown immediate effects of NPIs which seem intuitive,[7-9] others have found variability in effects,[11, 12] or concluded that such policies are ineffective.[15] To resolve this conundrum, we noted that not only policies and responses impact the state of an epidemic, but also the state of the epidemic regulates those responses, via risk perception. Thus the primary factor driving variation in mortality rates is not specific policies implemented, but rather different societies' responsiveness to perceived risk. By explicitly modeling the feedback loop between societal responses and the pandemic's progression, we estimated a measure of responsiveness and correlated it with future deaths. We then explored cultural antecedents of responsiveness.

Three findings emerge. First, the degree of responsiveness to evolving pandemic risks varies markedly among nations. Second, estimated responsiveness is highly predictive of future COVID-19 mortality rates. In fact, responsiveness is a stronger predictor of mortality outcomes than several intuitive predictors, including demographics, healthcare capacity, NPI policy stringency, the maximum reproduction number, and GDP per capita. Responsiveness encapsulates societal and policymaking sensitivity to the pandemic's risks: the number of daily deaths required to compel the adoption of sufficient responses to curb transmission, as well as the speed and effectiveness of policy implementation.

To understand the importance of responsiveness, consider a typical outbreak wave in a community. Initially the epidemic grows,

with increases in cases, deaths, and hence perceived risk. As the toll escalates, policymakers and the community are compelled to respond, adopting NPIs and other measures to reduce transmission and ultimately slowing the spread of the disease. This shift results in declining transmission rates, with mortality rates soon following suit. Over time, as the memory of the wave fades and perceived risk lessens, responses are relaxed, allowing renewed transmission. Eventually, the laxness of policy in the presence of infection, seeds the start of a new wave. In essence, the mix of responses converge to those required to keep the epidemic from growing exponentially or subsiding fully, keeping perceived risks at levels just tolerable for the community. Analytically, these response levels are those needed to keep the effective reproduction numbers near one. The specific death rates that trigger this strength of response, however, heavily depend on the community's responsiveness. Communities with higher responsiveness require lower death rates to trigger sufficient policies and adherence to those. This mechanism is fundamental in explaining how variation in responsiveness predicts observed death rates across communities. The oscillations in response due to this feedback loop also provide a mechanism for endogenous emergence of pandemic waves that complements other triggers such as new variants, loss of immunity, and seasonality.

Third, cultural attitudes partially account for variations in responsiveness. Hofstede's measures of uncertainty avoidance and power distance are associated with responsiveness. The association of power distance with responsiveness indicates that communities more willing to follow the mandates of a centralized government may be more responsive to a fast-changing public health threat. The inverse relationship between uncertainty avoidance and responsiveness may seem unexpected. However, this relationship may underscore the value of societal tolerance towards change and novelty in facilitating rapid policy responses and the adoption of potentially disruptive NPIs. We also note that combining responsiveness and Hofstede’s cultural constructs provides a more accurate prediction for mortality than either alone.

Our findings have significant policy implications. First, they challenge the perceived trade-off between saving lives and minimizing disruptions during the critical phase of a pandemic. When infection fatality rates are sufficiently high, the implementation of NPIs becomes inevitable as the threat of an exponential outbreak compels communities to control transmission. Regardless of timing, every community will need to adopt a mix of NPIs sufficient to curtail the exponential growth in deaths. Thus, the limited correlation between policy responses and deaths does not imply that NPIs against COVID-19 are ineffective, contrary to arguments raised by some.[15] Rather, the correlation with mortality vanishes because all communities ultimately needed to adopt stringent enough responses to curb exponential transmission. Communities that enacted the requisite policies earlier (i.e., not waiting for high levels of mortality) achieved life-saving results without imposing additional societal costs.

Second, policy-makers would be better off to focus their attention on responsiveness. Whereas all communities adopted comparable response levels, their responsiveness varied by two orders of magnitude. From protocols for rapid response, to having tighter response thresholds, and openly communicating with the public about the importance of responsiveness, policy makers can take actions that enhance responsiveness and thus could contribute significantly to reducing the burden of an epidemic. In fact, it is critical for policy makers to articulate the insight that heightened responsiveness and swifter action would preserve lives without requiring the implementation of more stringent policies. That could help secure public backing for more agile, responsive policies in managing future pandemics with major life-saving benefits. If all countries had the responsiveness of the top ten percentile, the COVID-19 death toll in the first two years could have been reduced by nearly an order of magnitude.

5. Conclusion

This global-scale study points to the importance of policy responsiveness rather than policy response in reducing mortality during a deadly pandemic. Responsiveness varies widely across nations. While cultural factors significantly influence responsiveness, they account for only about one-third of the variation in estimated responsiveness, indicating that policymakers and communities have scope to enhance responsiveness. Understanding the social mechanisms and organizational structures that enabled governments in certain countries to adopt more responsive policy stances, implement coherent sets of NPIs at lower risk levels, and encourage public adherence to these policies is crucial in preparing for future pandemics.

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Online Appendix to Accompany:

Why Similar Policies Resulted in Different COVID-19 Outcomes: How Responsiveness and Culture Influenced Mortality Rates

Contents

S1) Model specification

Our model (SEIRb) is an extension of the classic compartmental SEIR model [1], which incorporates behavioural responses that endogenously change contact rates as a continuous function of perceived risk. SEIRb is previously reported and validated in peer-reviewed venues and shows strong predictive power for future deaths despite its simplicity [2]. Figure S1 provides an overview of the model structure.

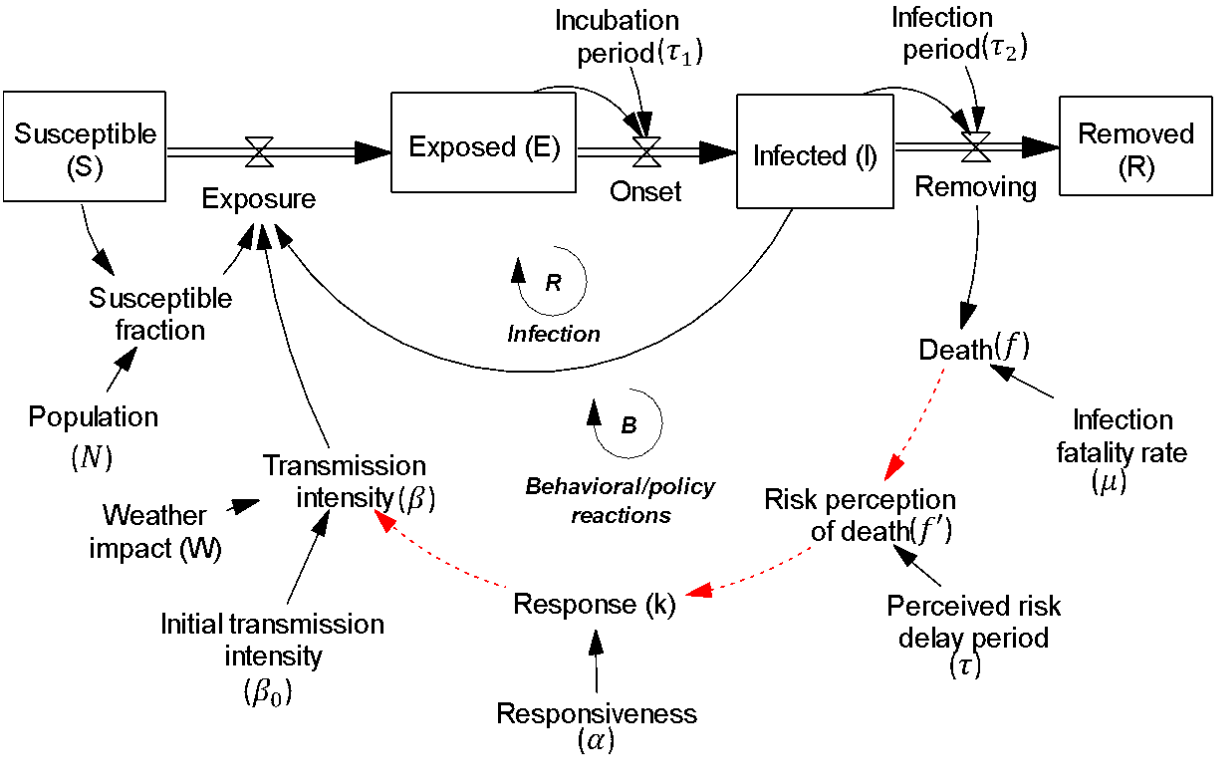


Figure S1: SEIR with a behavioural/policy reaction feedback

In this model, populations are divided into four groups: susceptible (S), pre-infectious (E), infectious (I), and removed from circulation due to recovery or death (R):

$$\frac{dS}{dt} = -\frac{\beta SI}{N} \quad (1)$$

$$\frac{dE}{dt} = \frac{\beta SI}{N} - \frac{E}{\tau_1} \quad (2)$$

$$\frac{dI}{dt} = \frac{E}{\tau_1} - \frac{I}{\tau_2} \quad (3)$$

$$\frac{dR}{dt} = \frac{I}{\tau_2} \quad (4)$$

$$f = \mu \frac{I}{\tau_2} \quad (5)$$

The three main parameters are the daily infectious contacts per case (β), the average latent period (τ_1), and the average infectious duration (τ_2). Total population $N = S + E + I + R$. The basic reproduction number (R_0), i.e. the expected secondary cases from an index case in a fully susceptible population, is thus:

$$R_0 = \beta\tau_2 \quad (6)$$

With a constant β and $R_0 >> 1$ [3, 4], the basic SEIR model predicts a rapid COVID-19 outbreak that infects most of a population in a few months, reaching herd immunity when a large fraction of the population has been infected [5]. However, behavioural and policy responses to risk change infectious contact rates (β) and may curtail the epidemic well before herd immunity. We thus allow β to go below β_0 in response to perceived risk of death (f'):

$$\beta = kW\beta_0 \quad (7)$$

where k represents response to change in perceived risks of f', and reflects the strength of responses affecting contact rates. $k(f')$=1 indicates pre-pandemic behaviour, whereas full societal lockdown may push k to smaller values, bringing β to ~0. W is the seasonal effect of weather on COVID-19 transmission, estimated elsewhere [6, 7]. k should be decreasing in f′, with $k(f' = 0) = 1$, but its exact functional form is not critical, so for simplicity we choose a following form:

$$k(f') = \frac{1}{1+\alpha f'} \quad (8)$$

This leads to

$$\beta = (\frac{1}{1+\alpha f'})W\beta_0 \quad (7')$$

α represents the collective government and society *responsiveness to risk* in a community. α=0 recovers the basic SIR model, and higher α values indicate a community more sensitive to the perceived risk of death from the disease. f' is modelled as an exponential average of reported per-capita daily death rate (f; Equation 9) resulting from an infection fatality rate of μ, with a time constant of τ reflecting the time it takes to perceive and respond to changing risks:

$$df'/dt = (f - f')/\tau \quad (9)$$

Recognizing that responding to growing risks and relaxing responses based on declining risks may occur on different timescales, we allow for an asymmetric adjustment time τ for increasing vs. decreasing perceived risk:

$$\tau = \tau_u \; if \; f'(t) < f(t) \quad \& \quad \tau_d \; if \; f'(t) \geq f(t) \tag{10}$$

In addition, for each country, we estimate the epidemic seeding time (t_Z).

This model includes 8 parameters $(\tau_1, \tau_2, \beta_0, \alpha, \mu, \tau_u, \tau_d, t_Z)$ as well as one time-varying input W. Based on prior literature, we specify $\tau_1 = 4$ and $\tau_2 = 10$ days [8, 9]. We calculate base IFR (μ) for each country based on age distribution [9] (see S3.a)), and use prior estimates for W [6, 7]. The remaining five parameters $(\beta_0, \alpha, \tau_u, \tau_d, t_Z)$ are estimated.

S1.a) Accounting for under-ascertainment

Under-ascertainment is a substantial challenge for models of COVID-19 transmission. The estimation of our model is not sensitive to under reporting of cases. To illustrate, consider true infection rates (r_E) for each country and how it compared to reported cases:

$$r_{EM} = \gamma_I r_E = \gamma_I \frac{\beta I S}{N} \tag{11}$$

The subscript *M* denotes reported (rather than true) values, and γ_I is the ratio of reported to true infections. To keep the model simple, we bypass the need to estimate ascertainment by approximating reported infection rates r_{EM} based on recent reported infections I_M, assuming that current infections are under-reported to the same degree as infections over the last few weeks:

$$r_{EM} = \gamma_I \frac{\beta I S}{N} \approx \frac{\beta I_M S}{N} \tag{12}$$

$$I_M = \int \quad (r_{EM} - \frac{I_M}{\tau_R}) dt \tag{13}$$

This approximation holds as long as γ_I is relatively stable on the timescale of the disease duration (~two weeks), even if it changes over longer time horizons.

In short, even though reported cases are significant undercounts (by an order of magnitude or more), the fractional change in reported cases is an unbiased estimator of fractional change in actual cases, as long as ascertainment rate is not changing over a period of a couple of weeks. As such, we can use that fractional change in cases to estimate transmission intensity directly.

S2) Estimation method

Our model yields an expected number of new reported infections (r_{EM}) and per-capita death rate (d_N) for each day for each country. Those expected numbers are specified in Equations 12 and 8 in S1), as a function of several known and unknown parameters and the state of the model prior to the current date. We simulate the system of differential equations captured in the model to calculate those outcomes over time. We estimate the vector θ of unknown parameters for each country by maximum likelihood, identifying the vector $\hat{\theta}$ that maximises the likelihood of observing the true reported infections and deaths y_{vt} (where t is time (day) and $v \in [i,d]$ denotes the series of infections or deaths) given $\hat{\theta}$.

We use a negative binomial [log-]likelihood function for both cases and deaths:

$$LL(\theta,\lambda_v) = \sum_v \sum_t -\frac{ln\,(1+\lambda_v x_{vt})}{\lambda_v} + \Gamma\left(x_{vt}+\frac{1}{\lambda_v}\right) - \Gamma\left(\frac{1}{\lambda_v}\right) - \left(x_{vt}+\frac{1}{\lambda_v}\right) ln\left(1+\lambda_v y_{vt}(\theta)\right) + x_{vt}(ln\left(y_{vt}(\theta)\right) + ln\,(\lambda_v))$$

Where x_{vt} is predicted infections or deaths (analogous to y_{vt}), λ_v are the scaling factors for the likelihood function, and $\Gamma(z)$ represents the natural logarithm of the generalized factorial function for $z-1$ (i.e. $\Gamma(z+1) = ln(z!)$ for integer z).

The 5 unknown parameters are listed in **Table S1**. We also estimate the negative binomial scaling factors (λ_i, λ_d), leading to a total of 7 estimated parameters for each country.

Table S1. Estimated parameters with allowed ranges.

Parameter	Symbol	Range	Units
Initial infectious contact rate	β_0	0.1-4.0	1/day
Responsiveness	α	0.001-1000	Dimensionless
Time to increase perceived risk	τ_u	1-100	Days
Time to reduce perceived risk	τ_d	10-400	Days
Patient Zero arrival time*	t_z	1-140	Day
Likelihood scaling factor (infections)	λ_i	1E-06-1	Dimensionless
Likelihood scaling factor (deaths)	λ_d	1E-12-0.6**	Dimensionless

* *Patient Zero arrival time* (i.e. epidemic seeding time) is expressed in days from the start of 2020 ** To avoid a potential computational problem in which the estimation algorithm ignores deaths relative to infections in the likelihood function, the scaling factor for deaths $\boldsymbol{\lambda_d}$ is defined relative to $\boldsymbol{\lambda_i}$ as 1E-06-0.6 x $\boldsymbol{\lambda_i}$

We estimate the model separately for each country. Separating countries significantly speeds up estimation and makes it feasible to conduct the full analysis with limited computing resources. For each country, we estimate the parameters using the Powell direction search method implemented in Vensim™ DSS simulation software, restarting the optimization at 20 random points in the feasible parameter space. Overall, estimation for all 130 countries takes approximately 12 hours when compiled and parallelized on a 48-core Windows 10 server.

Model and calibration files are available at https://github.com/tseyanglim/CovidRiskResponse.

S3) Data sources & preparation

Data used in Figure S2: Government response index is directly adopted from Oxford government response tracker database, and is aggregated on a weekly level. It is a previously published aggregated index that covers 16 policies in the domains of containment and closure, economic, and health system[12](https://github.com/OxCGRT/covid-policy-dataset/blob/main/documentation_and_codebook.md#calculation-of-policy-indices). Death rates for each country and region are collected from Oxford government response tracker database, sourced from Johns Hopkins University CSSE Covid-19 data repository (https://github.com/CSSEGISandData/COVID-19).

Data on daily confirmed cases and deaths come from the OurWorldInData (OWID) global COVID-19 database [10], which draws on the Johns Hopkins University CSSE COVID dashboard [11]. The CSSE dashboard in turn aggregates its data primarily from official sources such as the US Centres for Disease Control and Prevention (CDC), the European CDC, the World Health Organization, and national health ministries, updating at least daily.

We use OWID's 7-day rolling averages for new cases ('new_cases_smoothed') and deaths per million population ('new_deaths_smoothed_per_million'). COVID-19 case and death reporting data show strong weekly cycles in many countries, as well as occasional anomalous spikes due to e.g. irregularities in test reporting or redefinitions by government statistical agencies; using the rolling average data smooths out these cycles, which we are not attempting to model here, to better reflect underlying trends.

Our analysis includes all countries in the dataset with at least 10000 cumulative cases reported, and at least 20 days of data, by 31 Mar

2021. We exclude countries with fewer than 10000 cumulative cases to avoid skewing the results with outliers and ensure robust estimation. In total, 130 countries meet these criteria by 31 Mar 2021. As discussed in the main text, we restrict the estimation period to 31 Dec 2019 to 31 Mar 2021 to avoid the confounding impacts of vaccination and new variants, which are beyond the scope of this model. In the main analysis, we exclude countries with more than 100% excess mortality. The data are obtained from the Economist (https://raw.githubusercontent.com/TheEconomist/covid-19-the-economist-global-excess-deaths-model/main/output-data/export_country_cumulative.csv). In the Supplementary analysis we test three other conditions of excluding countries with 50% and 25% excess death, as well as including all countries.

For countries included, we utilise data starting from the date when they exceed 100 cumulative cases reported. Excluding early data entails a trade-off. Excluding it makes estimating the true basic reproduction number (R_0) more difficult –after forceful outbreaks in the first-affected countries, most others adopted various responses that brought R_e down below its pre-pandemic level (R_0). Furthermore, excluding the early data may cut out the initial dynamics of transmission. As a result, our estimated values for the initial reproduction number are likely underestimates of the basic reproduction number. On the other hand, many of the early cases reported in most countries were due to travellers, so this earliest importation-dominated stage therefore may not accurately reflect the community transmission dynamics we are modelling. Moreover, early on rapid changes in testing coverage impact our ability to assume ascertainment rates are stable in the τ time horizon as needed in our derivations (see Equation 12). We selected the 100 case cut-off to balance these competing considerations.

For prediction of deaths (but not model estimation), we use several other country statistics such as GDP, hospital beds per thousand people, median age, and so on, with data as compiled by OWID[1] [10]. We also use government response stringency data [12] and independent estimates of R_e [13] compiled by OWID and available through their COVID-19 data hub. The full list of additional variables used and corresponding OWID data codes is in **Table S2** below.

For mobility data we use Google's COVID-19 Community Mobility Reports[2], accessed via OWID[3] to provide consistent mapping for country names to other data we use. These data reflect changes in numbers of visitors relative to pre-pandemic levels, controlling for weekly (but not longer seasonal) cycles. We use the averaged value of percentage changes in visits to two categories of locations – workplaces, and

[1] https://raw.githubusercontent.com/owid/covid-19-data/master/public/data/owid-covid-data.csv
[2] https://www.google.com/covid19/mobility
[3] https://ourworldindata.org/covid-mobility-trends

retail & recreation venues – which best reflect normal everyday economic activity.

Data are downloaded and processed with Python 3 code, using Pandas and NumPy packages. For the full data processing code, see https://github.com/tseyanglim/CovidRiskResponse.

Table S2. Additional variables drawn from OWID dataset and variable codes.

Variable	Data code
Effective reproduction number R_e	reproduction_rate
Govt. response stringency index	stringency_index
Hospital beds per thousand population	hospital_beds_per_thousand
Median age	median_age
Per-capita gross domestic product (PPP)	gdp_per_capita

In addition, we use Hofstede's cultural dimensions [14, 15] to examine associations between collective responsiveness and cultural constructs. Hofstede's measures are obtained from Geert Hofstede's website and via this link: https://geerthofstede.com/research-and-vsm/dimension-data-matrix/. The dataset includes all six measures of power distance, uncertainty avoidance, individualism (vs. collectivism), masculinity (vs. femininity), long-term orientation, and indulgence for 111 countries or regions. While the dataset includes regional categories (e.g., Africa East, Africa West, Arab countries), we only use data on countries that are explicitly listed.

S3.a) Age multiplier of mortality calculation

Age strongly influences mortality rates, with older patients far more likely to die of COVID-19 [16]. To account for the impact of demographic differences on fatality rates and improve model estimation, we therefore calculate country-specific age multipliers of mortality based on each country's age structure.

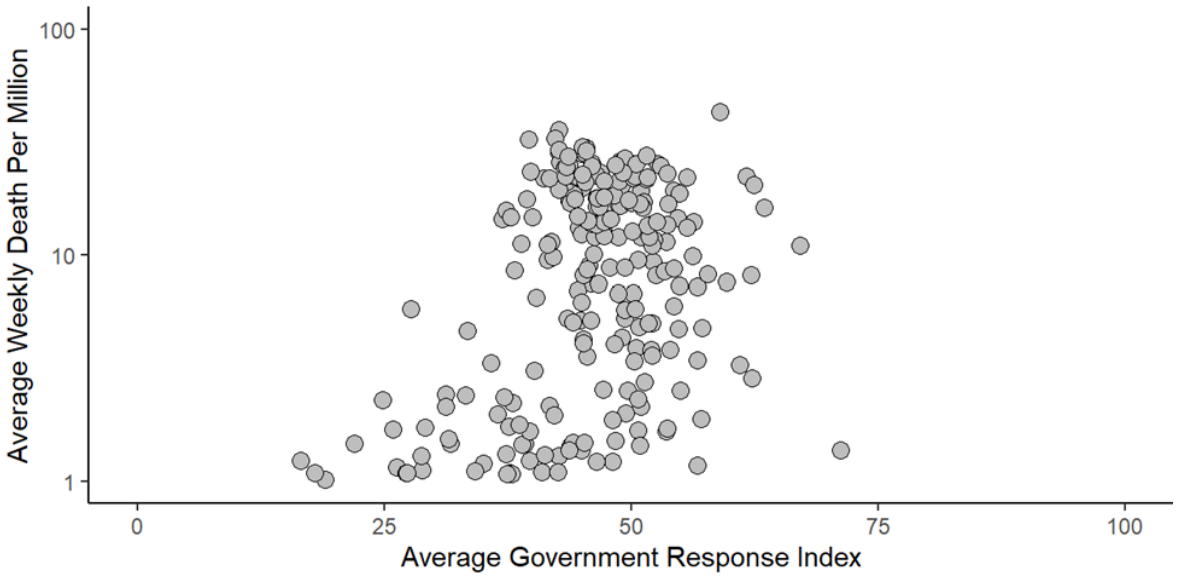
We use data from the World Bank's World Development Indicators [17] on the age distribution of each country's population in 10-year age strata to calculate an age-weighted average IFR multiplier for COVID patients by 10-year age group estimated.[16] This multiplier is applied to a baseline IFR of 0.50%. The resulting demography-adjusted country-specific IFR multipliers range from 0.28 (Uganda) to 3.02 (Japan), with a mean of 1.08 and median of 0.88 (Lebanon). For the handful of countries for which up-to-date demographic data are unavailable, we use a multiplier of 1, leading to a baseline IFR of 0.50%.

S4) Supplementary analysis

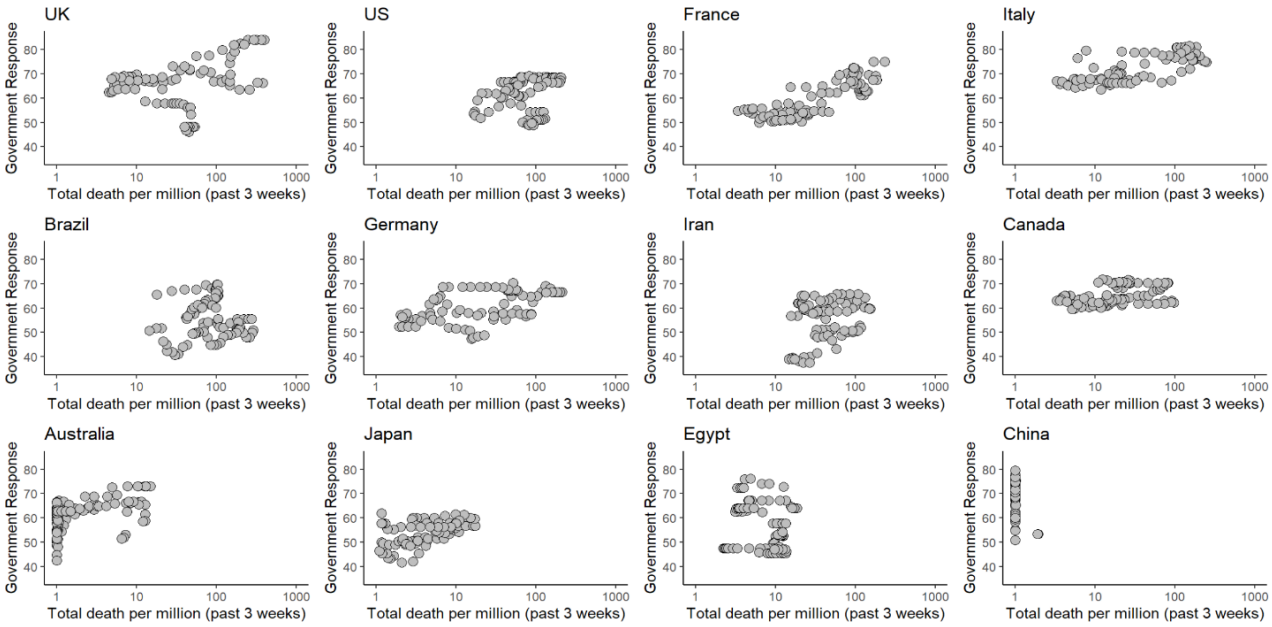
S4.a) Correlations of Policy Stringency with Lagged Death

Figure S2a plots death rates (average weekly deaths per million) against policy stringency (average weekly level of Government Response Index from OxCGRT),across 231 countries and regions(USA States) using data from 03 May 2020 to 01 Jan 2022. It shows that: 1) average (long-term) government responses are comparable across most regions (around 50); 2) average death rates vary by two orders of magnitude (note the logarithmic scale); and 3) the two are not strongly associated with each other. These three features summarize the puzzle of policy outcome variation.

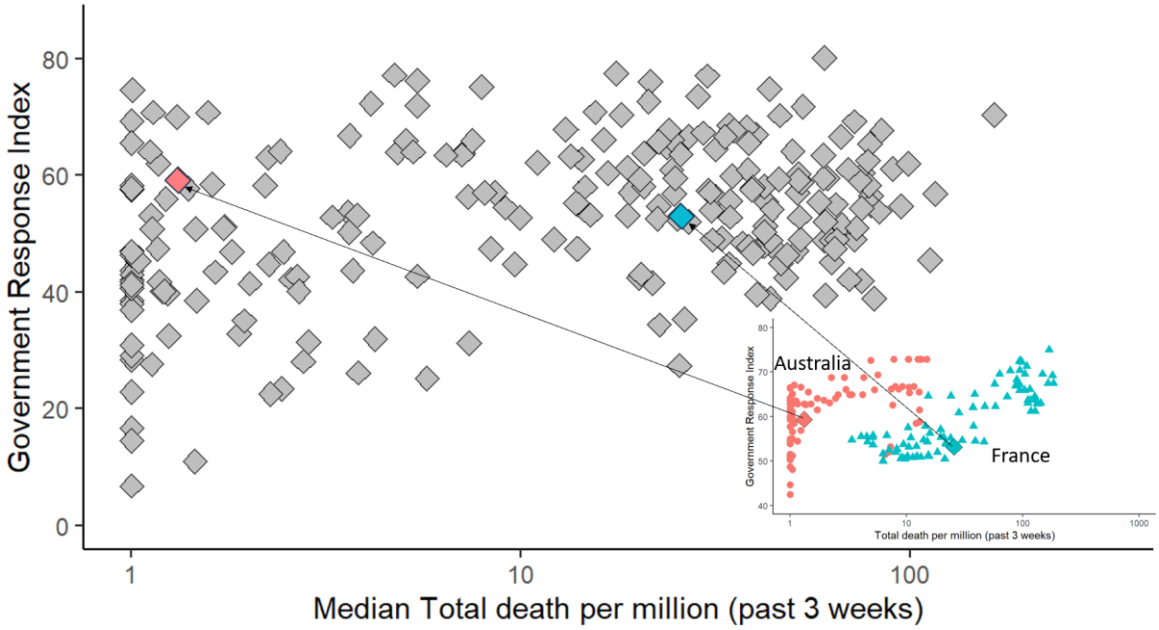
Figure S2b plots recent COVID-19 deaths (previous three-weeks, per million population), as a measure of perceived pandemic risk, against policy stringency (measured using weekly level data of government response index from Oxford COVID-19 Government Response Tracker),[10] across a sample of 12 countries (Panel b). A common relationship emerges where an increase in recent deaths corresponds with increased stringency of policies.



(a)



(b)



(c)

Figure S2: Variation in government policy responses. Panel a: Average reported death rates (logarithmic scale) against average Government Response Index in a global sample of 231 countries and regions. Each point represents average-over-time for a country or region. Data spans 03 May 2020 to 01 Jan 2022. Panel b: A sample of 12 countries. Each point represents data for a specific country in a specific week. Panel c: Government responses at median death rate. Main figure plots government response index at median (past 3-weeks) death rates in a

global sample of 231 countries and regions. Each point represents a country or region. Inset figure plots changes over time in government response index vs death rates (in past 3 weeks) for two countries (France: Green Triangles; Australia: Red Circles; each point represents data in a specific week) as examples and shows how each point in the main figure is calculated.

Figure S2c plots stringency of government response at median death rates for each country and region. It shows that while the resulting stringency levels are fairly similar across nations (levelling off around 60), the mortality levels at which the stringent policies are triggered (i.e., responsiveness) vary widely. Specifically, the government response stringency at median mortality rates is stable across most regions, despite the median mortality rates varying by two orders of magnitude (e.g., Australia reaches a high level of government response stringency at a much lower mortality rate than France). This points to the importance of analyzing the feedback loop that includes societal response and the state of the disease.[20, 21]

S4.b) Correlations with additional potential explanatory variables

Figure S3 shows additional correlations (or lack thereof) between reported per-capita death rates (averaged from 01 Apr-30 Sep 2021) and various country characteristics. Panel A shows the correlation between our estimated collective responsiveness and death per million per day, which is the strongest correlation among depicted variables in the Figure.

Panels B-F show a modest (or lack of) correlation between various variables and death rate. Particularly in Panel B age multiplier of mortality (which drives infection fatality rates) only modestly correlates with death rate. As depicted in Panel C, across countries, death rate is actually *positively* correlated with per-capita GDP. Hospital capacity and initial reproduction number do not show a strong association with death rate. Finally, as discussed in the paper the correlation between policy stringency and death is small, and interestingly positive, indicating the reciprocal relation between the two concepts.

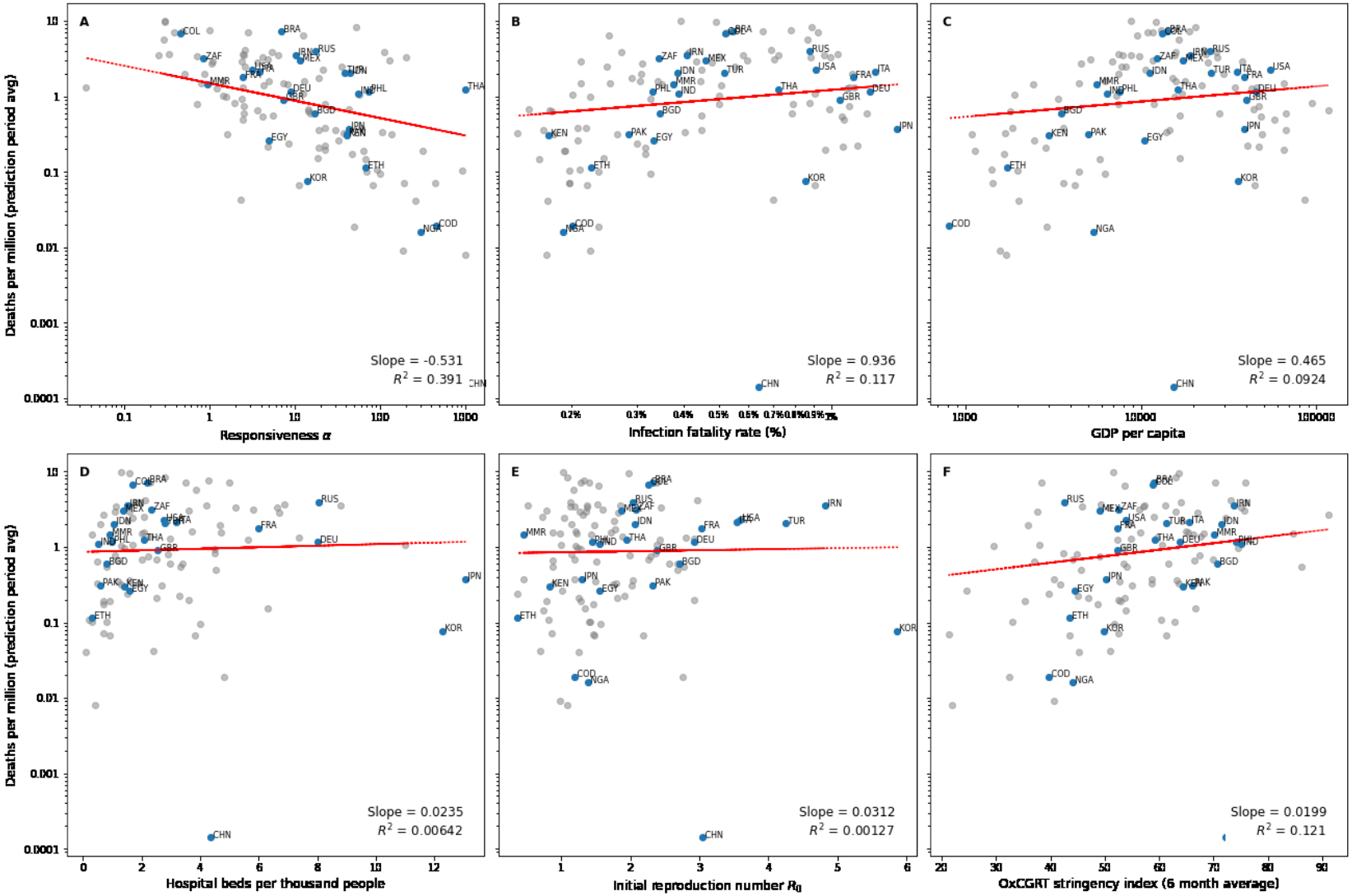


Figure S3. Reported death rates per million people (average over 6-months from 01 Apr-30 Sep 2021) correlated with various factors: A) collective responsiveness, B) age multiplier of mortality (informing infection fatality rate), C) GDP per capita (PPP), D) hospital beds per thousand people, E) initial reproduction number (R0), and F) policy stringency index.

Figure S4 shows estimated death rates per million against change in visits to retail & recreation venues and workplaces respectively, relative to pre-pandemic levels. The two categories separately show similar negative correlation to the combined mobility index, as shown in the main text.

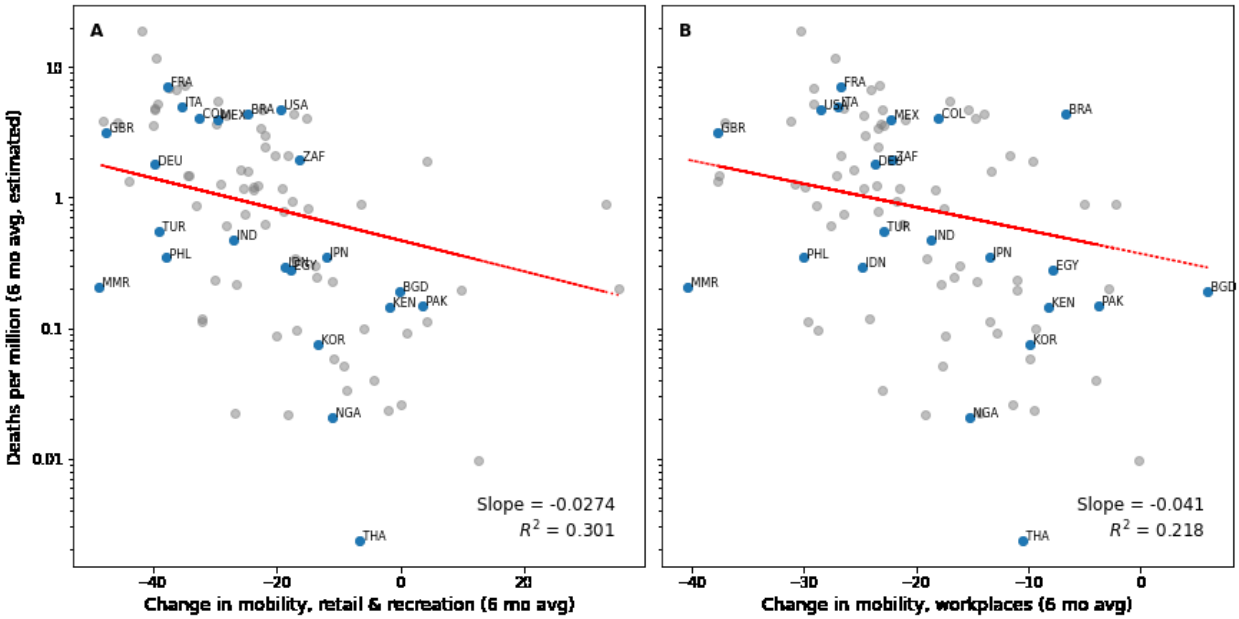


Figure S4. Estimated death rates per million (average over 6-months ending in Mar 31, 2021) correlated with change in mobility (during same period) relative to pre-pandemic levels for A) retail & recreation venues and B) workplaces.

S4.c) Parameter estimates

Table S3 shows summary statistics across all countries for estimated model parameters. For the full table of parameter estimates by country, see https://github.com/tseyanglim/CovidRiskResponse.

Table S3. Summary statistics of estimated parameter values.

Parameter	Symbol	Mean	Median	StDev
Initial infectious contact rate	β_0	1.86	1.36	1.55
Collective Responsiveness	α	72	11	187
Time to increase perceived risk (days)	τ_u	28.2	12.2	35.8
Time to reduce perceived risk (days)	τ_d	106	50.3	132
Patient Zero arrival time*	t_z	86.3	101	29.6
Likelihood scaling factor (infections)	λ_i	0.578	0.805	0.433
Likelihood scaling factor (deaths)	λ_d	0.235	0.117	0.25
* *Patient Zero arrival time* is expressed in days from the start of 2020				

S4.d) Estimated collective responsiveness values

Table S4 shows responsiveness values for different countries, expressed in terms of the death rate per million population that would be required to trigger responses sufficient to lower

infectious contact rates by 50%. Lower values therefore indicate greater responsiveness or sensitivity to death rates. Note the wide range of values estimated, with some countries (e.g. China, Thailand) far more responsive than others (e.g. Argentina, France). Consistent with various regressions and other analyses, the table excludes countries for which excess deaths exceed twice the reported COVID-19 deaths for the duration of analysis, or those whose estimated responsiveness could not be distinguished from zero (and thus 1/responsiveness is undefined).

Table S4. Estimated responsiveness values for different countries.

Country code	Population (millions)	Death rate per million resulting in 50% contact rate reduction (1/collective responsiveness)
AFG	41.1	0.07
AGO	35.6	0.015
ALB	2.8	1.392
ARE	9.4	0.052
ARG	45.5	2.084
ARM	2.8	0.401
AUS	26.2	0.091
AUT	8.9	28.458
AZE	10.4	0.204
BEL	11.7	0.828
BFA	22.7	0.001
BGD	171.2	0.059
BIH	3.2	0.352
BLR	9.5	0.081
BOL	12.2	0.425
BRA	215.3	0.146
BWA	2.6	0.188
CAN	38.5	0.07
CHE	8.7	0.414
CHL	19.6	0.382
CHN	1425.9	0.001
CIV	28.2	0.005
COD	99	0.002
COL	51.9	2.18
CPV	0.6	0.078
CRI	5.2	3.945
CUB	11.2	0.005
DEU	83.4	0.112
DNK	5.9	0.207
DOM	11.2	0.034
DZA	44.9	0.009
ECU	18	0.047
EGY	111	0.204
ESP	47.6	0.414

EST	1.3	1.992
ETH	123.4	0.015
FIN	5.5	0.045
FRA	67.8	0.404
GAB	2.4	0.054
GBR	67.5	0.136
GHA	33.5	0.012
GIN	13.9	0.001
GTM	17.8	1.161
GUY	0.8	2.975
HND	10.4	0.96
HTI	11.6	0.022
IDN	275.5	0.023
IND	1417.2	0.018
IRL	5	0.066
IRN	88.6	0.097
IRQ	44.5	0.645
ITA	59	0.262
JAM	2.8	0.029
JPN	124	0.023
KAZ	19.4	0.279
KEN	54	0.025
KGZ	6.6	0.426
KOR	51.8	0.072
KWT	4.3	0.136
LBY	6.8	0.691
LKA	21.8	0.008
LSO	2.3	0.053
LUX	0.6	0.517
MAR	37.5	0.045
MDA	3.3	0.207
MDG	29.6	0.011
MDV	0.5	0.052
MEX	127.5	0.088
MKD	2.1	1
MLI	22.6	0.004
MMR	54.2	1.058
MNE	0.6	3.491
MOZ	33	0.016
MRT	4.7	0.044
MWI	20.4	0.007
MYS	33.9	0.009
NAM	2.6	0.081
NGA	218.5	0.003
NLD	17.6	0.242

NOR	5.4	0.045
NPL	30.5	0.122
OMN	4.6	0.924
PAK	235.8	0.025
PAN	4.4	0.232
PER	34	3.337
PHL	115.6	0.014
POL	39.9	0.242
PRT	10.3	0.401
PRY	6.8	3.322
PSE	5.3	1.268
QAT	2.7	0.276
ROU	19.7	0.414
RUS	144.7	0.057
RWA	13.8	0.017
SAU	36.4	0.619
SDN	46.9	0.014
SEN	17.3	0.031
SGP	5.6	0.43
SLV	6.3	0.694
SOM	17.6	0.003
SSD	10.9	0.005
SWE	10.5	0.401
SWZ	1.2	0.389
SYR	22.1	0.119
TGO	8.8	0.002
THA	71.7	0.001
TJK	10	0.02
TUN	12.4	2.635
TUR	85.3	0.026
UGA	47.2	0.027
UKR	39.7	0.155
URY	3.4	0.019
USA	338.3	0.32
UZB	34.6	0.01
VEN	28.3	0.184
XKX	1.8	1.425
ZAF	59.9	1.182
ZMB	20	0.024
ZWE	16.3	0.017

Note: Variation in reciprocal of responsiveness across countries. Reported values are daily death rates triggering a response that brings infectious contacts to half the baseline value (i.e. 1/alpha).

S4.e) Predictor variable correlations & variance inflation factors

Table S5 shows correlations between all predictor variables used in the full death rate regression (**Exhibit 2** of main manuscript), as well as variance inflation factors (VIFs); all VIF values are < 3, indicating acceptably low multicollinearity between predictors.

Table S5. Correlation matrix and variance inflation factors for predictor variables in full regression.

	Log alpha	Log IFR	Log gdp per capita	Hosp beds	R0 est	Hist str 180	Variance Inflation Factor
log_alpha (Log10 of Responsiveness)	1	0.02	-0.018	0.11	0.064	0.268	1.12
log_IFR (Log10 of Age Multiplier of Mortality)	0.02	1	0.371	0.542	0.461	-0.432	1.94
log_gdp_per_capita (Log10 of GDP per Capita)	-0.018	0.371	1	0.151	0.339	-0.278	1.28
hosp_beds (Hospital Beds per Thousand Population)	0.11	0.542	0.151	1	0.429	-0.246	1.56
R0_est (Max Reproduction Number)	0.064	0.461	0.339	0.429	1	-0.056	1.5
hist_str_180 (Stringency Index)	0.268	-0.432	-0.278	-0.246	-0.056	1	1.46

S4.f) Inclusion of data from early pandemic

Figure S5 shows reported death rates per million people (averaged over prediction period, 01 Apr-30 Sep 2021) against collective responsiveness α estimated from 31 Dec 2019-31 Mar 2021, i.e. including the 'first wave' dynamics from early in the pandemic, with selected larger countries labelled. Similar to the main analysis (Exhibit 1 of the main manuscript), there is a strong negative correlation between responsiveness and subsequent death rate.

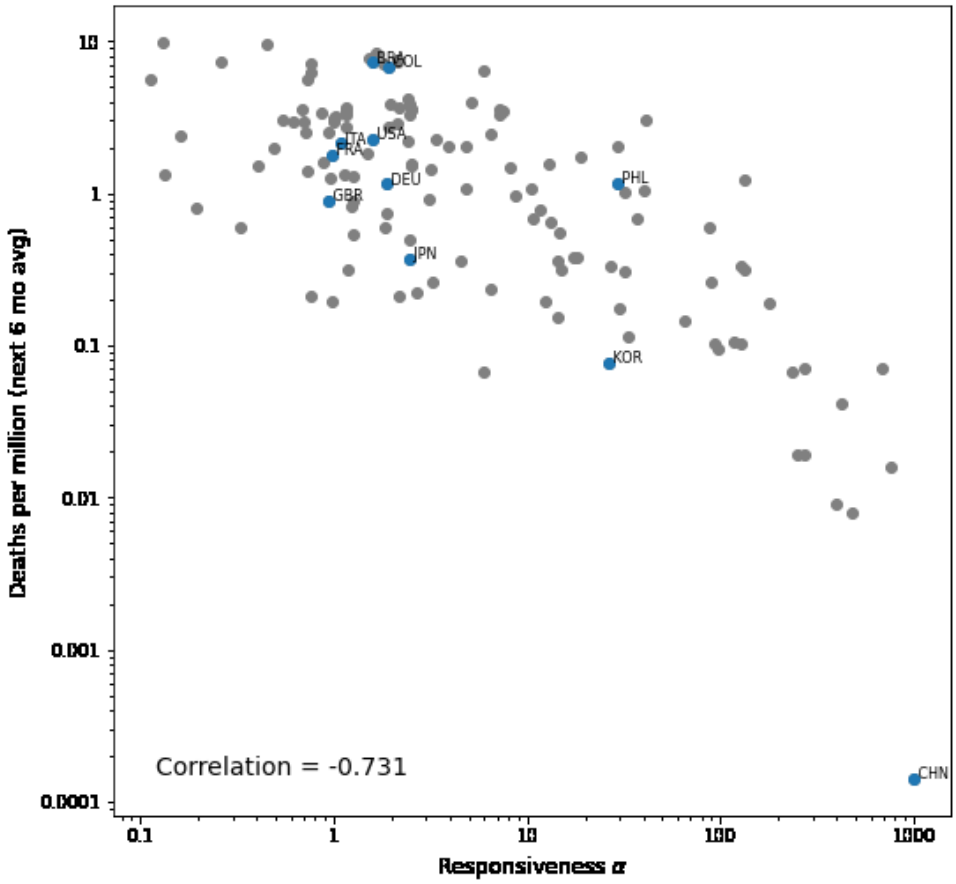


Figure S5. Reported daily deaths per million people (averaged over prediction period, 01 Apr-30 Sep 2021) against estimated collective responsiveness α (estimated from 31 Dec 2019-31 Mar 2021), with selected larger countries labelled. Source: Authors' analysis of data on daily confirmed cases and deaths come from the OWID global COVID-19 database.

S4.g) Use of estimated true infection & death data

Figure S6 shows estimated true death rates per million people (from IHME estimates, averaged over prediction period, 01 Apr-30 Sep 2021) against collective responsiveness α estimated from 01 May 2020-31 Mar 2021, with selected larger countries labelled. As in the main analysis (Exhibit 1 of the main manuscript), responsiveness negatively correlates with subsequent death rates, albeit more weakly (as expected due to dynamically changing under-reporting introducing additional variation in death rates).

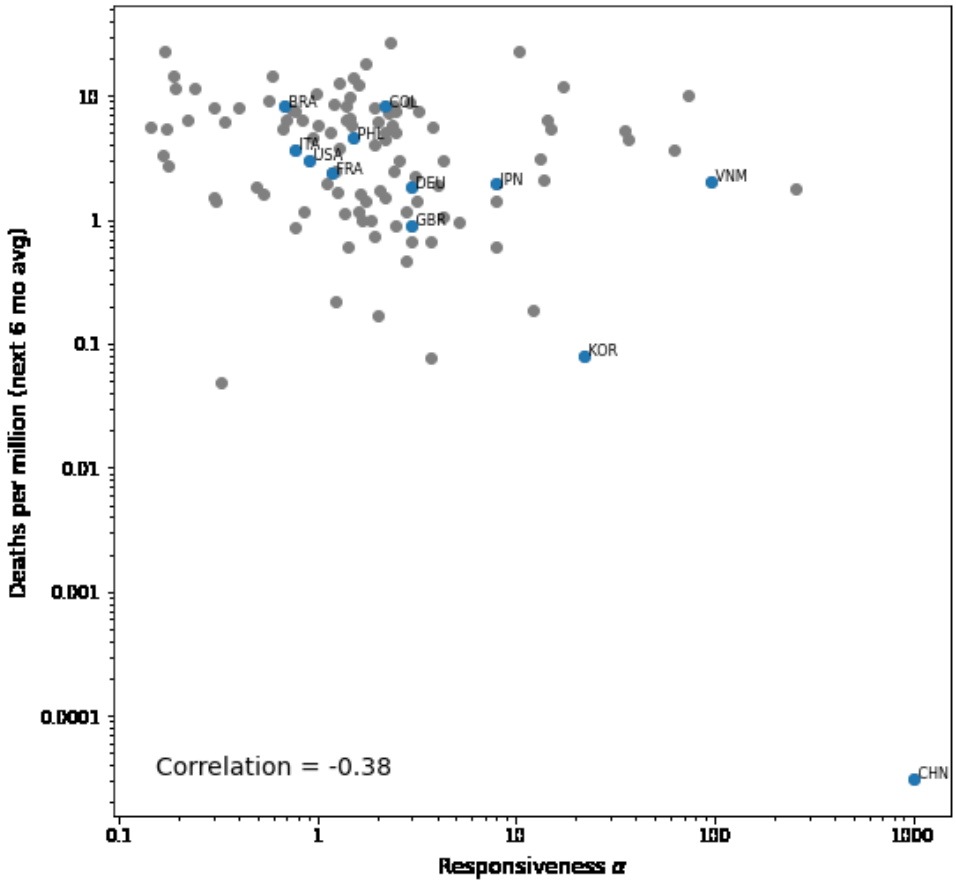


Figure S6. IHME's estimated daily deaths per million people (averaged over prediction period, 01 Apr-30 Sep 2021) against estimated responsiveness α (estimated from 01 May 2020-31 Mar 2021), with selected larger countries labelled. Source: Authors' analysis of data on daily confirmed cases and deaths come from the IHME COVID-19 database.

S4.h) Additional robustness checks for main regression

The following results are additional robustness checks for the main death rate regression (**Exhibit 2** of main manuscript) using different sources or subsets of data. Each shows predictors of cross-country variation in per capita mortality rates per million (reported mortality in all cases except Table S7, which uses estimated true mortality from IHME), averaged over the 180 days after April 1, 2021. In all cases, collective responsiveness remains the primary driver of variation in death rates and the main contributor to model fit. In short, all tables show the same qualitative result: no matter the inclusion criteria or data source, responsiveness is the most important (and often only) predictor of death rates in the prediction period.

Table S6 uses responsiveness α estimated from 31 Dec 2019-31 Mar 2021, including data from early in the pandemic, corresponding to Figure S5 above.

Table S6. Predictors of cross-country variation in mortality rates per capita. Dependent variable: per capita mortality (reported deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.816±0.243	-6.9 (0.000)	0.416	0.29 (0.14-0.60)
Age Multiplier of Mortality (log10)	-0.945±0.313	-2.7 (0.009)	0.058	0.66 (0.51-0.87)
GDP per capita (log10)	-0.429±0.123	-2.6 (0.013)	0.05	0.75 (0.64-0.88)
Hospital beds per thousand	0.020±0.020	0.6 (0.523)	-0.005	1.12 (0.89-1.42)
Initial reproduction number	-0.146±0.082	-1.5 (0.131)	0.012	0.74 (0.54-1.03)
Policy stringency (6 mo. avg.)	0.009±0.006	1.1 (0.285)	0.002	1.23 (0.91-1.65)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	56	**Adj. R^2**	0.542	***F* (p-value)** 12.1 (0.000)

Source: Authors' analysis of data from the OWID global COVID-19 database.

Table S7 uses estimated true (rather than reported) per capita mortality from IHME estimates as the dependent variable, corresponding to Figure S6 above. The responsiveness estimates also use IHME data, and are for the estimation period of May 2020–March 2021.

Table S7. Predictors of cross-country variation in mortality rates per capita. Dependent variable: IHME's estimated per capita mortality (deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.532±0.256	-5.3 (0.000)	0.162	0.44 (0.20-0.96)
Age Multiplier of Mortality (log10)	-0.287±0.123	-2.2 (0.033)	0.022	0.83 (0.70-0.97)
GDP per capita (log10)	-0.206±0.129	-1.9 (0.066)	0.014	0.77 (0.55-1.06)
Hospital beds per thousand	0.024±0.027	0.8 (0.450)	-0.002	1.15 (0.85-1.56)
Initial reproduction number	-0.096±0.075	-1.2 (0.229)	0.003	0.82 (0.60-1.11)
Policy stringency (6 mo. avg.)	0.015±0.004	3.0 (0.003)	0.049	1.62 (1.28-2.05)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	95	**Adj. R^2**	0.47	***F* (p-value)** 15.0 (0.000)

Source: Authors' analysis of data from the OWID global COVID-19 database and IHME database.

Table S8 and S9 show regression results excluding countries with different thresholds for excess deaths compared to reported deaths, indicating different levels of reliability in reporting (excess mortality exceeding official deaths by no more than 50% or 25% respectively). Table S10 instead shows regression results including all countries with available data, i.e. including those with excess mortality over 100% more than official deaths.

Table S8. Predictors of cross-country variation in mortality rates per capita, excluding countries with more than 50% excess death. Dependent variable: per capita mortality (reported deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.549±0.220	-4.0 (0.000)	0.294	0.33 (0.14-0.79)
Age Multiplier of Mortality (log10)	-0.416±0.361	-0.7 (0.463)	-0.009	0.83 (0.60-1.14)
GDP per capita (log10)	-0.216±0.236	-0.9 (0.366)	-0.003	0.86 (0.63-1.18)
Hospital beds per thousand	-0.010±0.028	-0.2 (0.845)	-0.019	0.94 (0.67-1.31)
Initial reproduction number	-0.146±0.155	-1.0 (0.312)	0.001	0.73 (0.37-1.41)
Policy stringency (6 mo. avg.)	0.014±0.009	1.0 (0.301)	0.002	1.39 (0.93-2.08)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	41	**Adj. R^2**	0.294	***F* (p-value)** 3.9 (0.005)

Source: Authors' analysis of data from the OWID global COVID-19 database.

Table S9. Predictors of cross-country variation in mortality rates per capita, excluding countries with more than 25% excess death. Dependent variable: per capita mortality (reported deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.546±0.232	-3.8 (0.001)	0.299	0.33 (0.13-0.83)
Age Multiplier of Mortality (log10)	-0.292±0.305	-0.4 (0.665)	-0.018	0.88 (0.67-1.15)
GDP per capita (log10)	-0.221±0.222	-0.9 (0.388)	-0.005	0.86 (0.64-1.16)
Hospital beds per thousand	-0.010±0.029	-0.2 (0.856)	-0.022	0.94 (0.66-1.34)
Initial reproduction number	-0.149±0.172	-1.0 (0.336)	-0.001	0.72 (0.35-1.51)
Policy stringency (6 mo. avg.)	0.018±0.011	1.2 (0.258)	0.007	1.49 (0.93-2.37)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	37	**Adj. R^2**	0.277	***F* (p-value)** 3.4 (0.011)

Source: Authors' analysis of data from the OWID global COVID-19 database.

Table S10. Predictors of cross-country variation in mortality rates per capita, including all. Dependent variable: per capita mortality (reported deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.460±0.122	-6.1 (0.000)	0.237	0.39 (0.24-0.64)
Age Multiplier of Mortality (log10)	0.171±0.206	1.0 (0.299)	0.001	1.12 (0.86-1.45)
GDP per capita (log10)	-0.015±0.119	-0.1 (0.895)	-0.006	0.98 (0.75-1.29)
Hospital beds per thousand	0.001±0.024	0.0 (0.983)	-0.007	1.00 (0.76-1.32)
Initial reproduction number	-0.057±0.089	-0.7 (0.463)	-0.003	0.89 (0.62-1.28)
Policy stringency (6 mo. avg.)	0.016±0.005	3.2 (0.002)	0.061	1.57 (1.16-2.11)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	100	**Adj. R^2**	0.38	***F* (p-value)** 11.2 (0.000)

Source: Authors' analysis of data from the OWID global COVID-19 database.

Tables S11 shows regression results excluding countries with 10% or more of the population having been fully vaccinated by the end of the estimation period (31 Mar 2021). Table S12 repeats the analysis excluding countries that are fully or partially vaccinated by 10% at the same time period.

Table S11. Predictors of cross-country variation in mortality rates per capita, excluding countries with 10% or more of the population having been fully vaccinated by the end of the estimation period (31 Mar 2021). Dependent variable: per capita mortality (reported deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.538±0.220	-4.0 (0.000)	0.274	0.35 (0.15-0.81)
Age Multiplier of Mortality (log10)	-0.392±0.355	-0.8 (0.439)	-0.007	0.82 (0.57-1.17)
GDP per capita (log10)	-0.242±0.225	-1.1 (0.268)	0.005	0.83 (0.59-1.17)
Hospital beds per thousand	0.031±0.024	0.7 (0.511)	-0.01	1.21 (0.91-1.62)
Initial reproduction number	-0.190±0.158	-1.3 (0.190)	0.014	0.67 (0.35-1.29)
Policy stringency (6 mo. avg.)	0.015±0.009	1.2 (0.249)	0.007	1.41 (0.95-2.10)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	44	**Adj. R^2**	0.284	***F* (p-value)** 3.9 (0.004)

Source: Authors' analysis of data from the OWID global COVID-19 database.

Table S12. Predictors of cross-country variation in mortality rates per capita, excluding countries with 10% or more of the population having been partially or fully vaccinated by the end of the estimation period (31 Mar 2021). Dependent variable: per capita mortality (reported deaths per million), averaged over the 180 days from 01 Apr 2021 onward.

	Coefficient (± std. err.)	Student's *t* (p-value)	Marginal adj. R^2 [a]	Effect Size (95% CI) [b]
Collective Responsiveness (log10)	-0.582±0.212	-3.8 (0.001)	0.355	0.26 (0.10-0.68)
Age Multiplier of Mortality (log10)	-0.803±0.387	-1.2 (0.225)	0.014	0.65 (0.43-0.98)
GDP per capita (log10)	-0.283±0.185	-0.9 (0.383)	-0.005	0.80 (0.61-1.06)
Hospital beds per thousand	0.048±0.033	0.8 (0.411)	-0.008	1.42 (0.88-2.28)
Initial reproduction number	-0.324±0.146	-1.9 (0.076)	0.064	0.48 (0.25-0.92)
Policy stringency (6 mo. avg.)	0.005±0.014	0.3 (0.762)	-0.023	1.12 (0.62-2.05)
[a] Marginal adj. R^2 = adj. R^2 for full model - adj. R^2 for model excluding this predictor				
[b] Effect size = multiplicative change in 6 mo. avg. daily deaths per million per 1 std. dev. change in predictor				
Countries (*n*)	28	**Adj. R^2**	0.405	***F* (p-value)** 4.2 (0.006)

Source: Authors' analysis of data from the OWID global COVID-19 database.

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