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Title: Seasonality and uncertainty in COVID-19 growth rates

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12   **Abstract**: The virus causing COVID-19 has spread rapidly worldwide and threatens

13   millions of lives. It remains unknown if summer weather will reduce its continued

14   spread, thereby alleviating strains on hospitals and providing time for vaccine

15   development. Early insights from laboratory studies of related coronaviruses predicted

16   that COVID-19 would decline at higher temperatures, humidity, and ultraviolet light.

17   Using current, fine-scaled weather data and global reports of infection we developed a

18   model that explained 36% of variation in early growth rates before intervention, with

19   17% based on weather or demography and 19% based on country-specific effects. We

20   found that ultraviolet light was most strongly associated with lower COVID-19 growth

21   rates. Projections suggest that, in the absence of intervention, COVID-19 will decrease

22   temporarily during summer, rebound by autumn, and peak next winter. However,

23   uncertainty remains high and the probability of a weekly doubling rate remained >20%

24   throughout the summer in the absence of control. Consequently, aggressive policy

25   interventions will likely be needed in spite of seasonal trends.

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28   **Main Text:**

29   Novel Coronavirus Disease 2019 (COVID-19) is causing widespread morbidity and mortality

30   throughout the world (*1, 2*). The SARS-CoV-2 virus responsible for this disease has infected

31   over 2.2 million people when this article went into review (*3*). Much of the world is

32   implementing non-pharmaceutical interventions, including preventing large gatherings,

33   voluntary or enforced social distancing, and contact tracing and quarantining, in order to prevent

34   infections from overwhelming healthcare systems and exacerbating mortality rates (*2, 4*).

35   However, these interventions risk substantial economic damage and thus decisionmakers are

36   currently developing plans for lifting them. Consequently, improved forecasts of COVID-19

37   risks are needed to inform decisions that weigh risks to both human health and economy (*2*).

38   One of the greatest uncertainties for projecting future COVID-19 risk is how weather will

39   affect its future transmission dynamics. SARS-Cov-2 might be particularly sensitive to weather

40   because it survives longer outside the human body than other viruses (*5*). Rising temperatures

41   and humidity in the northern hemisphere summer could reduce SARS-CoV-2 transmission rates

42   (*6-8*), providing time for healthcare system recovery, drug and vaccine development, and a return

43   to economic activity. Simultaneously, the southern hemisphere is entering winter, and we do not

44   know if winter weather will increase COVID-19 risks, especially in developing countries with

45   reduced healthcare capacity. Early analyses of COVID-19 cases predicted that high temperatures

46   would reduce transmission during the summer (*9-11*). These predictions have been widely

47   reported in mainstream media and are informing decisions about relaxing control efforts soon.

48   However, these analyses relied on the early stages of viral spread before the epidemic had

49   reached warmer regions and thus potentially conflated weather with initial emergence and global

50   transport.

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51   We estimate how weather affects COVID-19 growth rate using data through April 13th,

52   2020 by applying methods that improve model predictive accuracy, incorporate uncertainty, and

53   reduce biases. Based on emerging evidence, we developed several *a priori* predictions about how

54   weather, either directly or indirectly via modified human behaviors (e.g., aggregating indoors),

55   affects COVID-19 growth rate. Preliminary research on SARS-Cov-2 (*9, 10, 12*) and related

56   viruses (*8, 13*) predicted that COVID-19 growth would peak at low or intermediate temperatures.

57   However, other coronaviruses demonstrate weak temperature dependence, instead depending on

58   social or travel dynamics (*7*). High humidity also might decrease viral survival, limit

59   transmission of expelled viral particles, or decrease host resistance (*13-15*). Ultraviolet light

60   effectively kills viruses such as SARS-Cov-1 (*16*), and thus sunny days might decrease outdoor

61   transmission or promote immune resistance via vitamin D production (*17*). We also evaluate

62   demographic variables, assuming greater transmission in denser and older (>60) populations.

63   We modeled maximum growth rate of COVID-19 cases to estimate contributions from

64   underlying climate and population dependencies without healthcare interventions (e.g., social

65   distancing). Hence, we restrict analyses to the early growth phase before interventions reduced

66   transmission, but after community transmission began, when the vast majority of the population

67   was still susceptible to this novel virus. We estimated the average maximum growth rate (λ) as

68   the exponential increase of cases ($(\ln(N_t) - \ln(N_0)/t$, where N_t = cases at time, t, and N_0 = initial

69   cases) for the three worst weeks in each political unit (country or state/province depending on

70   available data (*3*)), where t = 7 days (see Supplementary materials for additional periods).

71   Testing and reporting of COVID-19 likely vary across political units. However, estimated

72   growth rates should remain robust to these biases assuming detection probabilities remain

73   constant during the short, one-week estimation period. We restricted analyses to locations with

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74   >40 cases to eliminate periods before local community transmission. Applying these criteria, we

75   used data from 128 countries and 98 states or provinces.

76   We applied Bayesian Markov Chain Monte Carlo methods with uninformative priors to

77   estimate parameters. We obtained daily infection data from (*3*) and 3-hour weather data from the

78   ERA5 reanalysis for the 14 days preceding case counts consistent with the 1-14 day infective

79   period (*18*). We used fine-scaled weather data rather than long-term climatic monthly means to

80   model observed weather-outbreak dynamics. Weather data was weighted by population size in

81   each 0.25° grid cell within each political unit to capture the weather most closely associated with

82   outbreaks in population centers. We used leave-one-out cross-validation to choose the best

83   models, which ranks model on predictive accuracy on excluded data. We included a random

84   country effect to account for differences in national control response times, health care capacity,

85   testing rates, and other characteristics intrinsic to country of origin.

86   The best model for predicting maximum COVID-19 growth rate predicted 36% of the

87   variation in COVID-19 growth rates (Fig. 1), and 17% excluding country effects. This model

88   included maximum daily ultraviolet light, mean daily temperature, proportion elderly, and mean

89   daily relative humidity (Fig. 2A). Competing models reflected the same qualitative results and

90   similar parameter estimates (see Supplementary materials). Ultraviolet (UV) light had the

91   strongest and most significant effect of tested meteorological variables on COVID-19 growth

92   (β_{UV} = -0.44, 95% credible intervals (Cis): -0.53, -0.36). Contrary to predictions, temperature

93   positively affected COVID-19 growth rate (β_{temp} = 0.23, 95% CIs: 0.15, 0.32), although this is

94   conditional on accounting for UV in the model (note that temperature and UV are moderately

95   correlated (r=0.75) in our data set and extensive testing was done to ensure coefficients estimates

96   were not an artifact of this correlation; see Supplementary Materials). As expected, relative

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97   humidity decreased growth rates, although not significantly, either by reducing the virus'

98   survival outside humans or reducing airborne transmission (β_{humid} = -0.05, 95% CIs: -0.11, 0.00).

99   Absolute humidity was strongly correlated with temperature (r = 0.88) and thus could be

100   exchanged with temperature with little difference in model performance. Contrary to predictions,

101   the proportion of elderly decreased COVID-19 growth rate ($\beta_{popsize}$ = -0.07, 95% CIs: -0.14, -

102   0.00), most likely due to outbreaks in developed countries with older populations. Population

103   density was not selected in any top model. The model was characterized by equally strong

104   random effects associated with country of origin (Fig. 2B). For instance, Turkey, Brazil, Iran, the

105   U.S., and Spain had the highest growth rates independent of modeled factors, whereas China,

106   Iceland, Burkina Faso, Sweden, and Cambodia had the lowest. The strong negative effect

107   associated with China indicates the effect of early interventions and is accounted for in our

108   model. Notably, while intervention will substantially influence the absolute values of growth

109   (i.e., the intercept in our model), our predictions can still be interpreted to represent relative

110   differences in risk throughout the year.

111   We next explored why earlier studies might have predicted a negative association

112   between temperature and COVID-19. Alone, temperature has a weak, negative effect on

113   COVID-19 growth rate in our model, which becomes positive after adding other factors, and in

114   particular, UV. Even with other parameters, temperature negatively affects COVID-19 early in

115   the pandemic (Fig. 3, top). Significant positive temperature dependence emerges by late

116   February following transmission to warmer, high-UV regions of climate space, like Africa (*19*)

117   (see Fig. 3 bottom for filling of climate space). Notably our analysis does not specifically attempt

118   to reproduce previous studies, so differences are expected depending on the details of decisions

119   in other studies. This finding urges caution in estimating climatic niches of new, pandemic

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120   pathogens before they reach an equilibrium distribution with climate. Initial climate associations

121   with viral outbreaks will first correlate with the narrower range of climatic variation found at the

122   emergence site and then in global transportation hubs, rather than reflecting ultimate biological

123   limits on growth and survival. We recognize that future data could alter our predictions further,

124   especially as COVID-19 becomes endemic (*15*). However, less variable model predictions and

125   exposure to the most common global climates by April (Fig. 3) suggest that model predictions

126   might have stabilized at least for now.

127   Using our model, we predicted potential COVID-19 growth rates in the upcoming

128   months relative to a weekly doubling rate (λ=0.1; Fig. 4). Based mostly due to variation in UV

129   and temperature, our model predicts that COVID-19 risk will decline across the northern

130   hemisphere this summer, remain active in the tropics, and increase in the southern hemisphere as

131   days shorten and UV declines (Fig. 4, left and right panels). However, given high uncertainty, a

132   non-negligible risk exists throughout the world for potential outbreaks in summer similar to that

133   observed at the outset of the pandemic (Fig. 4, middle panel, dark blue = 30% probability of

134   λ>0.1*)*. By September, declining daylength steadily increases predicted risks of COVID-19

135   outbreaks in the northern hemisphere until a peak in December-January, while risks decline in

136   the southern hemisphere. Although this model represents our best current estimate, a range of

137   outcomes still remain possible (Fig. 4 middle). Furthermore, these predictions of potential

138   growth need not be realized if appropriate interventions are enacted or a vaccine is developed.

139   The overall conclusion is that although COVID-19 might decrease temporarily during summer,

140   there is still a moderate probability that it is weakly affected by summer weather, and that it

141   could return in autumn and pose increasing risks by winter.

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142   Our predictions were robust to the manifold decisions made regarding data and model

143   structure. We explored the consequences of using different parameter comparisons, the effects of

144   shorter (7-day) time windows for aggregating weather data, different cut-offs for minimum

145   number of cases, varying number of weeks analyzed per political unit, whether we analyze the

146   first or worst weeks following the infection threshold, and if we included weather maxima and

147   minima instead of means and found no qualitative changes to results, with the exception that

148   maximum daily UV during at 14-day interval substantially outperformed the mean

149   (Supplementary materials). We also explored the effect of excluding data from China, which

150   lacked data prior to control measures in many cases, and found similar results.

151   Understanding the true contributions of weather to human pathogens requires combining

152   insights from observational analyses like this one and manipulative experiments that isolate

153   factors under controlled conditions (*5, 12*). Other causal factors correlated with weather variables

154   in our model could have contributed to our findings, including weather-associated human

155   behaviors (e.g., seasonal aggregations for education or religion). Despite initial suggestions that

156   seasonality would strongly control COVID-19, weather only explains 17% of the variation in

157   COVID-19 growth rates. Undescribed factors at the level of political units were just as important

158   as weather (19% of variation), and much of the variation (64%) remains unexplained. Future

159   studies should embed these meteorological insights into epidemiological models that include

160   human demography, movement, sociocultural behaviors, healthcare capacity, and political

161   interventions [e.g., (*2, 4, 15*)].

162   We demonstrate that COVID-19 growth rate increases with reduced ultraviolet light,

163   higher temperatures, and lower relative humidity. We predict that COVID-19 will oscillate

164   between the northern and southern hemisphere, based largely on seasonal variation in UV

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165   radiation and temperature without continuing interventions like social distancing. Despite a

166   possible, but highly uncertain, temporary summer reprieve in the north, COVID-19 is more

167   likely to return by autumn and threaten further outbreaks. The north should take this time to

168   build resilience against future outbreaks, while assisting countries in the tropics and southern

169   hemisphere. Uncertainty remains high, however, so we urge caution when making decisions such

170   as removing societal interventions before more permanent pharmaceutical solutions can be

171   implemented. Overcoming this pandemic will take extensive global collaborative scientific

172   efforts to unravel its biology as well as the continuing resolve of people worldwide adhering to

173   social restrictions.

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213   pneumonia. *New England Journal of Medicine*, (2020).

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221 **Figure 1 Observed and predicted maximum growth rates for COVID-19 along with**

222 **graphical partitioning of model components including (A) weather and demography, (B)**

223 **country effects, and (C) residual variation.** Country effects are shown as the difference in

224 growth rate between the country and the global mean. Only 17% of variation is explained by

225 seasonality, while 19% of variation arises from country specific factors which may include

226 quarantine policies, healthcare, or reporting practices.

227

228 **Figure 2 Median standardized estimates for weather and demographic factors (A) and for**

229 **country effects (B) for best predictive model with 95% credible intervals (light blue).**

230 Country codes in B follow GADM ISO3 conventions.

231

232 **Figure 3 The effect of temperature and UV on COVID-19 growth rate as the pandemic**

233 **spreads to new climates.** Top, early COVID-19 outbreaks (indicated by growth rate

234 proportional to blue symbol area) occurred in a subset of potential temperatures (°C) and

235 ultraviolet light (Joules/m^2) levels possible in a year (background gray-blue gradient) and for that

236 specific time period (red overlay) based on counts of 5-year averages of climate variables.

237 Bottom, Model coefficients and uncertainty through time demonstrates dynamic shifts and

238 stabilization of parameter estimates (50% and 95% credible intervals indicated by colored and

239 gray fills) and illustrates how earlier studies may have detected a negative temperature

240 dependence. Before Feb 24 patterns were dominated by data from China, until large jumps in

241 cases in Iran, Italy, and Japan appear in the data set, providing novel climate space to inform the

242 model and leading to an abrupt change in model coefficients.

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245 **Figure 4: Predicted potential growth rates of COVID-19 by month using best model.**

246 Leftmost column, Potential growth rate relative to weekly doubling time ($\lambda = 0.1$). Red indicates

247 faster than a weekly doubling rate and blue indicates slower rates. Central column indicates the

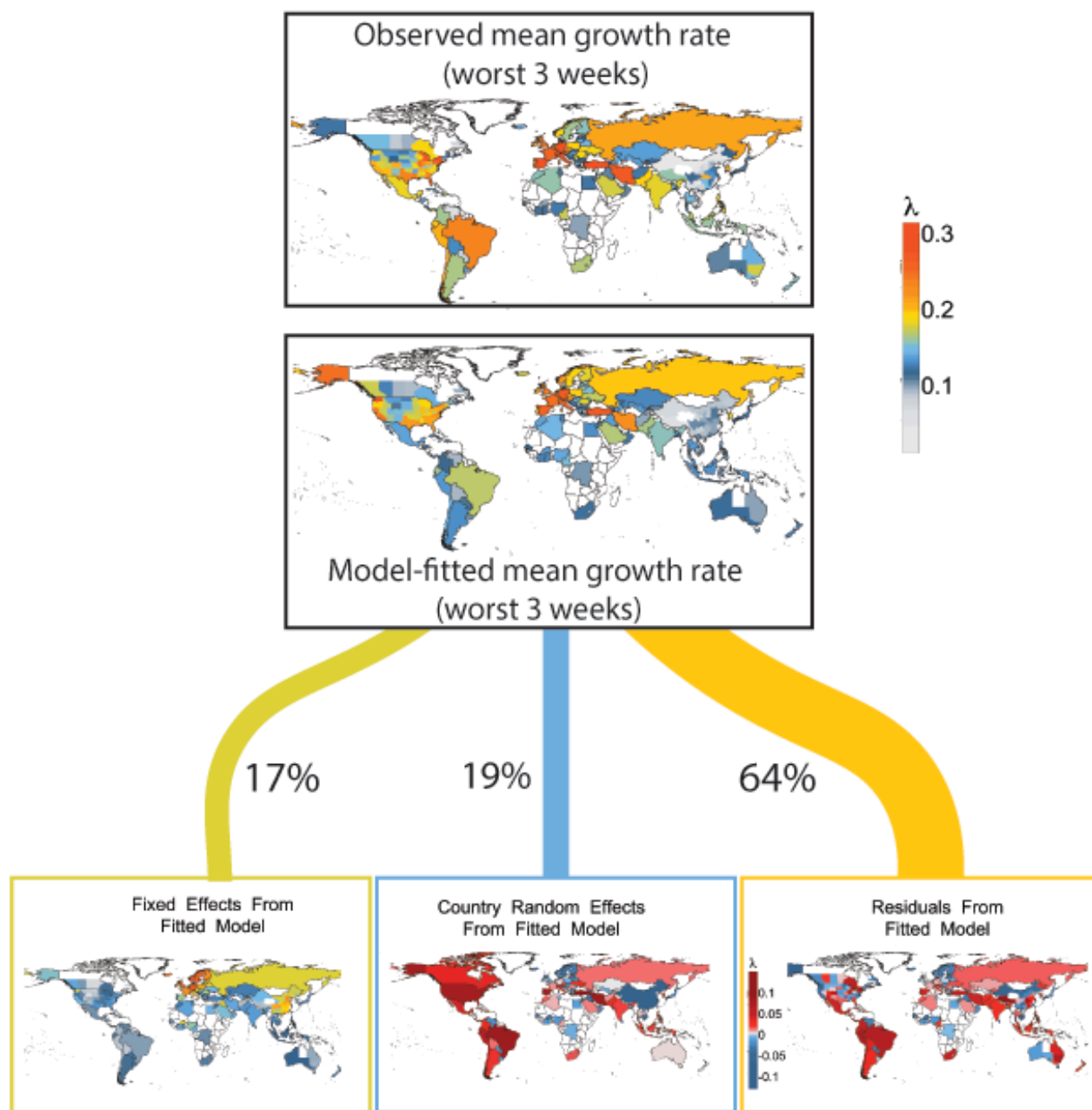248 posterior probability of growth rates exceeding a weekly doubling rate. Rightmost panel

249 indicates which predictor contributes most (based on predictor*coefficient) to COVID-19 growth

250 rate (λ) in each 0.25° cell, with stippling indicating negative contributions.

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251   **Fig. 1**

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256 **Fig. 2A**
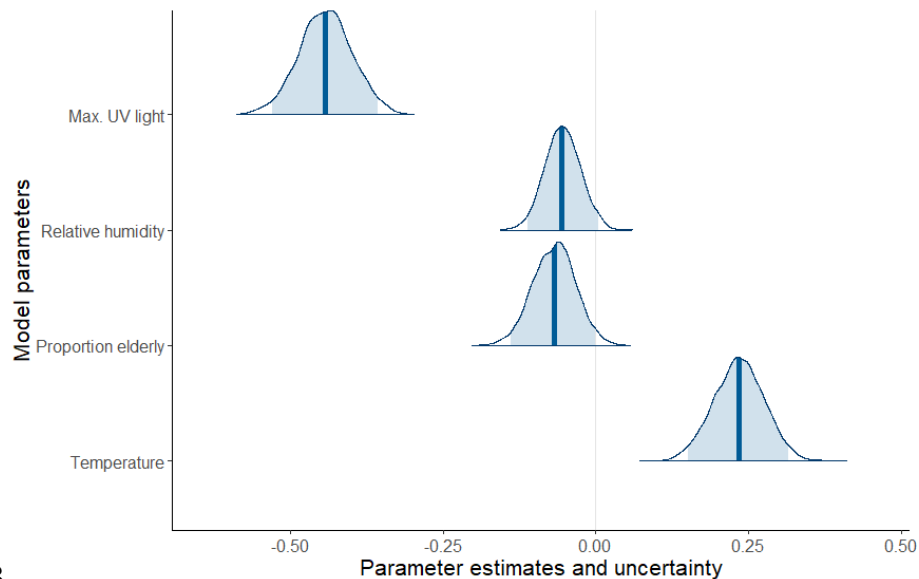
257 **B**
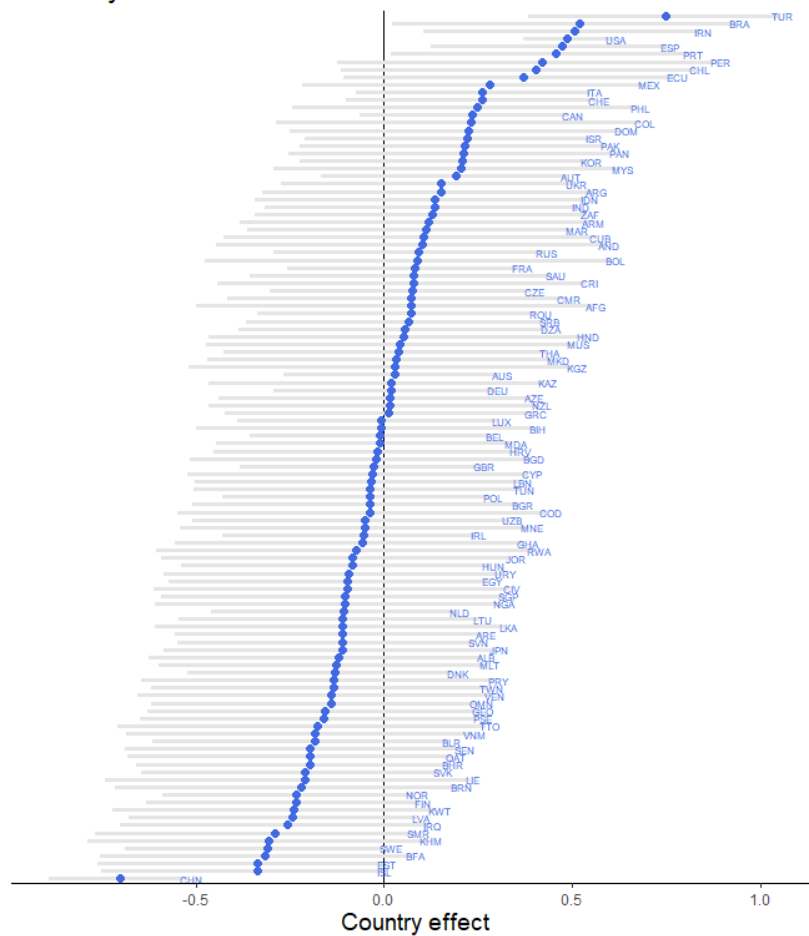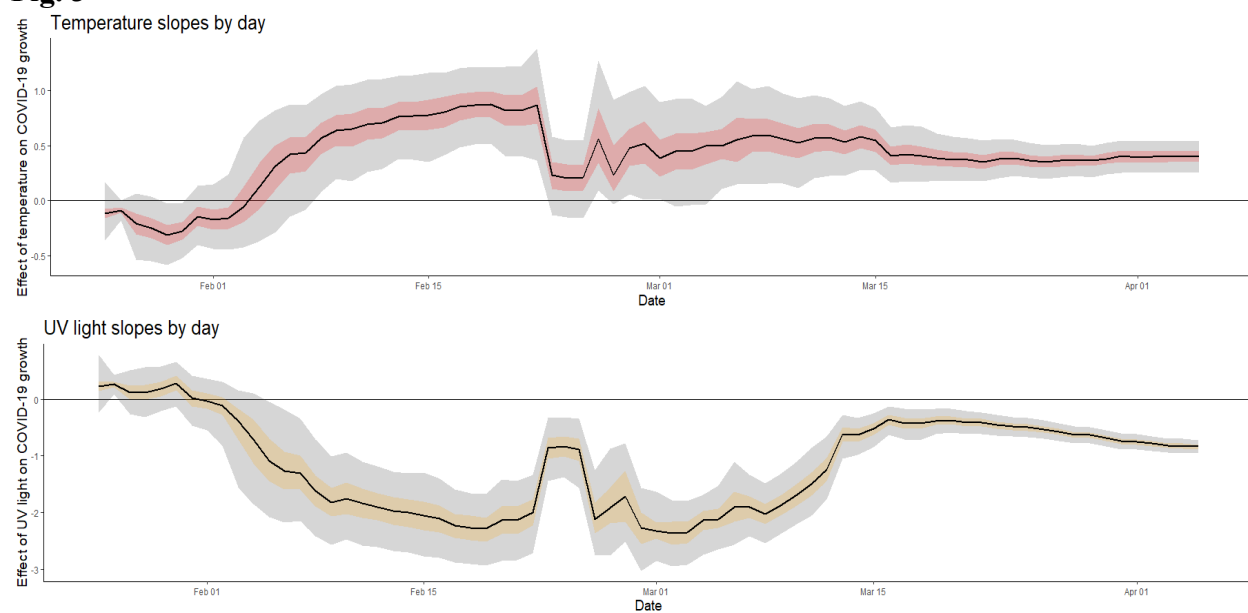

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259   **Fig. 3**

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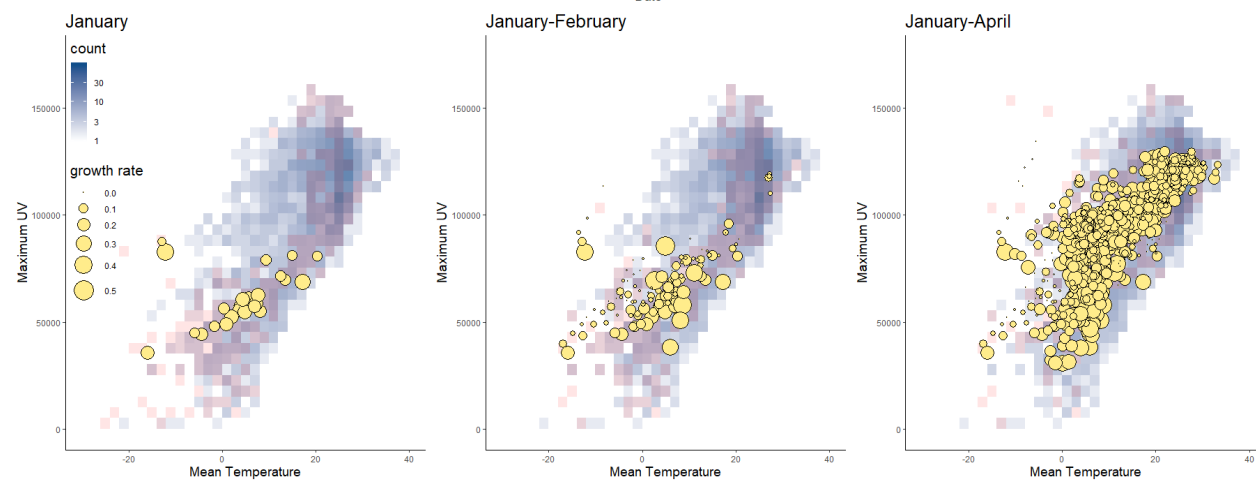
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266 **Fig. 4**

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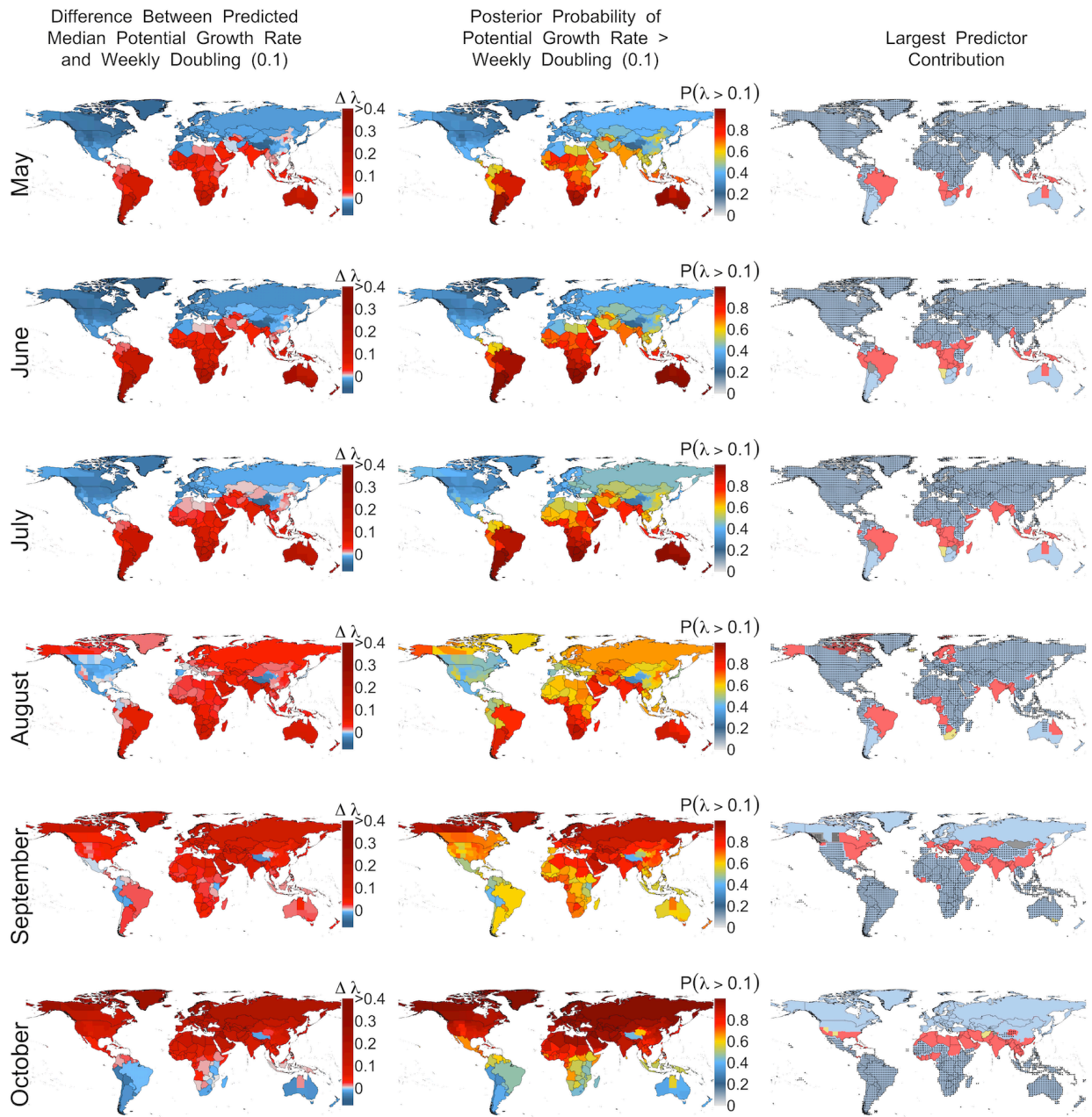
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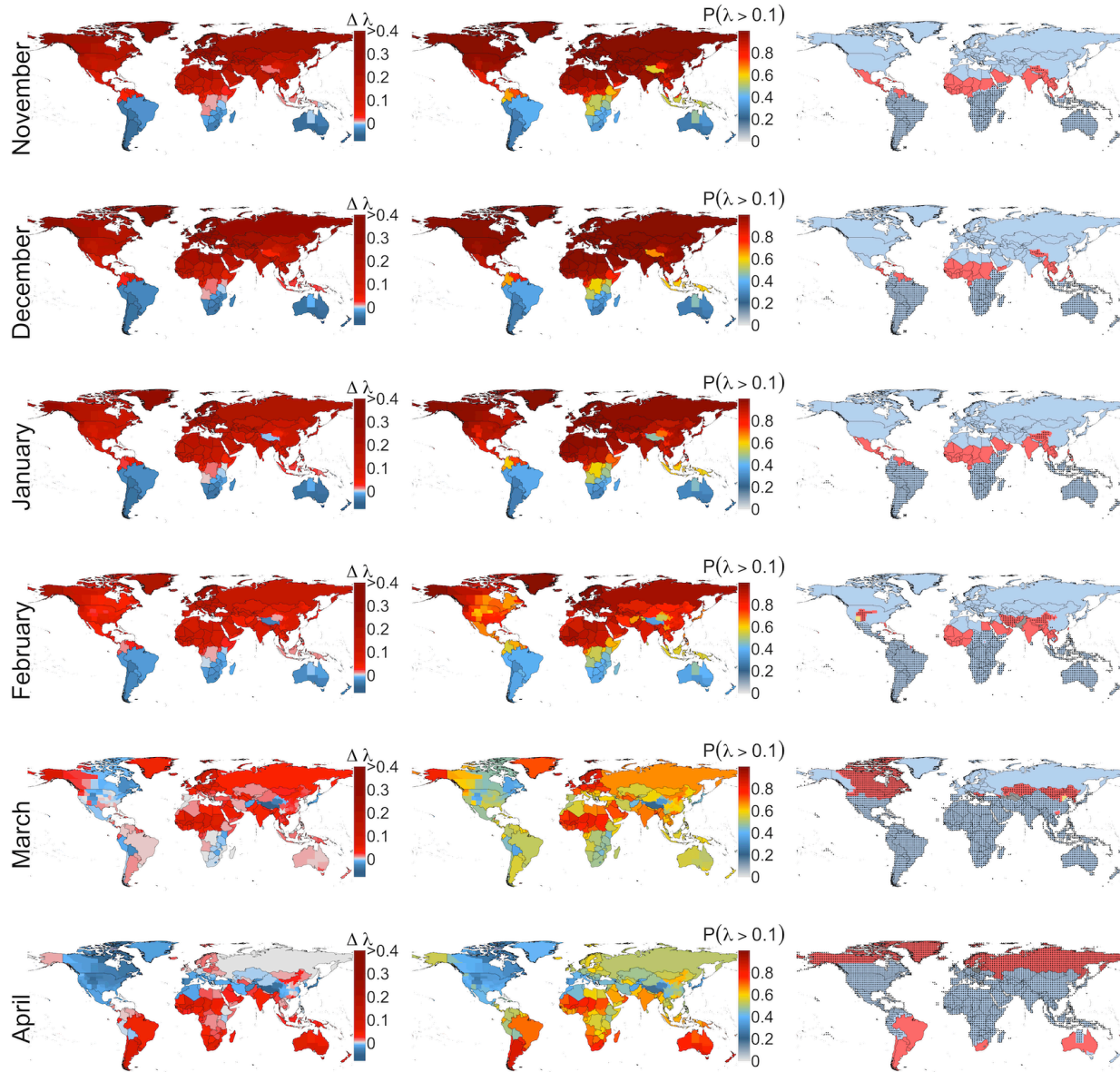
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Methods

Overview

We examined the weekly rate of increase in the number of COVID-19 infections as a function of weather, while controlling for human population structure, in order to determine the effects of the abiotic environment on the growth rate of infections (). Our selection of weather variables and the time frame within which we measured variation was based on the limited, but rapidly expanding, experimental and observational research on the survival and transmission of SARS-CoV-2 virus and human resistance to the resultant COVID-19 disease ([1–5]). We performed model selection to optimize model prediction of cross-validated data and performed comprehensive sensitivity analysis with respect to both data preparation and modeling decisions and found no qualitative differences between the findings represented in our best model and other models using different, but reasonable, decisions.

Infection Data

Daily infection data were obtained from the Johns Hopkins Center for Systems Science and Engineering ([6]), which documents country level aggregations of infected individuals, except in Australia, Canada, China, and USA, where state-level data are available. From these daily data, we calculated weekly growth rate () assuming an exponential model for the growth of the number of infected individuals, which fit well to COVID-19 dynamics during the early stages of spread. The starting point for one-week intervals were polity specific (either country or state level depending on the resolution of available data) and calculated beginning on the first day (denoted t0) that the number of infected individuals exceeded 40 (and 20 and 60; see sensitivity analysis below). This minimum was necessary to eliminate the early dynamics of COVID-19 in locations due primarily to transport from other regions rather than local, community transmission. This moving window approach allowed us to capture local differences in onset date of transmission without imposing any artificial cutoffs (e.g., based on calendar week). By summarizing the data in this way, we had 541 observations of distributed over 203 political units.

To capture periods when the spread rate was most severe, we chose to focus on the worst three (also two, four; see sensitivity analysis) weeks in each political unit based on the magnitude of lambda, for our model. We were primarily concerned about high rates of spread, and their possible drivers, so this decision controls for differences among polities in the onset of severe spread and differences in the timing of control measures that may reduce growth. Hence, a focus on maximum growth rates is the best, unbiased estimate of COVID-19 growth in the absence of control measures, and most likely to be influenced by weather. In sensitivity analyses, we also considered using the first 2,3, or 4 weeks following t0, and found similar, but more noisy results, owing to the likely variation among countries in the early rates of spread (e.g., in Thailand, growth was initially low before increasing rapidly).

Weather data

Weather data was aggregated from 3-hourly data downloaded from the ERA5 model by ECWMF ([7]) and averaged at 14 day intervals preceding the time period in which calculated for each polity. A 14 day interval captures the known infective period of SARS-CoV-2, where infections are known to occur from period of one to 14 days ([8]). Hence, we use the actual

319 observed weather during the period of viral transmission. This decision contrasts with previous
320 studies that used average monthly climate calculated over the interval 1970-2000 provided by
321 Worldclim (_9_). Notably, the biweekly averages we calculated are, on average, expected to reflect
322 higher temperatures due to climate change in the last 50 years compared to historic long-term
323 averages. Further, our biweekly estimates better reflect that actual conditions when infections
324 occurred, and thus are expected to better predict transmission if indeed they influence it.

326 Based on existing insights about SARS-CoV-2 and the onset of COVID-19, we considered the
327 following weather variables: temperature 2m above land surface, relative humidity, absolute
328 humidity, and total incoming UV radiation at the land surface. To align the weather data with
329 infection data for a given political unit, we determined the first day (t0) when more than 40
330 individuals were reported (also 20 and 60 infections; see below). We calculated the mean values
331 of the weather variables over the 14-day window preceding t0. For example, t0 for Connecticut,
332 USA was March 16 (when 41 records had accumulated), so the weather variables were averaged
333 over the 14-day window preceding March 10. This reflects the assumption that detected
334 infections between March 10-16 primarily occurred between February 24-March 9. Although
335 imperfect, the temporal autocorrelation of weather suggests that this is reasonable (e.g., even if
336 an infection occurred on March 2, there is typically high correlation on weather, then, and March
337 3-9).

339 Finally, note that we also explored the use of minimum and maximum values of weather
340 variables to account for the possibility that transmission was more likely driven by extreme
341 weather rather than average weather. We also considered using weekly rather than biweekly
342 intervals to reflect the possibility of shorter incubation periods. Outcomes were robust to these
343 decisions.

345 Previous studies have noted that the coarse spatial grain of infection data (country or state level)
346 makes it difficult to interpret weather variables in the context of such large spatial units (_10_). To
347 address this, we calculated weather averages over the quarter degree grid cells in a polity,
348 weighted by the population size in each cell. This resulted in weather covariates that better
349 reflect where most humans are and hence where infections occurred. Also, early maximum
350 transmission rates were usually located in large cities, and thus weights weather variation in line
351 with this bias.

Population data
354 We obtained human population data from Worldpop.org focusing on total human population
355 (density) and proportion of the population over age 60. Population density was hypothesized to
356 control for the number of interactions individuals in a location were likely to experience whereas
357 the proportion of people over 60 in a polity was hypothesized to control for reporting rate, given
358 that older people are more adversely affected by the disease and thus more likely to be tested.
359 Data were obtained at 1km resolution and summed to the quarter degree grid imposed by the
360 weather data. Polity information was obtained based on global standards (GADM.com). Each
361 quarter degree grid cell was assigned to a polity and cells were averaged over the polity.

Models

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364   We focus on the growth rate of COVID-19 cases,, rather than estimating a climate niche for the
365   virus based on its presence or absence or total number of cases, as explored in preliminary
366   studies (*11*). to avoid issues with disequilibrium in the virus' distribution. We focused on
367   estimating the rate of increase () of infected individuals, rather than directly modeling the
368   number of infected individuals, in order minimize the influence of different reporting biases in
369   different polities. We calculated as= (ln(N(t)) -ln(N(t0))/t where t was taken to be 7 days and t0
370   defined the start date for counting infections. This formulation is independent of reporting bias
371   under the assumption that the reporting bias is constant over the 7-day interval. To see this,
372   consider that the true number of infected individuals N^* is related to N via the proportion of
373   cases reported, p, such that $N=pN^*$. Substituting this expression for N into the expression for , it
374   is apparent that p cancels out. Hence so long as p is approximately constant across a 7-day
375   interval, it does not affect the estimate of growth rate.
376
377   We modeled with a hierarchical Bayesian Gaussian regression with a log link on the weekly
378   transmission rate. The full model included mean 14 day lagged temperature, mean 14 day lagged
379   relative humidity, mean 14 day lagged absolute humidity, mean 14 day lagged UV, human
380   population density and proportion of the population over 60. We used linear terms for all
381   variables but also considered a quadratic term for temperature based on suggestions of modality
382   in previous studies (*11–13*). Based on sensitivity analyses discussed below, we found that
383   maximum daily UV was a considerably better predictor than the mean (delta LOOIC = 4.x) so
384   we used the maximum in our best model. Country-level random effects were used to capture
385   differences in policies, health care or other locally specific behaviors. We also explored
386   state/province-level random effects (where applicable), but country-level effects performed
387   considerably better in all models explored based on model selection criteria.
388
389   **Model selection**
390   We were interested in developing models with high predictive ability. Thus, we performed
391   model selection using leave-one-out (LOO) cross validation. This technique iteratively uses all
392   data except for the ith data point to develop a model, then it predicts the left-out point, and uses
393   the divergence between model prediction and observation to rate model performance. The sum of
394   these divergences across all N data points is then converted into a standard measure of overall
395   model performance called the Leave-One-Out Information Criterion (LOOIC), where lower
396   numbers indicate models that better predict left-out data (*14*). This model selection method has
397   been found to excel over alternative Bayesian methods such as Deviance Information Criterion,
398   and is especially appropriate when the objective is prediction (*14*).
399
400   Model selection was performed by starting with the full model and using forward and backward
401   stepwise selection. The full model regressed the growth rate over a one-week window against
402   linear terms for mean temperature, mean UV, mean relative humidity, mean absolute humidity
403   population density, and proportion of the population over 60. We included a quadratic term for
404   temperature based on earlier studies suggesting a decline in of growth rate with temperature. We
405   also included an interaction term between temperature and UV to account for their correlation.
406   All these variables were calculated in the 7-day windows preceding the interval used to calculate
407   growth rate. During stepwise selection, we note that there were no cases of parameters trading
408   off with one another and that coefficients for each predictor always retained the same sign and
409   approximate magnitude regardless of which other predictors were in the model. The only

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410 exception to this was when UV was excluded from a model that included temperature; the
411 temperature effect dropped from positive to near zero. Hence it is important to interpret the
412 positive effect of temperature in our best model as accounting for the effect of temperature only
413 after UV has been included in the models.

415 Once we found the best suite of predictors (excluding the quadratic temperature term, the UV-
416 temperature interaction, absolute humidity, and population density), we explored whether using
417 the maximum or minimum daily values of each weather variable, and 7 versus 14 day lagged
418 intervals, improved LOOIC. The only case where we found significant model improvement
419 compared to the biweekly means was for maximum UV over both 7- and 14-day intervals. Since
420 the 14-day interval improved model performance most (based on LOOIC), we chose that as the
421 summary statistic for UV. Notably for all other weather variables there was negligible difference
422 in LOOIC when we used weekly versus biweekly means and hence we used biweekly values for
423 all variables for simplicity.

425 **Sensitivity analysis**
426 Sensitivity analysis for a variety of model decisions was conducted to determine whether our key
427 finding - the relation between COVID-19 growth rate and temperature, UV, and relative
428 humidity - was affected by any of our decisions. In all cases that follow, the median of the
429 temperature coefficient was positive, with a 95% credible interval sometimes overlapping zero
430 and sometimes not, depending on the model. In all cases, the median and 95% credible interval
431 for UV was negative. In all cases, the 95% intervals for relative humidity and population density
432 always overlapped zero but the medians were always negative and positive, respectively. The
433 quadratic temperature term never improved the model, indicating that there was no support for a
434 unimodal response to temperature.

436 Sensitivity to a number of data preparation steps was assessed. During data preparation, we
437 considered the 2, 3, and 4 worst weeks (highest lambda) following t0, as well as the first 2,3, and
438 4 weeks following t0. We chose different cutoffs (20,40,60) for numbers infected to account for
439 the difficulty in determining the time when spread became local, rather than imported. Due to the
440 strong control measures in place in China by the time our data set begins (January 22, 2020), we
441 also compared our best model with and without data from China and found no qualitative change
442 in outcomes.

444 **Coefficients over time**
445 To explore how our inference about different weather factors may have changed over time, as the
446 virus approaches a geographic and environmental equilibrium (which it may still not be at), we
447 fit a model each day since February 1, 2020, accumulating infection data up until the most recent
448 date of analysis. This analysis can illustrate (1) how earlier studies may have inferred a negative
449 dependence of growth on temperature, (2) the uncertainty inherent in earlier estimates of
450 temperature dependence, (3) the disequilibrium between COVID-19 and the environment early
451 in its spread, and (4) the smaller credible intervals, and hence increasing confidence in our model
452 based on more recent data. Note that the model used to illustrate this pattern (1) used the first
453 (rather than the worst) 3 weeks following t0 to accumulate data as early as possible and thus
454 reflect decisions made in earlier studies, and (2) used polity (rather than country) effects because
455 the data in early February was predominantly from China and thus country effects could not be

456   fit. Although early data gaps meant that we could not precisely replicate previous analyses with
457   this exercise, we obtained similar outcomes using this model for the present analysis (again
458   indicating model robustness). As well, this exercise demonstrated how conclusions from earlier
459   studies may have arisen, even with our more refined model, but based on a longer time series.
460

461   **Projections**
462   Future predictions of the *potential* growth rate in May-September was made by projecting our
463   highest performing model according to LOOIC. Importantly, we reinforce that our predictions
464   pertain to the possible growth rate in the absence of social distancing or other control measures
465   because it is based on a model fit with infections that occurred primarily before precautionary
466   policies were implemented. Note that even if a policy were implemented on, for example, March
467   14, we expect that infections reported in the next two weeks were initiated before the policy
468   began. Hence, we predict the underlying contribution of weather to future COVID-19 growth.
469   Importantly, these predictions reflect what would happen if other control measures are relaxed
470   and the natural dynamics of infection can begin again in a population with little resistance.
471   Currently governments are deciding when and how to relax control measures, often under the
472   assumption that weather will lessen the potential for spread in the upcoming months. Thus,
473   whereas we do not presume to predict the actual future growth rate of COVID-19, we do hope to
474   capture the potential maximum growth rate in order to inform the relative risks of alternative
475   control strategies.
476

477   To make future projections, we obtained monthly mean temperature and relative humidity
478   weather data from 2015-2019 from the same data source as above, under the assumption that
479   these recent years are representative of what to expect in the coming months. Notably hotter or
480   cloudier (lower UV) days in the coming months would suggest higher growth rates than we
481   predict. UV data was not available in a monthly aggregation, so we obtained the 3-hourly data
482   and aggregated it to monthly values. Human population was assumed to remain constant. We
483   projected the models without random effects (or equivalently at the mean value of 0) as we were
484   reluctant to assume that country-level policies, reporting, or health care potential will remain the
485   same in the future. We expect that different country-level effects will dominate in the future, but
486   predicting these offsets is beyond the scope of this study.
487

488   **Caveats**
489   As with any predictive study, we seek to use the best available data and understanding of
490   mechanisms to develop possible projections that make clear underlying decisions and
491   uncertainty. Ultimately, such predictions must be treated with appropriate caution given the
492   limited understanding of SARS-CoV-2 virus, human resistance, and its transmission dynamics
493   at this time. Thus, while we seek to inform decisions, those decisions must also recognize the
494   inherent uncertainty in any predictive model, but especially in the context of limited information.
495   Future data will ultimately be the arbiter of these predictions, and thus good predictive modeling
496   will require repeated bouts of model validation, revision, and re-projection as we learn more
497   about this virus.
498

499   In particular, we await mechanistic information on viral physiology and human resistance to
500   move beyond the correlative approach taken here by necessity. Mechanistic models apply
501   insights about an organism's intrinsic biology using parameters often collected from careful

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502 experimental manipulations. However, in the absence of this information, correlative models can
503 predict near-term dynamics with accuracy (*15*). Bayesian approaches like ours can integrate both
504 mechanistic and correlative knowledge as these pieces of information become available.
505
506 One thing that we do not account for in our model is human behavior and control measures. By
507 modeling maximum growth rate and using a threshold number of cases, we restrict our analyses
508 to the period during which the disease expanded quickly, following the beginning of community
509 transmission but before major control measures were implemented. For instance, most countries
510 began implementing national control measures in mid-March, which would influence infections
511 recorded into early April, based on a 14-day window for symptoms to emerge. Hence, we chose
512 to limit our data set to records before April 7. However, we note that following early April,
513 growth rates are expected to be much lower due to control measures, and these will continue to
514 be important to reduce growth rates below the potential values we predict here which do not
515 account for control.
516
517 We used available insights about SARS-Cov-2, related viruses, and observations of COVID-19
518 dynamics to select a list of factors that likely influence it. Although we purposefully limited
519 these variables to reflect our best knowledge and to avoid overfitting, certainly other climate and
520 epidemiological factors are likely missing from the model. Future studies should consider
521 embedding these climate insights into epidemiological models that include human demography,
522 immunity, movement, behaviors, medical capacity, and control efforts (*4*).
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527   **Supplementary Materials**
528
529   Figure S1. Posterior predicted probabilities of growth rate refelect weak trends with environment
530   and high uncertainty in predictions.

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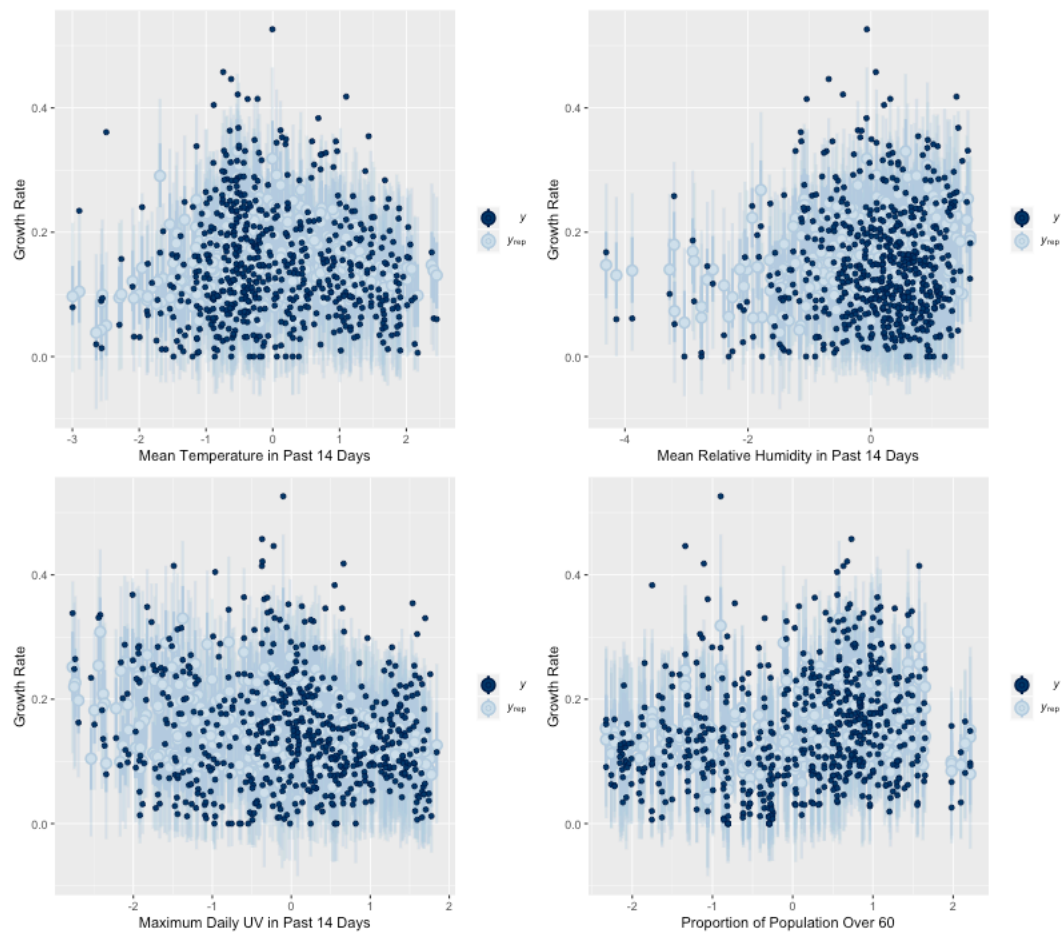
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