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individual responses, and consequently, worse epidemic outcomes.

# Information and behavioral responses during a pandemic: Evidence from delays in Covid-19 death reports<sup>\*</sup>

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ABSTRACT

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### 1. Introduction

The swift emergence of the Covid-19 global epidemic forced governments to adopt new policies and communication strategies (WHO, 2013, 2020). A particularly important novelty of this epidemic, relative to past outbreaks, was the dissemination of vast amounts of high-frequency (oftentimes daily) information about the prevalence of Covid-19 cases and deaths. Although the implicit assumption is that more informed agents are more likely to take actions to mitigate the spread of the virus, little empirical and theoretical work has focused on understanding the importance of the *accuracy* of this information.<sup>1</sup> In particular, governments across the world have made efforts to collect and communicate information to their citizens. If these government reports indeed matter for behavioral responses,<sup>2</sup> then reliable real-time surveillance systems are paramount not just for tracking the epidemic, but also for managing it. This may be particularly challenging in low-and middle-income countries, where diminished state capacity may impede the collection of accurate instantaneous information.<sup>3</sup>

Providing information is important for managing epidemics, but issues with data accuracy may hinder its

effectiveness. Focusing on Covid-19 in Mexico, we ask whether delays in death reports affect individuals'

beliefs and behavior. Exploiting administrative data and an online survey, we provide evidence that behavior,

and consequently the evolution of the pandemic, are considerably different when death counts are presented by

date reported rather than by date occurred, due to non-negligible reporting delays. We then use an equilibrium

model incorporating an endogenous behavioral response to illustrate how reporting delays lead to slower

In this paper, we shed light on the issues of government-provided information and its quality by asking whether the delays with which deaths are reported affect the evolution of the epidemic through their potential impact on behavior. We focus on the Covid-19 outbreak in Mexico, where reporting delays – that is, the time difference between when a death occurs and when it is publicly reported – are measurable and large. Hence, in this setting, daily death reports are not an accurate representation of the state of the epidemic.

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<sup>&</sup>lt;sup>1</sup> In the context of Covid-19, it has been shown that information-focused public policies have been important for managing behavior (Gupta et al., 2020; Briscese et al., 2020).

<sup>&</sup>lt;sup>2</sup> For other contexts, the evidence is mixed. For instance, for HIV in Africa, studies have found large effects of information on behavior (Dupas, 2011; Dupas et al., 2018), while for vaccination in the US, it has been shown to be mostly ineffective (Nyhan and Reifler, 2015; Sadaf et al., 2013). Additional factors seem to mediate individuals' responses to information. For example, Oster (2012) shows non-HIV mortality matters for adopting protective behaviors.

 $<sup>^3</sup>$  We abstract from instances where individuals may make simple adjustments in an attempt to account for data inaccuracies.

Mexico is ideal for studying these issues for at least three reasons. First, the delays in Covid-19 death reports are not only measurable and large, but vary greatly across states.<sup>4</sup> Gutierrez et al. (2020) documents that these delays are correlated with local measures of the capacity of the public healthcare system. The top panel in Fig. 1 depicts these delays at the national level by showing cumulative deaths as reported versus as occurred as well as the distribution of reporting delays in days. Second, Mexican officials routinely present information on confirmed Covid-19 deaths over time, giving salience to the number of *reported* deaths.<sup>5</sup> Lastly, the Mexican government chose a relatively lenient strategy that consisted of mostly optional lockdowns and stay-at-home recommendations, as well as restricting testing to symptomatic individuals seeking medical care. Hence, deaths have been particularly salient as a more reliable measure of the state of the epidemic and behavioral responses have been crucial for its containment.<sup>6</sup>

Exploiting detailed daily data that allow us to separately count reported versus occurred cases and deaths, we begin by showing descriptive correlations. We document that the number of *reported* deaths is a better predictor of the growth in the number of Covid-19 cases than the number of *occurred* deaths. This suggests that individuals may incorrectly make inferences about the risk of contagion by assuming that deaths reported are a good approximation of deaths occurred.

We complement these empirical results by fielding an online survey, where we randomized information about the epidemic. We compare respondents' beliefs regarding the severity of the epidemic and their reported intentions of complying with stay-at-home recommendations between groups that were shown the evolution of Covid-19 deaths by date reported versus the date on which they actually occurred. We find evidence consistent with individuals not fully accounting for reporting delays when adapting their behavior to the perceived risk prevalence.

Lastly, informed by these findings, we develop a simple equilibrium model that allows us to illustrate the impact that reporting delays have on the evolution of the pandemic through their impact on individuals' behavior. In the model, agents split their time at or away from home but risk getting infected when outside the home. Thus, the higher the prevalence of the virus, the higher the incentive to stay home. Agents rely on death reports provided by the government – which they take to be accurate – to form expectations about the current and future prevalence of the disease, updating expectations with each new report.

We calibrate this model to our setting and compare outcomes in a scenario where deaths are reported as occurred relative to a situation where reporting delays follow the empirical distribution we observe in Mexico. Inaccurate information due to reporting delays leads to individuals being slower to adopt protective behaviors and to more severe epidemic outcomes in terms of cases and deaths, with a peak of daily deaths 25% larger (266 additional deaths) than in the model without delays, which implies that eliminating delays in the model would decrease total deaths by 13,289 by day 120 of the epidemic. Moreover, the faster speed of the epidemic induced by slower reactions will tend to generate excessive responses later on, which may exacerbate the associated negative economic impacts.

Overall, our analysis suggests that improving the capacity to convey accurate information matters for how agents respond. Communicating more accurate information can change the full dynamics of the epidemic itself with lower total deaths, smaller activity fluctuations, and less intense outbreaks, which may also imply a better management of hospital capacity.<sup>7</sup> These results seem particularly relevant for settings with weak state capacity, where the reliability of the data has often come into question.<sup>8</sup> In a broader sense, our findings suggest that the accuracy of information – of any kind and at any moment – influences behavior that may lead to substantially different epidemic outcomes.

We contribute to three strands of the growing literature on the economics of Covid-19. First, our paper relates to those that have explored how messages and information affect various outcomes.<sup>9</sup> Akesson et al. (2020) provides different information about Covid-19 infectiousness, finding that individuals who received the larger estimate of contagion risk were actually less likely to report complying with mitigating behaviors. Falco and Zaccagni (2020) shows that reminders matter for stay-at-home compliance, while Breza et al. (2021) finds that messages from healthcare workers reduced mobility. Binder (2020) and Coibion et al. (2020) randomize information on government policies in the US to measure its impact on consumer beliefs and spending. While these studies focus on the effect of receiving information, our paper emphasizes the role of its accuracy.<sup>10</sup> In a similar spirit, Bursztyn et al. (2021) analyzes the role of veracity of messages in the US, but centers on opinion programs in the media.

Second, our paper relates to studies incorporating changes in behavior into dynamic epidemiological models (Fernández-Villaverde and Jones, 2020; Brotherhood et al., 2020). While we favor a simple and parsimonious approach, the novelty in the model we propose consists in explicitly incorporating frictions in behavior that may emerge from misinformed agents.

Lastly, we add to the set of papers focusing on identifying the additional restrictions and challenges that low- and middle-income countries face during Covid-19, including the capacity of the healthcare system, poverty, inequality, and corruption (Gallego et al., 2020; Gottlieb et al., 2020; Loayza, 2020; Monroy-Gómez-Franco, 2020; Ribeiro and Leist, 2020; Walker et al., 2020). We contribute to this line of work by focusing on the potential consequences of issues in conveying reliable real-time information. Given the relationship between reporting delays and state capacity (Gutierrez et al., 2020), this is likely to be an issue for many other low- and middle-income countries.

# 2. Motivating correlations

#### 2.1. Data

The Mexican government provides detailed patient-level information for all recorded Covid-19 cases, with daily updates. We observe the patients' state of residence, when they first sought medical attention for Covid-19 symptoms, the self-reported date for the onset of symptoms (all reports are symptomatic), the result of a Covid-19 laboratory test, and, if applicable, the date of death. With each daily update, patients may transition from unconfirmed to confirmed Covid-19, and from alive to dead.

<sup>&</sup>lt;sup>4</sup> Delays in reporting deaths have been documented across many settings (AbouZahr et al., 2015; Bird, 2015).

<sup>&</sup>lt;sup>5</sup> See, for instance, this government website: https://coronavirus.gob.mx/datos/.

<sup>&</sup>lt;sup>6</sup> See, for example, https://globalhealthsciences.ucsf.edu/sites/ globalhealthsciences.ucsf.edu/files/la\_respuesta\_de\_mexico\_al\_covid\_esp.pdf for details on how information on cases and deaths in Mexico has been interpreted.

<sup>&</sup>lt;sup>7</sup> For example, Gutierrez and Rubli (2020) shows a strong relationship between hospital capacity and increases in in-hospital mortality during the 2009 H1N1 epidemic in Mexico.

<sup>&</sup>lt;sup>8</sup> See, for example, undercounting in India (https://www. nytimes.com/2021/04/24/world/asia/india-coronavirus-deaths.html? searchResultPosition=1) and Ecuador (https://www.nytimes.com/2020/04/ 23/world/americas/ecuador-deaths-coronavirus.html?searchResultPosition= 3), last accessed May 13, 2021.

<sup>&</sup>lt;sup>9</sup> Other mediating factors that the literature has analyzed include sociodemographic characteristics (Papageorge et al., 2020; Knittel and Ozaltun, 2020), political beliefs (Allcott et al., 2020; Baccini and Brodeur, 2020; Barrios and Hochberg, 2020), social capital (Bargain and Aminjonov, 2020; Brodeur et al., 2020; Ding et al., 2020; Durante et al., 2020), and the media (Simonov et al., 2020).

<sup>&</sup>lt;sup>10</sup> Studies have also analyzed the link between risk perceptions and prosocial behavior during Covid-19 (Campos-Mercade et al., 2021; Abel et al., 2021; Brañas-Garza et al., 2020).



(a) Reporting delays: deaths by date reported and by date occurred



(b) Treatment arms in the survey: cumulative deaths by date reported and date occurred

Fig. 1. Delays in death reports in Mexico and the information treatments in the survey. Notes: These plots depict reporting delays in Mexico and the information treatments in the survey. Focusing on the top panel, the plot on the left shows total deaths in Mexico up to September 30, 2020. The solid line corresponds to cumulative death counts based on the date of occurrence, while the dotted line uses the date on which deaths were reported. The plot on the right shows the distribution of delays in death reports measured in days (difference between when a death occurred and when it was reported). These graphs use information provided up to February 11, 2021. In the bottom panel, we show the information treatments. The plot on the left shows cumulative deaths in Mexico based on the date they were reported. The plot on the right shows them by when they actually occurred. Each plot shows data from March 22 to May 15, using information reported up to May 27, 2020. We include the cumulative number of deaths by date reported in Sweden as a reference.

The testing rate for Covid-19 in Mexico has been, by design, one of the lowest.<sup>11</sup> Only symptomatic individuals seeking medical attention are tested. Hence, deaths are arguably more precisely measured than cases. Furthermore, this feature has made deaths more salient in this context.

From the dataset published on February 11, 2021, we compute the number of Covid-19 cases and deaths per state-week according to the date on which they occurred. We restrict to cases from March 14 to September 30, 2020, allowing up to four months for all occurred cases and deaths during this period to be reported. We then recover the number of weekly reported cases and deaths in each state from the changes in the updated database from one week to the next, allowing us to track the number of reported and occurred cases and deaths over time.

## 2.2. Empirical correlations

In a standard model with exogenous behavior, the growth rate of cases is fast at first, but then slows down as more individuals stop being susceptible. Since deaths increase over time, one would expect a negative correlation between the growth rate of cases and the number of deaths, particularly before the epidemic peaks. If agents' behavior responds to the epidemic – with individuals reducing their exposure when prevalence is high – an increase in deaths could then predict a decrease in epidemic growth.

We consider two measures of deaths in our data: the true count (occurred) and a noisy signal (reported). We then estimate correlations conditional on state and time fixed effects (FE), controlling for other features of the epidemic curve, and ask whether the growth rate is more responsive to reported or occurred deaths. In an epidemic model without endogenous behavior and assuming we cannot fully control for the shape of the curve, then one might expect the correlation between growth in cases and occurred deaths to be stronger statistically, due to noise in reported deaths. Likewise, in a model with endogenous behavior, perfectly informed agents should respond to actual deaths and not to reported deaths. However, if agents are not perfectly informed, the growth rate of cases may have a stronger correlation with this imprecise measure instead.

We compute two similar measures of the growth rate of Covid-19 cases for each state-week in our data. We take the percentage change in the number of patients that self-reported having first shown symptoms from week t to t + 1 and the change from week t to t + 2, and estimate:

$$y_{s,t} = \beta_1 \times \ln(\text{Occurred Deaths})_{s,t-1} + \beta_2 \times \ln(\text{Reported Deaths})_{s,t-1} + \lambda_s + \gamma_t + \Pi \mathbf{X}_{s,t} + \varepsilon_{s,t}$$
(1)

<sup>&</sup>lt;sup>11</sup> See, for example, https://ourworldindata.org/coronavirus-testing.

Table 1	
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Correlates of the growth in Covid-19 cases.

	Growth in cases from $t$ to $t + 1$			Growth in cases from $t$ to $t + 2$		
	(1)	(2)	(3)	(4)	(5)	(6)
ln(Reported Deaths) <sub>s,1-1</sub>	-0.0852*** (0.0244)	-0.0821*** (0.0248)	-0.0841** (0.0337)	-0.144** (0.0571)	-0.149** (0.0565)	$-0.172^{**}$ (0.0817)
$\ln(\text{Occurred Deaths})_{s,t-1}$	0.0103 (0.0303)	0.0133 (0.0296)	0.0179 (0.0331)	-0.0254 (0.0713)	-0.0300 (0.0723)	0.0384 (0.0875)
Controls:						
Cases in period t	Yes	Yes	Yes	Yes	Yes	Yes
Growth in cases $t - 1$	No	Yes	Yes	No	Yes	Yes
Growth in deaths $t - 1$	No	No	Yes	No	No	Yes
Observations R-squared $H_0$ : $\beta_1 = \beta_2$	734 0.495 0.063	734 0.497 0.065	700 0.470 0.123	734 0.548 0.324	734 0.549 0.328	700 0.565 0.211

Notes: This table shows how reported and occurred deaths correlate with the growth in Covid-19 cases as reported in the data. Observations are at the state-week level and we show estimates of Eq. (1). Columns 1–3 show the growth rate from week *t* to *t* + 1, while columns 4–6 consider the rate from *t* to *t* + 2. All regressions include state and week fixed effects. Different columns include different additional controls as indicated. Robust standard errors are in parentheses. \*\*\* p < 0.01, \*\* p < 0.05, \* p < 0.1.

where  $y_{s,t}$  is the growth rate of Covid-19,  $\lambda_s$  are state FE,  $\gamma_t$  are week FE,  $\mathbf{X}_{s,t}$  is a vector of epidemic characteristics (such as the number of cases in time *t*), and  $\epsilon_{s,t}$  is the error term.

Table 1 shows the estimates. The dependent variable in the first three columns is the percentage change in the number of self-declared symptoms from week t to t + 1, while the last three columns consider the change from t to t + 2. Columns 1 and 4 include state and week FE and log Covid-19 cases in week t as controls. Columns 2 and 5 add the percent change in new cases between week t - 2 and t - 1. Columns 3 and 6 also control for the percent change in occurred and reported deaths between week t - 2 and week t - 1.

Across specifications, the coefficient associated with the number of reported deaths is negative and significantly different from zero at a high confidence level. Column 1 indicates that when reported deaths double, there is an associated decline of 0.085 points in the growth rate of cases. In contrast, the coefficient associated with the number of occurred deaths is smaller in magnitude and statistically insignificant.

We interpret these correlations as motivating evidence that the growth rate in Covid-19 cases is more responsive to the *noisy* measure of deaths rather than the *actual* number of deaths, even after we control for overall time trends and features of the epidemic. We conjecture that this relationship may be driven by individuals incorrectly inferring Covid-19 prevalence from the number of reported, instead of occurred, total deaths. These empirical correlations motivate the question of whether reporting delays could impact individuals' perceptions and actions.

# 3. Online survey

## 3.1. Survey description and respondents' characteristics

To shed further light on whether delays matter, we conducted an online survey with a randomized informational treatment presenting the evolution of total deaths either by date reported or by actual date of death. The survey ran from May 28 to June 8, 2020, with participants recruited mainly from an ITAM mailing list of faculty, administrative staff, and students (N = 1, 022).<sup>12</sup>

After the initial questions on socioeconomic characteristics and pre-intervention perceptions, respondents were taken to a new screen showing (randomly) one of the two graphs depicted in the bottom panel of Fig. 1. Half (N = 508) were shown the plot on the left, which plots cumulative deaths in Mexico by date reported. The rest were instead shown the plot on the right with cumulative deaths by actual date of

occurrence. Both figures show counts from March 22 to May 15, using data up to May 27. As a reference, we also included the cumulative number of deaths by date reported in Sweden.<sup>13</sup> Both plots contain truthful information, although the plot on the left understates total deaths as occurred by 41% on average, with a difference of up to 2055 deaths on May 11.

Afterwards, participants answered questions on whether they believed the epidemic in Mexico was evolving faster than Sweden, the expected number of total Covid-19 cases and deaths over the whole epidemic outbreak, and how often they expected to leave their home in the following weeks.

Due to the composition of our mailing list, our participant characteristics suggest they belong to a relatively young, educated, and high-income group. Hence, we cannot infer the distribution of beliefs and behavior in the general population. Lastly, although observable differences between our treatment groups are small (Table S2 in the supplementary materials), we control for those characteristics below.

### 3.2. Empirical strategy

We estimate the following equation:

$$y_i = \alpha_0 + \alpha_1 \times [\text{Info By Date Occurred}]_i + \Psi \mathbf{X}_i + v_i$$
 (2)

where  $y_i$  is an outcome for respondent *i*,  $\alpha_0$  is a constant, [Info By Date Occurred]<sub>*i*</sub> is an indicator for receiving the informational treatment that displayed cumulative deaths by actual date of death,  $X_i$  is a vector of observable characteristics (see Table S2), and  $v_i$  is the error term. The coefficient  $\alpha_1$  measures the average difference in the outcome for respondents that were shown the cumulative death toll by date of occurrence relative to those with information by date reported.

We explore three outcomes. First, for simplicity, we construct two binary measures for whether the epidemic is evolving faster in Mexico than in Sweden, depending on whether respondents considered the epidemic evolving "faster" or "much faster" than in Sweden, or strictly considering it "much faster". Second, regarding beliefs about the epidemic's toll, we assign the total number of expected cases and deaths to be equal to the mid-value of the interval chosen by

<sup>&</sup>lt;sup>12</sup> For the full survey questions, see the online repository https://github. com/tgstavares/covidinfo.

<sup>&</sup>lt;sup>13</sup> These data were obtained from https://ourworldindata.org/coronavirus. Sweden followed a similar strategy of relatively light restrictions (Juranek and Zoutman, 2020). The epidemic in Mexico had been compared to Sweden's by government authorities. See, for example, https://twitter.com/HLGatell/ status/1257694745322819586?s=20 and https://www.milenio.com/politica/ ya-aplanamos-la-curva-lopez-gatell, last accessed June 29, 2020.

respondents.<sup>14</sup> Lastly, for social distancing, we use both the number of days respondents expect to leave their homes in four weeks, and an indicator for expecting to leave their house three or more times.<sup>15</sup>

### 3.3. Results

Table 2 shows the results. Each panel corresponds to a different pair of outcomes. Columns 1 and 4 consider the full sample. We then decompose results by respondents' priors based on self-reported knowledge about the total number of cases pre-intervention.<sup>16</sup> Columns 2 and 5 restrict to respondents that reported fewer cases than the truth (low prior) and columns 3 and 6 show those with a larger number (high prior).

Presenting cumulative deaths by actual date of occurrence shifts beliefs towards a perception of a faster spreading epidemic: the fraction of respondents considering that the epidemic was progressing much faster than in Sweden increased by 26 percentage points relative to those shown the plot by date reported. We take this as a first stage result showing that our informational intervention had the expected effect.

Respondents also predict a higher toll of the epidemic when shown deaths by date of occurrence. Individuals who saw the evolution of occurred deaths announced an expected total with, on average, 14% more cases and 11% more deaths. This effect is larger in the low prior subsample. For the self-reported intentions of staying home – which may differ from actual behavior – the results are consistent with information presented by actual date of death having a positive impact. Showing the graph by date of occurrence is associated with a decrease in the number of times people expect to leave their homes (four percentage points or 10% lower probability of leaving three or more times), particularly for the low prior subsample (11 percentage points or 32%).<sup>17</sup>

Notwithstanding the limited statistical power due to our small sample and the relatively small differences in the information provided, we interpret these results as evidence that the delays with which deaths are reported are very likely to affect perceptions about the state of the epidemic and, consequently, compliance with social distancing. These findings also suggest that individuals do not fully incorporate reporting delays when forming beliefs about the epidemic, even in our highly educated sample. We proceed incorporating these insights into an equilibrium model.

#### 4. Model of equilibrium behavior

We present an equilibrium model to illustrate the impact of reporting delays on epidemic dynamics through the endogenous behavioral response of agents. We follow Greenwood et al. (2019) and Brotherhood et al. (2020), with Covid-19-specific compartments as in Fernández-Villaverde and Jones (2020). The structure is purposely simple and allows for standard extensions.<sup>18</sup> The main difference is how agents update expectations about Covid-19 prevalence . Given a prior, agents form plans about consumption and leisure over their life-cycle. The government informs about deaths, which agents take as accurate.<sup>19</sup> Agents then update their prior in order to rationalize the number of deaths as reported by the government. This also changes their planning over economic decisions in the remainder of their life-cycle, thus affecting the dynamics of the epidemic.

*States.* Each period represents one day in discrete time. There is a continuum of ex-ante identical agents that can spend time at and away from home. Let *j* be an agent's health status. The initial state *j* = *s* (**susceptible**) is never infected. Being outside may lead to *j* = *i* (**infected**). Infected agents can contaminate susceptible ones (with a uniform mixing contact rate). With probability  $\gamma$  contagiousness ceases and a *j* = *c* (**recovering**) process follows. Agents exit this state with probability  $\theta$ , and a share  $1 - \delta$  become *j* = *r* (**recovered**), and a share  $\delta$  become *j* = *d* (**dead**). We assume that recovered agents are permanently immune. The future is discounted at rate  $\beta$ .

*Utility and hours.* Each agent is endowed with a single unit of labor every period, divided into work/leisure hours *n* outside and hours at home h = 1 - n. Flow utility is derived from hours outside and at home according to  $u(n, h) = \log n + \lambda_h \log h + b$ , where *b* captures the benefit of remaining alive over being dead, which delivers a normalized utility of zero. Hence:

$$u(n) = \log n + \lambda_h \log (1 - n) + b.$$

(i) =

*Infections.* Susceptible agents are at risk of infection when spending hours outside the home. The probability of getting infected  $\pi$  is assumed to be proportional to the time spent outside the home *n* and a belief about the aggregate transmission risk  $\tilde{\Pi}_t$  that is allowed to be different from the real transmission risk  $\Pi_t$ :

$$\pi\left(n,\tilde{\Pi}_{t}\right)=n\tilde{\Pi}_{t}.\tag{3}$$

*Value functions.* We assume that hours outside are constrained to  $\bar{n} < 1$  for infected and recovering individuals. Value functions are then given by:

$$V(s,t) = \max_{n \in \{0,1\}} \left\{ u(n) + \beta \left( \left[ 1 - \pi \left( n, \widetilde{\Pi}_t \right) \right] V(s,t+1) + \pi \left( n, \widetilde{\Pi}_t \right) V(i) \right) \right\}$$
(value susceptible)

$$\max_{n \in (0,\bar{n})} \{ u(n) + \beta [\gamma V(c) + (1 - \gamma) V(i)] \}$$
 (value infected)

$$V(c) = \max_{n \in (0,\bar{n})} \{ u(n) + \beta ((1 - \theta) V(c) + \theta [(1 - \delta) V(r) + \delta V(d)]) \}$$

(value recovering)

$$V(r) = \max_{n \in \{0,1\}} \{ u(n) + \beta V(r) \}$$
 (value recovered)

$$V(d) = 0$$
 (value dead)

*Laws of motion.* Letting n(j,t) be the optimal hours outside for states j = s, i, c, r, the laws of motion are given by:

$$M_{t+1}(s) = M_t(s) - \pi \left( n(h,t), \Pi_t \right) M_t(s)$$
 (mass susceptible)

 $<sup>^{14}</sup>$  We assigned a value of 3,000,000 for "more than 2,000,000 cases", and 300,000 for "more than 200,000 deaths".

<sup>&</sup>lt;sup>15</sup> We assign a value of 3.5 for the "3-4 times" category, and 5 times for "5+". We show results in Figure S3 using indicators for each of the possible response categories for all our outcomes.

<sup>&</sup>lt;sup>16</sup> The low prior subsample are those who reported a case load lower than 50,000 (47.7%), while the high prior group are those that reported over 50,000 cases (Figure S4). The true number was 56,594 (see https://twitter.com/HLGatell/status/1263264663283908609?s=20, last accessed June 29, 2020).

<sup>&</sup>lt;sup>17</sup> We obtain positive coefficients for the high prior subsample, although not precisely estimated. This may be consistent with a fatalistic response (Akesson et al., 2020). As modeled by Kerwin (2020) for HIV in Africa, perceptions of a higher risk of contagion may lead to low marginal costs of risk-taking behavior, leading individuals to take on more risks in a "rationally fatalistic" response. While we cannot definitely confirm it, our results for the high prior subsample in panel C of Table 2 are consistent with this response and warrant further exploration in the future.

<sup>&</sup>lt;sup>18</sup> Extensions include macroeconomic implications, savings, non-pharmacy initiatives, testing, vaccines, optimal lockdowns, and age and asset heterogeneity (Eichenbaum et al., 2020a,b; Acemoglu et al., 2020; Alvarez et al., 2020; Kaplan et al., 2020).

<sup>&</sup>lt;sup>19</sup> Information could also focus on cases instead of deaths, and the results would follow through. We assume that agents do not perfectly anticipate the frictions associated with how the government provides information. With perfect knowledge, beliefs about prevalence would be accurate even with delays. This is unlikely given the content of the government's press conferences and due to the degree of sophistication required of individuals.

#### Table 2

Estimates of informational treatments on perceptions and behavior

	(1)	(2)	(3)	(4)	(5)	(6)			
	Full sample	Low prior	High prior	Full sample	Low prior	High prior			
Panel A: Pandemic's evolution	Compared to Sweden								
	Faster or much	ı faster		Much faster					
Information by date occurred	0.195*** (0.024)	0.169*** (0.035)	0.198*** (0.034)	0.258*** (0.030)	0.220*** (0.044)	0.266*** (0.041)			
Observations R-squared Mean dependent variable	1022 0.077 0.81	488 0.100 0.82	534 0.097 0.80	1022 0.082 0.37	488 0.105 0.39	534 0.101 0.35			
Panel B: Expected toll	Beliefs on full impact of epidemic outbreak								
	Log expected total cases			Log expected total deaths					
Information by date occurred	0.144*** (0.055)	0.203** (0.085)	0.108 (0.072)	0.108** (0.055)	0.173** (0.082)	0.0414 (0.074)			
Observations R-squared Mean dependent variable	1022 0.020 12.97	488 0.044 12.88	534 0.028 13.05	1022 0.014 10.81	488 0.035 10.76	534 0.022 10.85			
Panel C: Social distancing	Number of times expected to leave the house in 4 weeks								
	Number of times			Three or more times					
Information by date occurred	-0.0442 (0.093)	-0.286** (0.136)	0.154 (0.130)	-0.0368 (0.029)	-0.105** (0.042)	0.0161 (0.041)			
Observations R-squared Mean dependent variable	1022 0.119 2.20	488 0.120 2.14	534 0.150 2.25	1022 0.089 0.35	488 0.096 0.33	534 0.115 0.37			

Notes: This table presents the results from estimating Eq. (2). Each panel corresponds to two different outcome variables constructed from survey responses (see text for details). Columns 1 and 4 show results for the full sample. Columns 2, 5, 3 and 6 stratify the sample by respondents' prior on their knowledge of the number of Covid-19 cases in Mexico up to May 20 into low and high reported cases, respectively. The estimates are the average difference between the responses in the treatment group that received information based on the actual date of death relative to information based on date of reports. Regressions include control variables as listed in Table S2. Robust standard errors are reported in parentheses. \*\*\* p < 0.01, \*\* p < 0.05, \* p < 0.1.

$$\begin{split} M_{t+1}(i) &= M_t(i) - \gamma M_t(i) + \pi \left( n(h,t), \Pi_t \right) M_t(s) \qquad \text{(mass infected)} \\ M_{t+1}(c) &= M_t(c) - \theta M_t(c) + \gamma M_t(i) \qquad \text{(mass recovering)} \\ M_{t+1}(r) &= M_t(r) + (1 - \delta) \theta M_t(c) \qquad \text{(mass recovered)} \\ M_{t+1}(d) &= M_t(d) + \delta \theta M_t(c). \qquad \text{(mass dead)} \end{split}$$

Total population is normalized to 1, so that:

 $1 = M_t(s) + M_t(i) + M_t(c) + M_t(r) + M_t(d), \quad \forall t.$ 

Aggregate probability of infection. The (instantaneous) Poisson rate of infection is given by:

$$\hat{\Pi}_t = \Lambda n(i,t) M_t(i), \tag{4}$$

where  $\Lambda$  is the biological transmissibility of the disease. Hence:<sup>20</sup>

$$\Pi_t = 1 - \exp\left(-\hat{\Pi}_t\right) = 1 - \exp\left[-\Lambda n\left(i,t\right)M_t\left(i\right)\right].$$
(5)

Information and priors. Agents accurately know all the parameters of the model, but are unaware of the initial mass of infectious individuals  $M_0(i)$ , forming a prior  $\tilde{M}_0(i)$ , which may be different from the truth. In this case, forecasts of the epidemic dynamics made by agents will be biased. In particular, given a history of labor supply  $\{n(s,t), n(i,t)\}_{t=0}^{t'} \equiv \{n(s)^{t'}, n(i)^{t'}\}$  for any  $t' \ge 0$ , the following probabilities emerge as potentially different:

$$\Pi_{t'} = 1 - \exp\left[-\Lambda n\left(i, t'\right) M_{t'}\left(i; n\left(s\right)^{t'}, n\left(i\right)^{t'}\right)\right]$$
(6)

$$\tilde{\Pi}_{t'} = 1 - \exp\left[-\Lambda n\left(i, t'\right) \tilde{M}_{t'}\left(i; n(s)^{t'}, n(i)^{t'}\right)\right],$$
(7)

where  $M_{t'}$  and  $\tilde{M}_{t'}$  are obtained from substituting  $\left\{n(s)^{t'}, n(i)^{t'}\right\}$  in the laws of motion (mass susceptible) and (mass infected) using, respectively,  $M_0$  and  $\tilde{M}_0$ , from t = 0..., t'.

# 4.1. Definition of an equilibrium

A belief-biased equilibrium in this economy with a mass of agents at time  $t' \ge 0$  of  $M_{t'}(j)$ , j = s, i, c, r, d consists in a sequence of infection probabilities  $\{\Pi_t\}_{t=t'}^{\infty}$  and  $\{\tilde{\Pi}_t\}_{t=t'}^{\infty}$ , initial beliefs  $\tilde{M}_{t'}(j)$ , and hour allocations  $\{n(j,t)\}_{t=t'}^{\infty}$  for each  $j \in \{s, i, c, r\}$ , such that:

- 1. given  $\tilde{M}_{t'}(j)$  and  $\{\tilde{H}_t\}_{t=t'}^{\infty}$ , n(j,t) solves the values in (value susceptible)–(value recovered);
- 2. given  $\{n(j,t)\}_{t=t'}^{\infty}$ , the resulting laws of motion from (mass susceptible)–(mass dead) using  $M_{t'}(j)$  are consistent with  $\{\Pi_t\}_{t=t'}^{\infty}$ ,
- 3. given  $\{n(j,t)\}_{t=t'}^{\infty}$ , the resulting laws of motion from (mass susceptible)–(mass dead) using  $\tilde{M}_{t'}(j)$  are consistent with  $\{\tilde{\Pi}_t\}_{t=t'}^{\infty}$ .
- 4.2. Model analysis

The static solution for hours outside is given by:

$$n^* = \arg \max_{n \in (0,1)} \{u(n)\} = \frac{1}{1 + \lambda_h}.$$

This also corresponds to hours outside for fully recovered individuals,  $n(r,t) = n^*$ . Assuming that the maximum hours outside for infected and recovering individuals is capped by the static optimal, these individuals will supply:  $n(i, t) = n(c, t) = \bar{n} < n^*$ .

This then yields closed-form solutions:

$$V(r) = \frac{u(n^*)}{1-\beta}$$
$$V(c) = \frac{u(\bar{n}) + \beta\theta(1-\delta)V(r)}{1-\beta(1-\theta)}$$

<sup>&</sup>lt;sup>20</sup> Given an instantaneous Poisson rate of infection  $\hat{H}$ , the probability of infection within  $\bar{t}$  time is given by an exponential distribution with  $Prob(t < \bar{t}) = 1 - \exp(-\hat{H}\bar{t})$ . For a single period,  $\bar{t} = 1$ , we then have that  $Prob(t < 1) = 1 - \exp(-\hat{H})$ .

E. Gutierrez et al.

$$V(i) = \frac{u(\bar{n}) + \beta \gamma V(c)}{1 - \beta (1 - \gamma)}$$

As for susceptible agents, first order conditions imply:

$$\begin{aligned} \frac{\partial u(n)}{\partial n} &= \beta \frac{\partial \pi \left(n, \widetilde{H}_{t}\right)}{\partial n} \left(V\left(s, t+1\right) - V\left(i\right)\right) \\ \Rightarrow \quad \frac{1}{n} - \frac{\lambda_{h}}{1-n} &= \beta \widetilde{H}_{t} \left(V\left(s, t+1\right) - V\left(i\right)\right), \end{aligned}$$

That is, the marginal benefit of spending hours outside is equated with the discounted expected marginal cost of being infected in utility units. It is also easy to see that in this environment, V(s, t + 1) > V(i) for any  $\pi(n, \tilde{\Pi}_t) > 0$ . This implies that hours supplied by susceptible individuals are  $n(s, t) < n^*$  for any  $\tilde{\Pi}_t > 0$ , and moreover, the larger is the perceived risk of transmissibility  $\tilde{\Pi}_t$ , the lower is the supply of hours outside the home.

### 4.3. Update of beliefs based on deaths reported by the government

If agents do not receive information during the course of the epidemic, the equilibrium outcome from the definition in Section 4.1 follows and the epidemic runs its course based on agents' behavior and potentially misspecified beliefs. But if the government provides information on current deaths  $D_t \neq \tilde{M}_t^{prior}(d)$ , then agents update their information about prevalence in order to match their forecast with what the government is announcing. Agents will then get an updated set of beliefs for the masses at t given by  $\tilde{M}_t^{posterior}(j)$  for each j = s, i, c, r, d, where now  $D_t = \tilde{M}_t^{posterior}(d)$ . This new information consists in an unexpected ("MIT") shock to update beliefs that will change behavior plans until the end of the epidemic according to the definition of the equilibrium.

# 4.4. Delays in collection of deaths

Due to physical constraints in data collection, the government may actually provide biased information about the current level of deaths  $D_t \neq M_t(d)$ , such that:

$$D_{t} = D_{t-1} + f\left(\Delta M_{t}(d), \Delta M_{t-2}(d), \dots, \Delta M_{1}(d)\right) < M_{t}(d)$$

where the function *f* captures that the government identifies the previous periods' new deaths with delays. Under these conditions, agents update their prior into a wrong posterior, that is,  $D_t = \tilde{M}_t^{posterior}(d) \Rightarrow \tilde{M}_t(j) \neq M_t(j)$  for j = s, i, c, r, d.

#### 4.5. Simulation of an epidemic with delays in deaths reported

Given a parametrization and specification of delays, we numerically simulate the model following the definition of the equilibrium in Section 4.1 using value function iteration.

*Calibration.* The discount factor  $\beta = 0.98^{1/365}$  is set to capture a 2% annual interest rate. Parameters associated with infectiousness, resolving, and death rates are calibrated in order to target standard findings from the medical literature (Bar-On et al., 2020). The remaining parameters are targeted to closely follow features of the Mexican economy. We assume that the initial population is 120 million and the time zero number of infected are 120 individuals (0.0001% of the total population). We use Mexican time use surveys to calibrate the parameter  $\lambda_h$  by targeting an expenditure of 36% of available hours in activities outside the home before the outbreak. The parameter *b* captures a drop in total hours outside the home during the epidemic of 50% as suggested by Google Mobility data. Lastly, the baseline contagion rate parameter *A* is set to generate a basic reproduction number of two (Marioli et al., 2020). Table S3 summarizes these parameters.

*Reporting delays.* We capture the distribution of the difference in days between deaths as reported and deaths as occurred as given in the data for the whole country. Specifically, we use the following formula:

$$D_{t} = D_{t-1} + p_{t} \Delta M_{t} (d) + \dots + p_{t-60} \Delta M_{t-60} (d)$$

where the coefficients  $\{p_t, \ldots, p_{t-60}\}$  capture the same density as what we observe in a histogram of the data. This effectively allows for a delay of up to two months between when a death occurred and when it was reported. The distribution of delays shows an average of 8.6 days and a standard deviation of 11.5 days (see Fig. 1).

*End of the epidemic.* We allow for the introduction of a vaccine, which is expected ex-ante. After becoming available at time  $t^{vacc} = 350$  days, transmissibility immediately becomes zero.

#### 4.6. Results

Fig. 2 compares the epidemic dynamics in a model without delays (solid line) relative to one with delays as described above (dashed line). Delays lead to a faster progression of the epidemic, implying 25% higher daily deaths at the peak (266 additional deaths) and 3,706 additional total deaths by the end of the outbreak (Table S4). This implies that eliminating delays would decrease total deaths by 13,289 at day 120 of the epidemic. Furthermore, the slow adjustment of hours at the onset contributes to a higher prevalence at the peak, jumping from 0.67% to 0.90% of the population. Once the government informs about deaths, agents realize that the spread of the disease is serious and thus adjust their behavior more abruptly. This causes a larger collapse of hours spent outside than in the case without delays. Relative to prepandemic levels, hours away from home dip up to 46% without delays, but 50% with delays.

The bottom row in Fig. 2 plots how deaths are reported and how they occurred, as well as the difference between the true prevalence of infected individuals and the corresponding biased belief. The first graph shows how beliefs about the mass of the infected population are consistently different from the truth. In particular, when the epidemic starts, this leads to an underestimation of the severity of the disease, and therefore a lower behavioral response from agents.<sup>21</sup>

*Extreme cases of delays.* Aggregate delays mask large variations at finer geographical levels (Gutierrez et al., 2020). Taking the extreme cases among the 32 states in Mexico, delays in the state of Tamaulipas are on average four times larger than in Querétaro (Figure S6). To highlight our effects, we simulate the model under the same parameters as before, but using a delay distribution modeled after each of these states. We, again, allow for delays of up to 60 days.<sup>22</sup>

Fig. 3 shows the results. The epidemic progresses much faster when delays are larger, as in Tamaulipas. Agents are slower to react to the progression of the disease, which contributes to a larger peak, with a share of 1.06% of the population infected relative to 0.73% if delays occur as in Querétaro (Table S5). Due to the acceleration in contagion, peak deaths increase to 1522 in the case of large delays, which is 38% more than in the case of small delays. By the end of the epidemic, the additional deaths relative to the case without delays are more than three times greater with large delays than with shorter delays

<sup>&</sup>lt;sup>21</sup> Our results are consistent with the predictions generated by standard epidemiological models. For example, an SIR model based on Capistran et al. (2021) would predict 70,000 deaths by day 120 of the epidemic as opposed to 67,000 in our baseline model. However, using data as reported to estimate the model would generate only 51,000 deaths by day 120 (Gutierrez et al., 2020). Furthermore, since SIR models fix behavior exogenously, analysis on how the epidemic may change due to frictions in data reports cannot be investigated. <sup>22</sup> If delays are larger than that in Tamaulipas, then we may be undercounting deaths. However, this is unlikely given the delay distribution. Furthermore,

ing deaths. However, this is unlikely given the delay distribution. Furthermore, the model only uses the distribution of delays (which should be the same when undercounting deaths) and not the actual death count.



Fig. 2. Simulation results of behavioral model with and without delays in death reports. Notes: These graphs show the simulation results from the model by computing the equilibrium as defined in Section 4.1. The top row shows results for a situation without reporting delays (solid line) and with delays calibrated to the Mexican data (dashed line). The plots in the top panel show the mass of susceptible individuals, total deaths, daily number of deaths, and hours supplied outside the home. Focusing on the bottom row, the plot on the furthest left shows the beliefs agents have about the mass of infected individuals over time from the onset of the epidemic. We then show the evolution of delays, and both total and daily deaths as occurred and as reported in the scenario with reporting delays.



Fig. 3. Simulation results of behavioral model with and without delays in death reports: Small vs. large delays. Notes: These graphs show the simulation results from the model by computing the equilibrium as defined in Section 4.1. The top panel and the leftmost plot in the bottom panel show results for a situation without reporting delays (solid line), with short delays as in Querétaro (short-dashed line), and large delays as in Tamaulipas (long-dashed line). We show the mass of susceptible individuals, total deaths, daily number of deaths, and hours supplied outside the home. The last two plots in the bottom row compute the forecast error of the mass of infected individuals for the short and long delays scenarios as well as the reporting delays in each case.

(5,440 vs. 1600), or in absolute terms, reducing delays in Tamaulipas to the level observed in Querétaro lowers the toll of total deaths by the 120th day by 15,644, or by 20,802 if delays are completely eliminated.

The last two plots in Fig. 3 help explain the difference in epidemic dynamics between both extreme cases. The next-to-last panel shows

how delays evolve, with Querétaro stabilizing at around three days while Tamaulipas stabilizes at 14 days. In the last panel, we show that at the beginning of the epidemic, agents would underestimate the prevalence of the infected population by almost a full log point (-63%) in the presence of large delays relative to a 0.4 log point (-33%) in the case of shorter delays.

These extreme examples further suggest that tackling the issue of delays may require more localized efforts. For instance, improving death registry in Querétaro may be easier than in Tamaulipas, where delays are so much larger, which could suggest that a more cost-effective intervention in this state would require using statistical prediction methods to adjust the data for delays.

#### 5. Conclusion

The Covid-19 pandemic has been a large experiment on the use of high-frequency and detailed data as a policy lever during a public health shock. The extent to which disseminating information may help mitigate the effects of an epidemic depends largely on its accuracy and reliability, and how individuals may update their priors and respond. In settings with low state capacity, collecting accurate real-time information may be particularly challenging. In this paper, we showed that delays in death reports during Covid-19 are sizeable in Mexico and affect individual beliefs and behavior, leading to more severe epidemic outcomes.

From a policy perspective, our results highlight the importance of accurate real-time information, or at least being clear about the shortcomings of the available data. Increasing collection efforts may be very costly, and while government resources during a pandemic may be better spent on other palliative measures, our findings suggest that simple, low-cost interventions – such as using statistical prediction techniques to correct for delays – may improve outcomes.<sup>23</sup> Overall, our empirical and simulation results underscore the role of accuracy when using information as a public health tool for mitigating an epidemic.

#### Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

#### Data availability

Data will be made available on request.

#### Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.jdeveco.2021.102774.

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<sup>&</sup>lt;sup>23</sup> In that scenario, there may be additional considerations, such as a preference to exaggerate if concerns about public health are larger than economic ones (de Véricourt et al., 2021). However, this may backfire if individuals are rationally fatalistic in their responses (Akesson et al., 2020).

#### E. Gutierrez et al.

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