



Global effect of city-to-city air pollution, health conditions, climatic & socio-economic factors on COVID-19 pandemic

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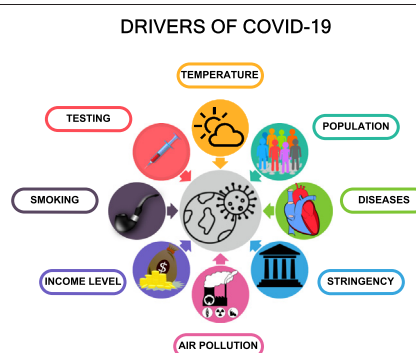
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HIGHLIGHTS

- Air pollutant species like ambient PM_{2.5}, nitrogen dioxide and ozone spur COVID-19 cases.
- Underlying health conditions like cardiovascular diseases, diabetes and smoking increase risk of COVID-19 outcomes.
- High ambient temperature and relative humidity have mitigation effect on COVID-19.
- Population size, aged population, extreme poverty and income level are confounding factors of COVID-19.
- Government stringency on COVID-19 containment and accessibility to hospital beds can reduce COVID-19 cases.

GRAPHICAL ABSTRACT



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ABSTRACT

The rate of spread of the global pandemic calls for much attention from the empirical literature. The limitation of extant literature in assessing a comprehensive COVID-19 portfolio that accounts for complexities in the spread and containment of the virus underscores this study. We investigate the effect of city-to-city air pollutant species, meteorological conditions, underlying health conditions, socio-economic and demographic factors on COVID-19 health outcomes. We utilize a panel estimation of 615 cities in 6 continents from January 1 to June 11, 2020. While social distancing measures, movement restrictions and lockdown are reported to have improved environmental quality, we show that ambient PM_{2.5} remains unhealthy and above the acceptable threshold in several countries. Our empirical assessment shows that while ambient PM_{2.5}, nitrogen dioxide, ozone, pressure, dew, Windgust, and windspeed increase the spread of COVID-19, high relative humidity and ambient temperature have mitigation effect on COVID-19, hence, decreases the number of confirmed cases. We report 66.3% of countries projected to experience a second wave of COVID-19 if government stringency and safety protocols are not enhanced. By extension, our assessments demonstrate that several factors namely underlying health conditions, meteorological, air pollution, health system quality, socio-economic and demographics spur the reproduction effect of COVID-19 across countries. Our study highlights the importance of government stringency in containing the spread of COVID-19 and its impacts.

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1. Introduction

The confirmed cases of COVID-19 global pandemic have surpassed 14 million as of July 2020, with corresponding 602,656 cases of deaths

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(4.3% death rate), and 7,894,890 recovery cases (Lauren, 2020). Top 10 countries of confirmed cases include the US (3,647,715), Brazil (2,046,328), India (1,039,084), Russia (758,001), Peru (345,537), South Africa (337,594), Mexico (331,298), Chile (326,439), the UK (294,803), and Iran (269,440) [see Fig. 1]. Similarly, hotspot countries with reported deaths include the US (139,266), Brazil (77,851), the UK (45,318), Mexico (38,310), Italy (35,028), France (30,155), Spain (28,420), India (26,273), Iran (13,791), and Peru (12,799) [see Fig. 2].

To contain the spread and reduce fatalities from COVID-19, there are several Government responses instituted across countries ranging from public health system, economic Response, closure and containment (ACAPS, 2020). Public health system responses toward improving health system quality include public information campaigns, testing policy, contact tracing, emergency investment in healthcare, and investment in vaccines. The economic response toward the alleviation of economic burden comprises income support, debt contract relief, fiscal measures, and international support (Sarkodie and Owusu, 2020a). Besides, closure and containment directives from Government include school closure, workplace closure, public transport closure, public even cancellation, restrictions on gathering, stay at home requirements, restriction on internal movement, and international travel controls. Aside government responses toward the pandemic, other factors and conditions are reported to increase the spread of coronaviruses. Factors and conditions that affect the exposure to coronaviruses include underlying health conditions (CDC et al., 2020), meteorological factors (Liu et al., 2020; Sarkodie and Owusu, 2020b), environmental conditions (Bourouiba, 2020; Sarkodie and Owusu, 2020c), socio-economic and demographic factors (Zhou et al., 2020).

The trend of COVID-19 reproduction rates presented in Fig. 3 heralds the second wave of spread across countries. Reproduction rates greater than 1 (from green to red) indicates increasing growth trajectory of COVID-19 cases whereas value below 1 (from yellow to blue) signals COVID-19 cases will fizzle out. Evidence shows that 66.3% countries are projected to experience a second wave, with hotspot countries namely Latvia, Chad, Estonia, Mongolia, Equatorial Guinea, Iceland, Thailand, Andorra, Australia and Guinea Bissau. In contrast, safe countries with decreasing spread and unlikely second wave of spread include

Tanzania, UK, Paraguay, Gabon, Denmark, DR Congo, Sweden, Sudan, Spain, and Qatar (Fig. 3). The dynamics of reproduction rates are affected by several public health factors, climatic conditions and government response toward the containment of the spread. The rate of spread of COVID-19 signals the complexities in assessing its growth path for containment.

There exist inconsistent results on the impact of climatic factors on COVID-19—owing to the failure to account for heterogeneous effects across countries. While studies report the mitigation effect of COVID-19 on ambient air pollution, other existing literature report air pollution and climatic factor attributed spread of COVID-19 (Ma et al., 2020; Xie and Zhu, 2020; Zhu et al., 2020). The complexity of the spread and containment of COVID-19 requires broader data consultation, location dynamics, robust and consistent estimation techniques. Such characteristics are ignored in the existing literature. Besides, there are several confounding factors aside air pollution and climatic factors that require inclusion to unravel the widespread dynamics of the virus. Contrary to the extant literature, we present a comprehensive empirical assessment of COVID-19 pandemic by controlling for underlying health conditions, government response, socio-economic and demographic factors, climatic and environmental conditions from 615 cities in 6 continents. The statistical hypotheses tested in this study include:

First, H_0 : Climatic factors have no effect on COVID-19 cases.

H_1 : Climatic factors have either positive or negative effect on COVID-19 cases.

Second, H_0 : Socio-economic and demographic factors have no effect on COVID-19 deaths.

H_1 : Socio-economic and demographic factors have either positive or negative effect on COVID-19 deaths.

Third, H_0 : Underlying health conditions have no effect on COVID-19 health outcomes.

H_1 : Underlying health conditions have either positive or negative effect on COVID-19 health outcomes.

Fourth, H_0 : Stringency and availability of beds have no effect on COVID-19 cases.

H_1 : Stringency and availability of beds have negative effect on COVID-19 cases.

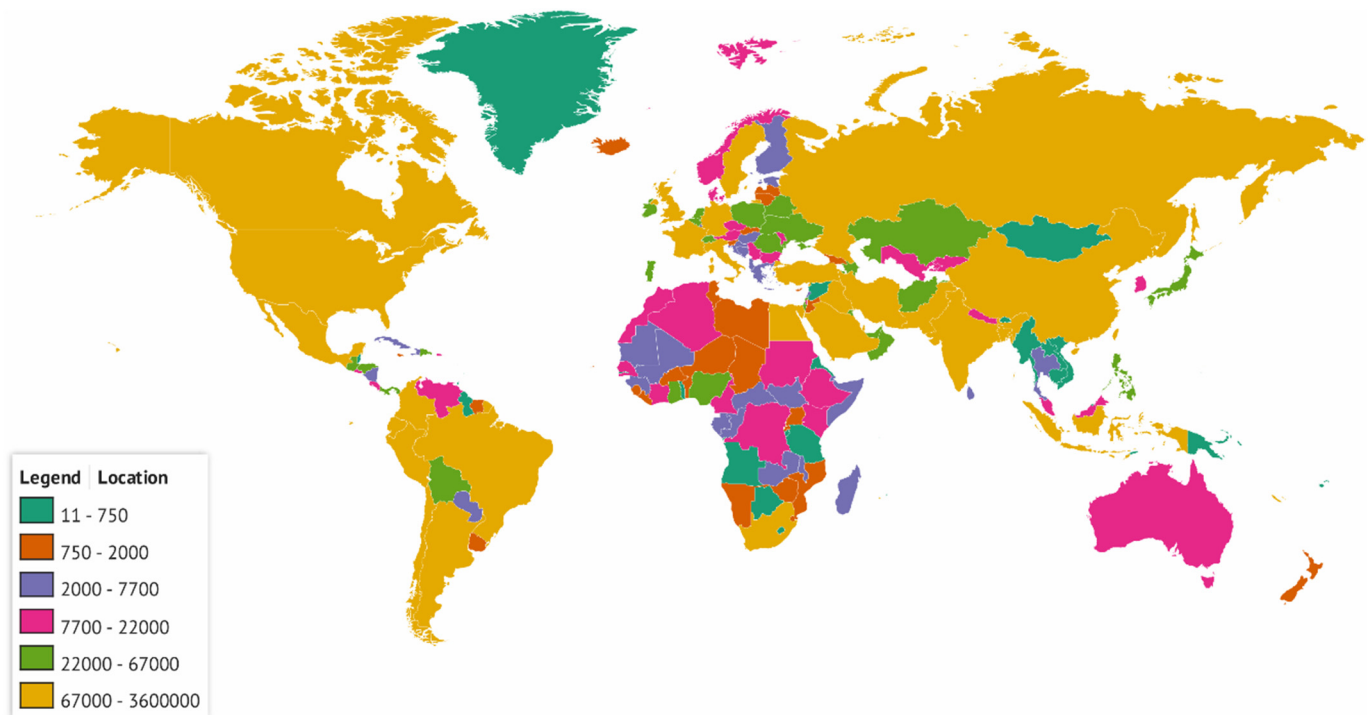


Fig. 1. Global distribution of COVID-19 confirmed cases [as of July 2020]. Data source: John Hopkins (Lauren, 2020).

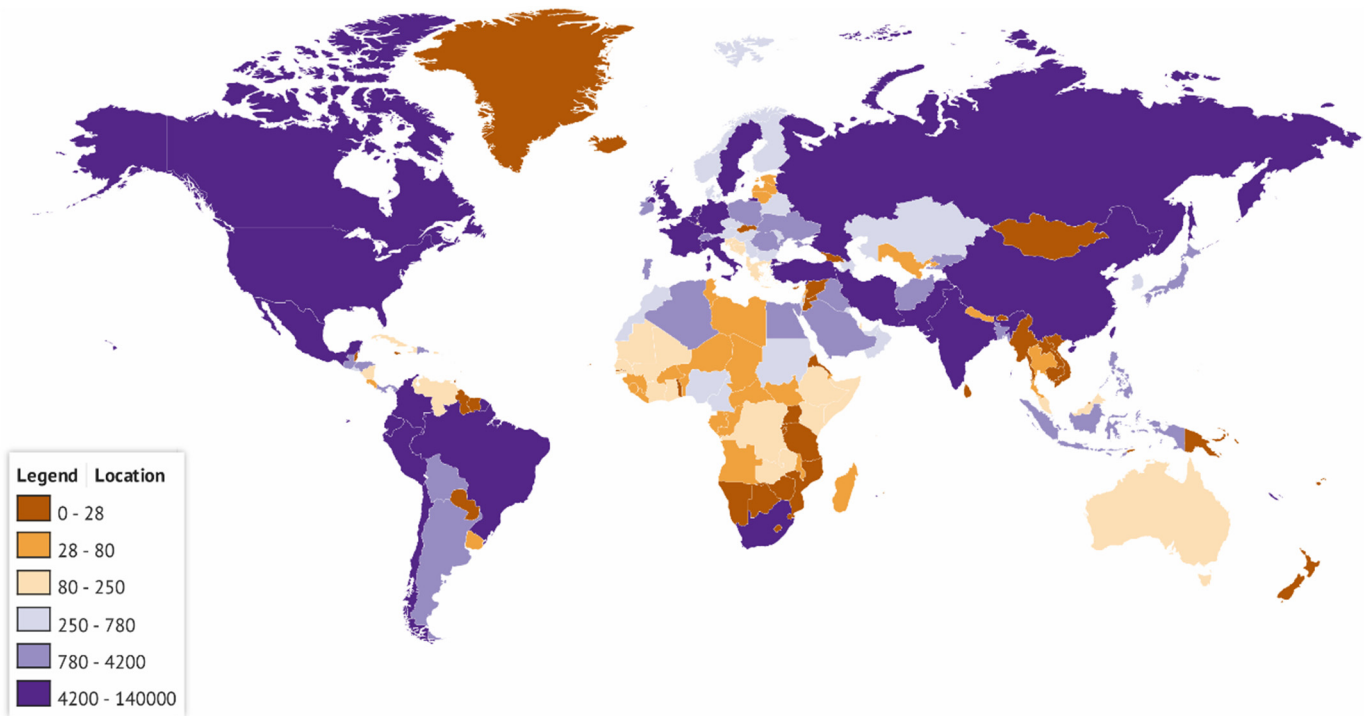


Fig. 2. Global distribution of COVID-19 reported death cases.

Finally, H_0 : Concentrations of air pollutants have no effect on COVID-19 cases.

H_1 : Concentration of air pollutants have positive effect on COVID-19 cases.

To test the several hypotheses proposed in this study, we use robust and consistent cross-sectional time series technique that

controls for unequal distribution of data and heterogeneous effects across cities. In this scenario, we can account for city-to-city, and country-to-country heterogeneity—that is critical for modeling the pandemic. We identify and demonstrate the importance of accounting for unobserved common factors that spur or degrade COVID-19 health outcomes.

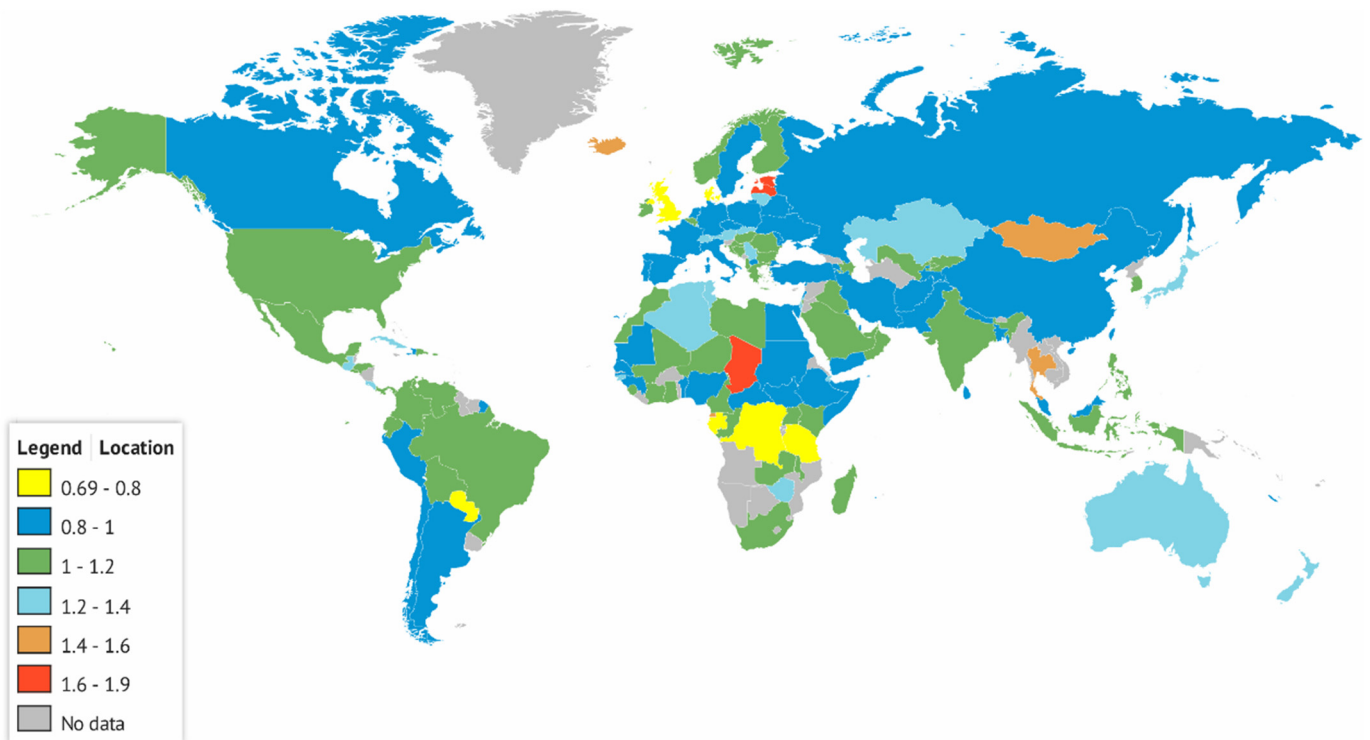


Fig. 3. Global distribution of COVID-19 reproduction rates. Legend: Reproduction rates greater than 1 (from green to red) indicates a potential increase of COVID-19 cases whereas rates below than 1 (from yellow to blue) suggest decreasing levels of COVID-19 cases with no potential escalation.)

2. Methodology

2.1. Data

Our empirical assessment of COVID-19 entails the collation of data series from multiple sources for 615 Cities in 6 Continents (Fig. 4) spanning January 1 to June 11, 2020. Data on particulate matter 2.5 (PM_{2.5}—μg/m³), ozone (O₃—μg/m³), nitrogen dioxide (NO₂—μg/m³), dew (°C), relative humidity (%), pressure (hPa), wind gust (m/s), wind speed (m/s), ambient temperature (°C) were extracted from World Air Quality Index project (WAQI, 2020), whereas reproduction rates were obtained from EpiForecasts (Abbott et al., 2020). Data on cardiovascular diseases, prevalence of diabetes, male and female Smokers, total tests conducted across cities, Government stringency in containing the spread of the virus, total number of hospital beds per thousand population, total number of COVID-19 deaths reported, population, Aged older than 65, GDP per capita—a proxy for income level and extreme poverty were obtained from Our World in Data (OWID, 2020). The selection of data series with specific characteristics is due to the complexity of coronaviruses. Climate change disrupts weather patterns by changing the frequency of temperature, precipitation, humidity, wind speed, dew and pressure. These changes in weather patterns affect the concentrations of atmospheric pollutants such as PM_{2.5}, O₃, and NO₂. Hence, the interaction between weather conditions and concentrations of atmospheric pollutants affects human immune response to morbidities (De Sario et al., 2013). Thus, confounding factors such age, existing lifestyle (smoking), health conditions (CVD, diabetes), and health quality (hospital beds, testing) underpin health outcomes. This implies that several socio-economic, political and climatic factors affect the spread of coronaviruses. Figs. 1–4 were constructed using visualization tools provided by Knoema (2020). The corresponding statistical analysis of the data series is presented in Table 1. Our initial dataset comprises 615 cities and 98,480 observations. Jarque-Bera test presented in Table 1 shows that the data series are not normally distributed, hence, we applied a logarithmic conversion to control for potential heteroskedasticity. We find an average of 4 beds per 1000 population, a situation that can be worse in low-income countries. This means that low-income countries with high population might struggle with containing the spread especially through hospitalization. We observe 46 μg/m³ mean ambient PM_{2.5} far above the 25 μg/m³ air quality standard by WHO. This by implication means that the lockdown might have had little impact on ambient air pollution.

2.2. Model estimation

The novel coronavirus (COVID-19) is a global pandemic with public health importance, hence, has characteristics of global common shock and transboundary effects. Failure to account for such characteristics across countries, cities or continents render the model estimation spurious. The outlined data characteristics can be accounted for in a technique that controls for cross-section dependence. Despite the global common shock and transboundary effects, the cross-sectional time series may suffer from heterogeneity—due to differences in the onset of COVID-19, intervention and treatment across countries. Thus, both cross-section dependence and heterogeneity alter the consistency and robustness of COVID-19 estimation and modeling across countries. Due to the dynamics of COVID-19 spread, previous studies highlighted the importance of accounting for unobserved common factors and individual-specific effects (Owusu and Asumadu, 2020). The unequal distribution of data on COVID-19 cases, underlying health conditions, climatic factors, socio-economic and demographic factors across countries pose another challenge in the selection of an estimation technique that controls for unevenly spaced data, cross-section dependence and heterogeneity. Here, we utilized the panel standard error corrected estimation technique that accounts for city-level heteroskedastic errors. In addition, the cross-sectional time series approach adopted solves for contemporaneous correlation across cities. For brevity, the generic estimation procedure can be expressed as:

$$\ln Y_{i,t} = \beta \times \ln X_{i,t} + \varepsilon_{i,t} \tag{1}$$

where $\ln Y$ represents the logarithmic transformation of COVID-19 confirmed cases and deaths, respectively, β is the estimated parameter, $\ln X$ denotes the regressors, $i = 1, \dots, N$ is the number of sampled cities, $t = 1, \dots, T_i$ is time periods in city i , $\varepsilon_{i,t}$ is the error term with autocorrelation along time periods and/or exhibit contemporaneous correlation across cities. Because our panel data is unbalanced, the covariance of the error term expressed as:

$$E[\varepsilon\varepsilon'] = \sum_{N \times N} \otimes I_{T_i \times T_i} \tag{2}$$

where $\sum(\cdot)$ is the city-by-city covariance matrix with identity matrix I . The model specification of Eq. (1) can be presented as:

$$\text{Model 1 : } \ln \text{CASES}_{i,t} = \beta_1 \ln \text{TEMP}_{i,t} + \beta_2 \ln \text{PRESS}_{i,t} + \beta_3 \ln \text{HUM}_{i,t} + \beta_4 \ln \text{DEW}_{i,t} + \beta_5 \ln \text{GUST}_{i,t} + \varepsilon_{i,t} \tag{3}$$

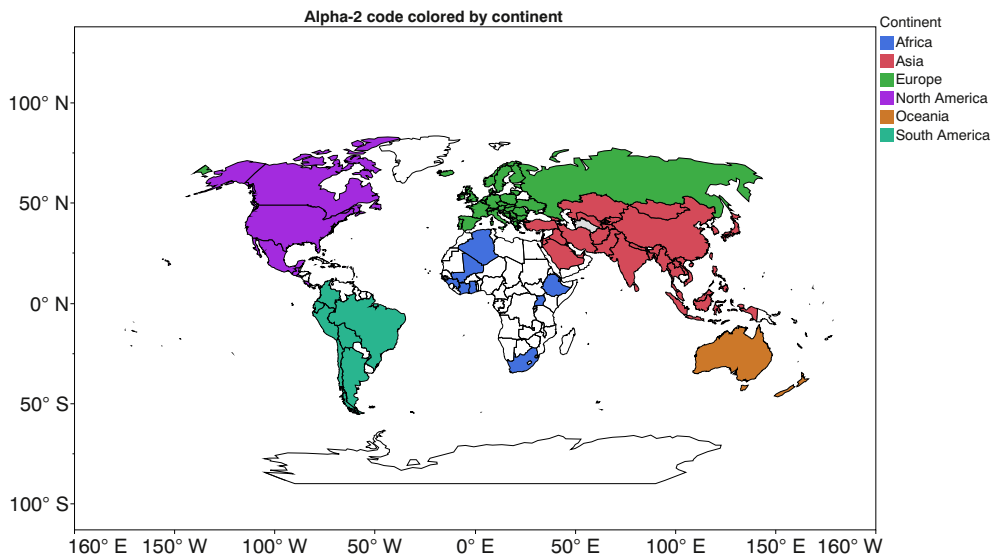


Fig. 4. Sampled data from 615 Cities in 6 Continents for empirical analysis.

Table 1
Statistical analysis of data series.

Variable	Mean	Std. Dev.	Skewness	Kurtosis	Jarque-Bera	Probability	Observations
Aged 65 older	13.038	7.340	-0.198	2.203	3.25E+03	0.000	98,480
CVD	169.193	109.040	0.922	4.263	2.05E+04	0.000	98,480
Dew	5.093	8.489	0.547	3.337	5.38E+03	0.000	98,480
Diabetes prevalence	7.323	3.731	0.005	3.412	6.97E+02	0.000	98,480
Extreme poverty	2.005	5.086	3.574	15.776	8.79E+05	0.000	98,480
Female smokers	13.075	10.730	0.261	1.729	7.75E+03	0.000	98,480
GDP per capita	28,101.120	17,478.620	-0.005	2.089	3.41E+03	0.000	98,480
Hospital beds/thousand	3.994	3.424	1.282	4.107	3.20E+04	0.000	98,480
Humidity	59.683	27.449	-0.909	2.960	1.36E+04	0.000	98,480
Male smokers	28.718	14.108	-0.477	2.972	3.73E+03	0.000	98,480
NO ₂	7.554	7.545	2.292	17.430	9.41E+05	0.000	98,480
O ₃	16.218	13.211	0.636	7.581	9.27E+04	0.000	98,480
PM _{2.5}	46.334	43.104	2.180	19.152	1.15E+06	0.000	98,480
Population	2.46E+08	4.51E+08	2.12E+00	5.77E+00	1.06E+05	0.000	98,480
Pressure	890.180	322.489	-2.489	8.412	2.22E+05	0.000	98,480
Stringency index	42.651	34.973	-0.025	1.349	1.12E+04	0.000	98,480
Temperature	12.493	9.617	0.130	3.396	9.20E+02	0.000	98,480
Total cases	75,912,550	255,078,400	5.350	32.972	4.16E+06	0.000	98,480
Total deaths	4926.993	15,647.760	4.703	26.830	2.69E+06	0.000	98,480
Total tests/thousand	4.760	13.042	4.550	35.643	4.71E+06	0.000	98,480
Wind gust	1.913	4.024	3.539	39.412	5.65E+06	0.000	98,480
Wind speed	1.458	5.307	34.154	1495.470	9.16E+09	0.000	98,480

where $\ln \text{CASES}_{i,t}$ is the total number of COVID-19 confirmed cases, $\ln \text{TEMP}_{i,t}$ denotes temperature, $\ln \text{PRESS}_{i,t}$ represents pressure, $\ln \text{HUM}_{i,t}$ signifies humidity, $\ln \text{DEW}_{i,t}$ means dew, and $\ln \text{GUST}_{i,t}$ is wind gust. In Model 1, we hypothesize that meteorological factors affect the spread of COVID-19 cases across cities.

$$\text{Model 2: } \ln \text{CASES}_{i,t} = \beta_1 \text{PM}_{2.5i,t} + \beta_2 \ln \text{O}_{3i,t} + \beta_3 \ln \text{NO}_{2i,t} + \beta_4 \ln \text{SPEED}_{i,t} + \varepsilon_{i,t} \quad (4)$$

where $\ln \text{PM}_{2.5i,t}$ is particulate matter 2.5, $\ln \text{O}_{3i,t}$ is ozone, NO_2 denotes nitrogen dioxide, and SPEED is the wind speed. In Model 2, we test the hypothesis that climatic conditions alter the spread of COVID-19 cases across cities.

$$\text{Model 3: } \ln \text{CASES}_{i,t} = \beta_0 \ln \text{CASES}_{i,t-1} + \beta_1 \ln \text{CVD}_{i,t} + \beta_2 \ln \text{DIA}_{i,t} + \beta_3 \ln \text{SMOK}_{i,t} + \varepsilon_{i,t} \quad (5)$$

where $\ln \text{CVD}_{i,t}$ is the rate of cardiovascular diseases, $\ln \text{DIA}_{i,t}$ represents the prevalence of diabetes, and $\ln \text{SMOK}_{i,t}$ denotes smokers. In Model 3, we ascertain the impact of underlying health conditions on COVID-19 cases.

$$\text{Model 4: } \ln \text{CASES}_{i,t} = \beta_1 \ln \text{TESTS}_{i,t} + \beta_2 \ln \text{STRIN}_{i,t} + \beta_3 \ln \text{BEDS}_{i,t} + \varepsilon_{i,t} \quad (6)$$

where $\ln \text{TESTS}_{i,t}$ is the total tests conducted across cities, $\ln \text{STRIN}_{i,t}$ represents Government stringency in containing the spread of the virus, $\ln \text{BEDS}_{i,t}$ is the total number of hospital beds per thousand population. In Model 4, we test the hypothesis that testing, stringency and availability of beds affect the cases of COVID-19.

$$\text{Model 5: } \ln \text{DEATHS}_{i,t} = \beta_1 \ln \text{CVD}_{i,t} + \beta_2 \ln \text{DIA}_{i,t} + \beta_3 \ln \text{SMOK}_{i,t} + \varepsilon_{i,t} \quad (7)$$

where $\ln \text{DEATHS}_{i,t}$ is the total number of COVID-19 deaths reported, $\ln \text{CVD}_{i,t}$ is the rate of cardiovascular diseases, $\ln \text{DIA}_{i,t}$ represents the prevalence of diabetes, and $\ln \text{SMOK}_{i,t}$ denotes smokers. In Model 5, we hypothesize that underlying health conditions escalate COVID-19 deaths.

$$\text{Model 6: } \ln \text{DEATHS}_{i,t} = \beta_1 \ln \text{POP}_{i,t} + \beta_2 \ln \text{AGED}_{i,t} + \beta_3 \ln \text{INCOME}_{i,t} + \beta_4 \ln \text{POV}_{i,t} + \varepsilon_{i,t} \quad (8)$$

where $\ln \text{POP}_{i,t}$ denotes population, $\ln \text{AGED}_{i,t}$ represents the aged older than 65, $\ln \text{INCOME}$ is GDP per capita—used as a proxy for income level,

and $\ln \text{POV}_{i,t}$ denotes extreme poverty. In Model 6, we test socio-economic and demographic factors affect reported cases of COVID-19 deaths.

3. Results

3.1. Trends of air pollutants

The implementation of social distancing measures is reported to have improved air quality. In Figs. 5–7, we assess the concentrations of ambient air pollution, ozone and nitrogen dioxide across countries during the lockdown. We observe that while air quality improved significantly during the lockdown, the 24-h mean ambient air pollution (see Fig. 5) remains unhealthy ($>25 \mu\text{g}/\text{m}^3$) across the cities (top 10 countries) of, inter alia, Bangladesh ($160.40 \mu\text{g}/\text{m}^3$), Mali ($148.32 \mu\text{g}/\text{m}^3$), Mongolia ($116.87 \mu\text{g}/\text{m}^3$), Pakistan ($116.79 \mu\text{g}/\text{m}^3$), Uganda ($116.25 \mu\text{g}/\text{m}^3$), India ($107.15 \mu\text{g}/\text{m}^3$), Nepal ($101.34 \mu\text{g}/\text{m}^3$), Indonesia ($100.12 \mu\text{g}/\text{m}^3$), Myanmar ($99.58 \mu\text{g}/\text{m}^3$), and Laos ($97.18 \mu\text{g}/\text{m}^3$). In contrast, both ozone (Fig. 6) and nitrogen dioxide (Fig. 7) concentrations for 8-h per day and 1-h per day, respectively for all cities are lower ($30 \mu\text{g}/\text{m}^3 - 17.5 \mu\text{g}/\text{m}^3$) than WHO guidelines for air quality [ozone $-100 \mu\text{g}/\text{m}^3$; $\text{NO}_2 - 200 \mu\text{g}/\text{m}^3$].

3.2. Heterogeneous effects

Our empirical estimation began by testing for heterogeneous effects using the modified Wald test statistic for heteroskedasticity. We observe in Table 2 that the null hypothesis of homogeneity is rejected at $p\text{-value} < 0.01$ —confirming the city-level heterogeneity and justifying the application of panel standard error corrected estimation technique to control heteroskedastic errors across cities. We validated the estimated model using the conditional marginal effects with the heteroskedastic-corrected variance-covariance matrix of the panel standard error corrected estimator and regressors fixed at means. The subsequent derivatives expressed as elasticities vary in magnitude but similar qualitative signs and significance as the panel standard error corrected estimates. We further observe that the estimates are within the 95% confidence band, hence, confirming the robustness and consistency of the estimated model (Fig. 8). The estimated models in Table 2 are statistically significant at 1% level with predictive power between 0.33 and 1.00—implying that the regressors explain 33%–100% variations in COVID-19 health outcomes.

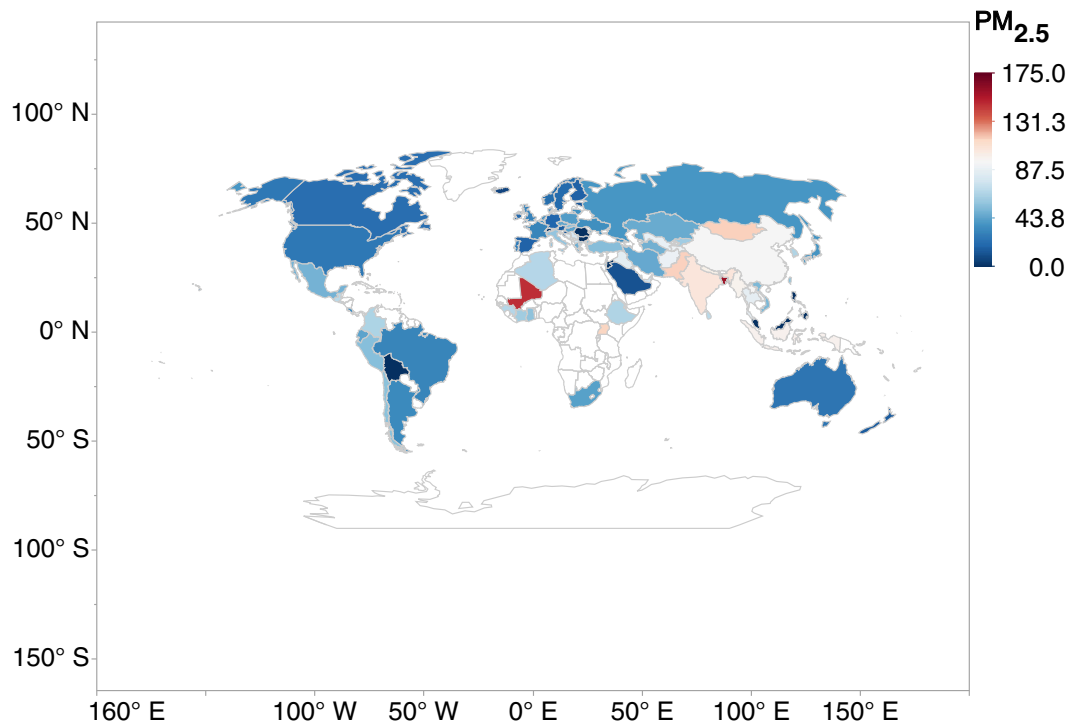


Fig. 5. Global distribution of ambient air pollution ($PM_{2.5}$ — $\mu\text{g}/\text{m}^3$) from January 1 to June 11, 2020 across 615 cities in 6 continents.

3.3. Weather-pandemic interaction

We hypothesized via the empirical analysis that meteorological factors affect the spread of COVID-19 cases across cities. We observe in Table 2 (Model 1) that there exists a strong positive relationship (p -value < 0.01) between pressure, dew, Windgust and confirmed cases of COVID-19. Hence, an increase in atmospheric pressure, Windgust

and dew escalates the spread of COVID-19 by 2.46% (CI [2.297, 2.616]), 0.34% (CI [0.259, 0.428]) and 0.10% (CI [0.047, 0.153]), respectively. In contrast, we find a strong negative association (p -value < 0.01) between ambient temperature, relative humidity and COVID-19 cases. This implies that increasing levels of ambient temperature and relative humidity have the potential of reducing the spread of COVID-19 by -0.74% (CI [$-0.915, -0.556$]) and -1.25% (CI [$-1.450, -1.052$]).

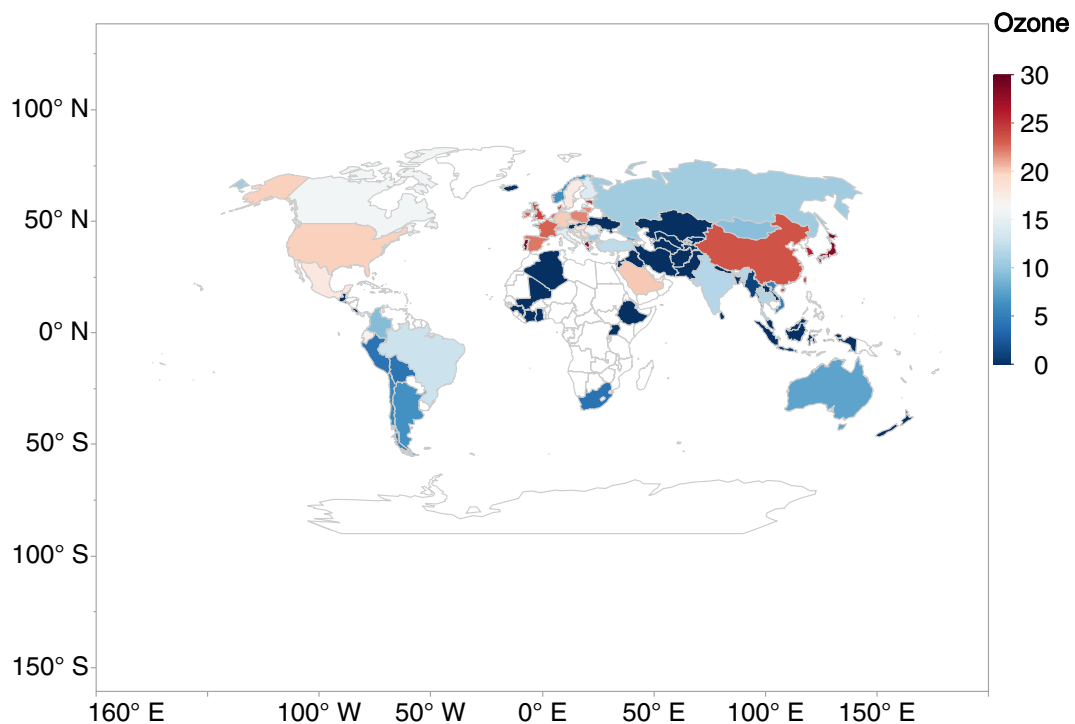


Fig. 6. Global distribution of ozone (O_3 — $\mu\text{g}/\text{m}^3$) from January 1 to June 11, 2020 across 615 cities in 6 continents.

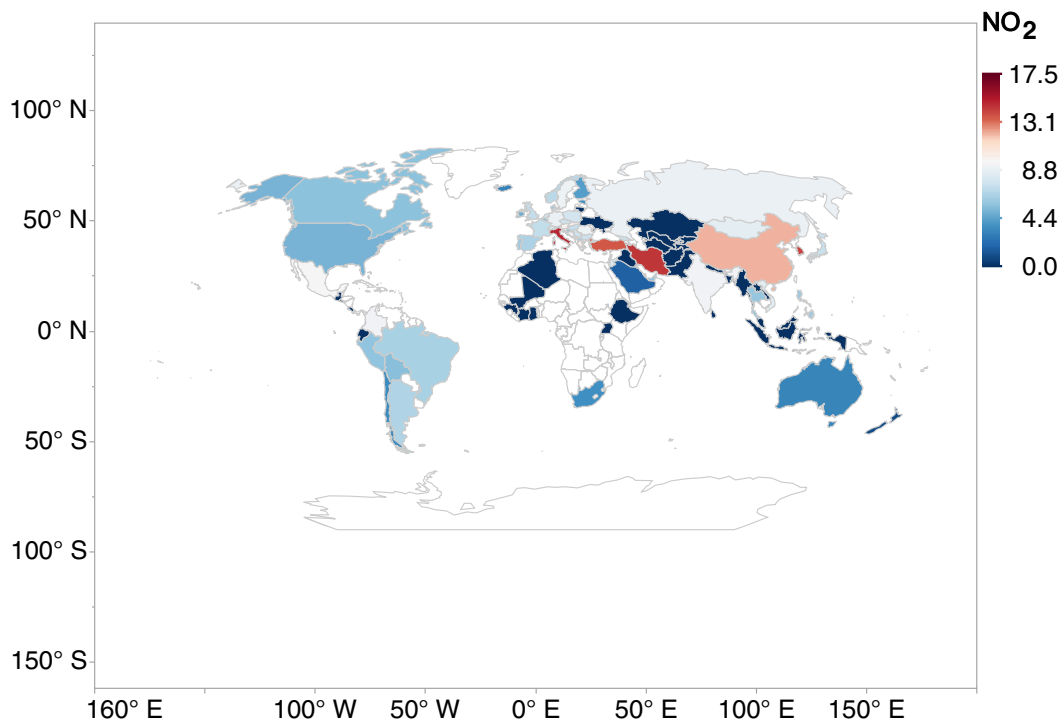


Fig. 7. Global distribution of nitrogen dioxide (NO_2 — $\mu\text{g}/\text{m}^3$) from January 1 to June 11, 2020 across 615 cities in 6 continents.

3.4. Pollutants-pandemic interaction

We tested the hypothesis that air pollutant conditions alter the spread of COVID-19 cases across cities. Evidence from Table 2 (Model 2) reveal a strong positive link (p -value < 0.01) between air pollutant species, windspeed and COVID-19 cases. By implication, increasing levels of ambient $\text{PM}_{2.5}$, ozone, NO_2 and windspeed increases the propagation rate of COVID-19 by 0.31% (CI [0.291, 0.327]), 0.37% (CI [0.355, 0.391]), 0.13% (CI [0.107, 0.148]), and 0.05% (CI [0.041, 0.064]), respectively.

Next, we assessed the impact of underlying health conditions on COVID-19 cases by controlling for omitted variable bias. We find a positive and significant (p -value < 0.01) lagged confirmed cases, inferring that victims of COVID-19 can be re-infected if safety protocols are not observed. Incidence of cardiovascular diseases, diabetes and smoking have a strong positive relationship with confirmed cases of COVID-19. Correspondingly, increasing prevalence of cardiovascular diseases, diabetes and smoking spur COVID-19 cases by 0.002% (CI [0.000, 0.004]), 0.039% (CI [0.036, 0.042]), and 0.027% (CI [0.025, 0.030]).

3.5. Health quality-pandemic interaction

We tested the hypothesis that COVID testing, stringency and availability of beds affect COVID-19 cases. The corresponding results presented in Table 2 (Model 4) show that increasing COVID-19 tests accelerates COVID-19 cases by 1.01% (CI [1.007, 1.020]). Contrary, stringent measures and increase of hospital beds decline COVID-19 cases by 0.67% (CI [−0.689, −0.645]) and 0.29% (CI [−0.306, −0.280]). Thus, increasing testing capacity through contact tracing increases the chances of discovering more cases of COVID-19. However, stringent containment measures and expansion of hospital bed capacity mitigate the spread of COVID-19 cases.

3.6. Health conditions-pandemic interaction

Besides, we hypothesized the effect of underlying health conditions on COVID-19 deaths with results presented in Table 2 (Model 5). We

find that the prevalence of cardiovascular diseases, diabetes and smoking increases COVID-19 attributable deaths by 0.22% (CI [0.178, 0.253]), 0.25% (CI [0.230, 0.265]) and 0.04% (CI [0.035, 0.039]). This suggests victims of COVID-19 with underlying conditions such as cardiovascular diseases, diabetes and habit of smoking have a higher chance of dying from COVID-19 infection.

3.7. Socio-economic & demographics-pandemic interaction

Similarly, we examined the impact of socio-economic and demographic factors on reported cases of COVID-19 deaths. We observe in Table 2 (Model 6) that population, aged and extreme poverty has a positive and significant impact on COVID-19 deaths whereas income level reduces deaths. Thus, expansion in population size, aged 65 older and extreme poverty spur COVID-19 deaths by 0.34% (CI [0.320, 0.359]), 1.47% (CI [1.343, 1.593]), and 0.08% (CI [0.048, 0.110]), respectively. In contrast, an increase in income level reduces the potential of COVID-19 deaths by 0.45% (CI [−0.501, −0.396]).

4. Discussion

The institution of lockdown, movement restrictions and other social distancing measures are reported to have improved environmental quality. Assessment of air pollutant species from real-time data underscores that nitrogen dioxide and ozone concentration levels reduced significantly below the acceptable standards for air quality. Air pollutant species are mostly transportation and industrial related emissions, hence, a lockdown would have reduced the emissions significantly (Chen et al., 2020). Though ambient air pollution might have declined in several countries, however, ambient $\text{PM}_{2.5}$ remains unhealthy and above the threshold, contradicting previous reports in extant literature (Chen et al., 2020; Sarkodie and Owusu, 2020a). The reduction of ambient $\text{PM}_{2.5}$ in some countries can be attributed to a decline in the concentration of nitrogen dioxide and ozone, evidenced in our study and others (Wang et al., 2020). We further observe that while ambient $\text{PM}_{2.5}$ is relatively lower in high-income countries, concentrations are higher in low-income economies like Bangladesh, Mali, Mongolia, Pakistan,

Table 2
Estimated parameters of COVID-19 expanded drivers.

Variables	Model 1	Model validation	Variables	Model 2	Model validation
Temperature	-0.735*** [-0.915, -0.556] (0.091)	-0.195*** [-0.243, -0.147] (0.024)	PM _{2.5}	0.309*** [0.291, 0.327] (0.009)	0.460*** [0.436, 0.484] (0.012)
Pressure	2.457*** [2.297, 2.616] (0.081)	1.600*** [1.496, 1.705] (0.053)	Ozone	0.373*** [0.355, 0.391] (0.009)	0.421*** [0.405, 0.436] (0.008)
Humidity	-1.251*** [-1.450, -1.052] (0.101)	-0.491*** [-0.569, -0.413] (0.040)	NO ₂	0.127*** [0.107, 0.148] (0.010)	0.103*** [0.087, 0.119] (0.008)
Dew	0.343*** [0.259, 0.428] (0.043)	0.068*** [0.052, 0.085] (0.009)	Windspeed	0.052*** [0.041, 0.064] (0.006)	0.016*** [0.013, 0.020] (0.002)
Windgust	0.100*** [0.047, 0.153] (0.027)	0.017*** [0.008, 0.026] (0.005)	-	-	-
R-squared	0.96	-	-	0.33	-
Prob > Chi ²	0.000	-	-	0.000	-
Obs	13,102	13,102	-	17,753	17,753
Cities	441	441	-	386	386
MWALD [†]	0.000	0.000	-	0.000	0.000

	Model 3	Model validation		Model 4	Model validation
Cases _{t-1}	0.986*** [0.986, 0.987] (0.000)	0.977*** [0.976, 0.977] (0.000)	Total tests	1.013*** [1.007, 1.020] (0.004)	1.350*** [1.341, 1.360] (0.005)
CVD	0.002* [0.000, 0.004] (0.001)	0.001* [0.000, 0.002] (0.001)	Stringency index	-0.667*** [-0.689, -0.645] (0.011)	-0.308*** [-0.318, -0.297] (0.005)
Diabetes prevalence	0.039*** [0.036, 0.042] (0.002)	0.010*** [0.009, 0.010] (0.000)	Hospital beds	-0.293*** [-0.306, -0.280] (0.007)	-0.043*** [-0.045, -0.041] (0.001)
Smokers	0.027*** [0.025, 0.030] (0.001)	0.013*** [0.012, 0.014] (0.000)	-	-	-
R-squared	1.00	-	-	0.99	-
Prob > Chi ²	0.000	-	-	0.000	-
Obs	71,466	71,466	-	31,405	31,405
Cities	584	584	-	487	487
MWALD [†]	0.000	0.000	-	0.000	0.000

	Model 5	Model validation		Model 6	Model validation
CVD	0.216*** [0.178, 0.253] (0.019)	0.229*** [0.189, 0.269] (0.020)	Population	0.339*** [0.320, 0.359] (0.010)	0.339*** [0.320, 0.359] (0.010)
Diabetes prevalence	0.247*** [0.230, 0.265] (0.009)	0.415*** [0.386, 0.444] (0.015)	Aged 65 older	1.468*** [1.343, 1.593] (0.064)	1.468*** [1.343, 1.593] (0.064)
Smokers	0.037*** [0.035, 0.039] (0.001)	0.356*** [0.333, 0.379] (0.012)	Income level	-0.449*** [-0.501, -0.396] (0.027)	-0.449*** [-0.501, -0.396] (0.027)
-	-	-	Extreme poverty	0.079*** [0.048, 0.110] (0.016)	0.079*** [0.048, 0.110] (0.016)
R-squared	0.55	-	-	0.57	-
Prob > Chi ²	0.000	-	-	0.000	-
Obs	59,014	59,014	-	43,935	43,935
Cities	594	594	-	445	445
MWALD [†]	0.000	0.000	-	0.000	0.000

Notes: [..., ...] denotes 95% Confidence Interval; (.) is the standard error; ***, * denote statistical significance at 1 and 10% level; CVD—Cardiovascular disease; MWALD—modified Wald test statistic for heteroskedasticity; † rejection of the null hypothesis of homoskedasticity. Estimated from a total of 97,828 observations in 615 cities across the globe. Legend: Model 1 – Cases = $f(\text{Temperature, Pressure, Humidity, Dew, Windgust})$; Model 2 – Cases = $f(\text{PM}_{2.5}, \text{O}_3, \text{NO}_2, \text{Windspeed})$; Model 3 – Cases = $f(\text{Cases}_{t-1}, \text{CVD, Diabetes, Smokers})$; Model 4 – Cases = $f(\text{Tests, Stringency, Beds})$; Model 5 – Deaths = $f(\text{CVD, Diabetes, Smokers})$; and Model 6 – Deaths = $f(\text{Population, Aged, Income, Poverty})$.

Uganda, among others. This perhaps explain why mortality-attributed ambient air pollution is high in developing countries (Owusu and Sarkodie, 2020).

Meteorological factors play a critical role in the spread and containment of COVID-19, hence, can increase or decrease confirmed cases across countries. Our empirical assessment shows that while pressure, dew, Windgust, and windspeed increase the spread of COVID-19, high ambient temperature and relative humidity have mitigation effect on

COVID-19, shrinking the number of confirmed cases. Outdoor environment with high ambient temperature and relative humidity is reported to shorten the lifetime of the virus and eventually degrade and destabilize its environmental conditions for survival (Casanova et al., 2010). Similarly, the risk of COVID-19 transmission is reportedly lower in outdoor environment (Weed and Foad, 2020). It is reported that the survival and lifespan of coronaviruses are active in cold and wet environment compared to dry and warm regions. In the case of

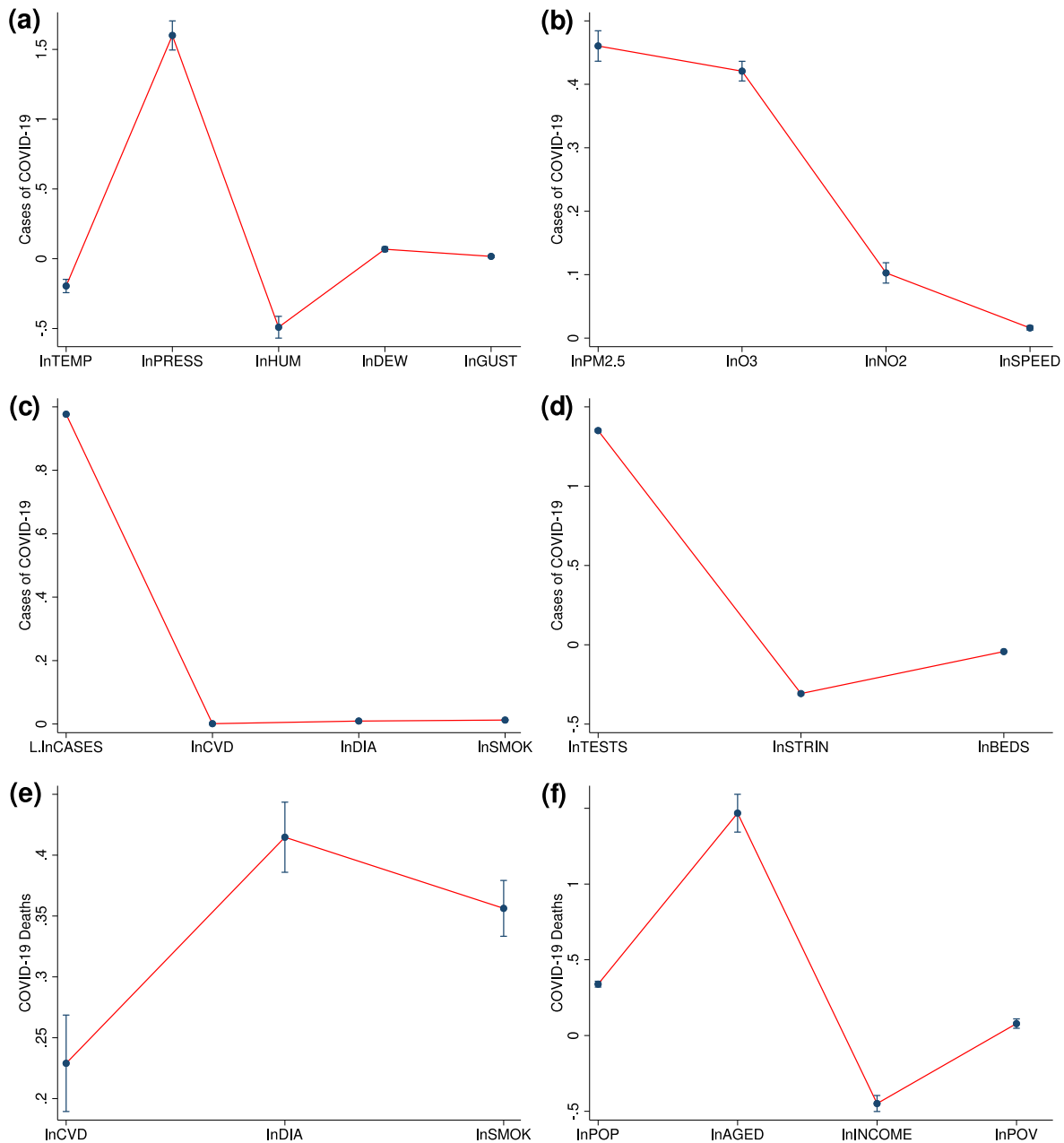


Fig. 8. Estimated models validation (a) Model 1 (b) Model 2 (c) Model 3 (d) Model 4 (e) Model 5 (6) Model 6. Legend: The navy coloured spikes denote 95%, LnCASES—the total number of COVID-19 confirmed cases, InTEMP—Temperature, InPRESS—Pressure, InHUM—Humidity, InDEW—Dew, InGUST—Wind gust, InPM25—Particulate matter 2.5, InO3—Ozone, InNO2—Nitrogen dioxide, InSPEED—Wind speed, InCVD—Cardiovascular diseases, InDIA—Prevalence of diabetes, InSMOK—Male and Female Smokers, InTESTS—Total tests conducted across cities, InSTRIN—Government stringency in containing the spread of the virus, InBEDS—Total number of hospital beds per thousand population, InDEATHS—Total number of COVID-19 deaths reported, InPOP—Population, InAGED—Aged older than 65, InINCOME—GDP per capita—a proxy for income level, and InPOV—Extreme poverty.

windspeed and pressure, its distribution mechanism of suspended droplets of the virus through coughing or sneezing escalates the spread of the contagion by air-borne transmission (Asadi et al., 2020; Contini and Costabile, 2020).

Besides, we find a strong positive relationship between air pollutant species and spread of COVID-19 cases. The escalation effect of ambient PM_{2.5}, nitrogen dioxide and ozone is indirectly linked to its oxidation and pro-inflammatory mechanism of the lungs and systemic alteration dynamics of the human system via immunological activities (Contini and Costabile, 2020). Implying that the prevalence of asthma, chronic obstruction pulmonary diseases, pneumonia and among other respiratory diseases are connected to prolonged exposure to air pollutant species (WHO, 2016). The severity of ambient air pollution is reported to have affected the 2002–4 severe acute respiratory syndrome that

led to over 349 deaths in China (Cui et al., 2003). Other studies have reported mortality-attributed to ambient air pollution due to cardiovascular diseases, and asthma (Lelieveld et al., 2019; Williams et al., 2019). This corroborates our finding of a strong positive relationship between the prevalence of cardiovascular diseases, diabetes, smoking and confirmed cases of COVID-19. Persons with underlying health conditions including cardiovascular diseases, diabetes and daily habits like smoking are more susceptible to COVID-19 due to weak immune system. Risk factor for the outcome of respiratory diseases and underlying health conditions like cardiovascular diseases, diabetes and smoking are more vulnerable to die from COVID-19 (CDC et al., 2020; Guan et al., 2020). This confirms the empirical assessment of our study, which reveals the prevalence of cardiovascular diseases, diabetes and smoking increases COVID-19 attributable deaths.

Our empirical analysis shows that aside underlying health conditions and smoking, population, persons aged 65 years older and extreme poverty may act as confounders in COVID-19 related deaths. However, increasing level of income reduces potential deaths from the virus. In contrast, pollution-driven economic growth is reported to increase the spread of COVID-19 through its effects on air pollution (Magazzino et al., 2021). Besides, the extent of the spread of COVID-19 differs across cities and countries according to socioeconomic and demographic factors (i.e., age structure and population size). More men are reported to die from COVID-19 pandemic compared to women across most countries, however, excess deaths (i.e., all causes of COVID-19 deaths) are relatively equal (Kontis et al., 2020). Population size reduces accessibility to beds and intensive care units when health situations deteriorate. It is even more pronounced among the extremely poor population, especially in developing countries. Health care quality in developed countries cannot be compared to low-income countries, hence, the confounding effects of population size, aged population and income level determine the spread and containment of COVID-19. For example, high mortality rates of COVID-19 among inpatients aged 65 years older are reported compared to young and active adults (Zhou et al., 2020). Thus, older generation with weak immune system and underlying health conditions are more prone to the infection and COVID-19 deaths (Covid and Team, 2020). Similarly, positive correlation between COVID-19 outcomes and racial minorities in the US is reportedly linked to underlying health conditions (Mahajan and Larkins-Pettigrew, 2020). This infers that racial health disparities driven by limited access to healthcare, and poor standards of living may increase the risk of COVID-19 health outcomes. From a public health perspective, our study reveals that while testing capacity boosts the detection of COVID-19 cases, government stringency on containing the spread of COVID-19 and health system improvement such as increasing hospital beds can reduce COVID-19 cases and corresponding deaths.

5. Conclusion

As a contribution to the growing literature on the spread and containment of COVID-19 pandemic, we empirically assessed the overarching effect of air pollutant species, meteorological conditions, underlying health conditions, socio-economic and demographic factors on COVID-19. We utilized a panel estimation of 615 cities in 6 continents from January 1 to June 11, 2020. Our study was limited in terms of coverage, data availability, and unequally-spaced distribution of data, hence, restricting the selection of estimation techniques. Nevertheless, our estimated model controlled for city-city heterogeneous effects, cross-section dependence and omitted variable bias. Our study demonstrated that air pollutant species have detrimental health effects, leading to the spread of COVID-19 and subsequently inducing mortality rates. Meteorological conditions like pressure, dew, windgust, and windspeed were found to escalate the spread of COVID-19, however, high relative humidity and ambient temperature have potential effects in reducing the viability and lifespan of the virus, hence, declining the number of confirmed cases. We further identified confounding factors such as population size, aged population, extreme poverty and income level that act as unobserved common factors affecting either the spread or containment of COVID-19. This implies that several factors determine the survival, transmission and degradation of the virus. From a policy perspective, while climatic factors may be out of control, stringent health policies including timely testing and provision of hospital beds may have a positive long-term impact on containing the spread of the virus. Future research should aim at conducting an experimental assessment of the severity and impact of confounders on coronaviruses.

CRediT authorship contribution statement

Samuel Asumadu Sarkodie: Conceptualization, Formal analysis, Funding acquisition, Methodology, Software, Validation, Visualization,

Writing – review & editing. **Phebe Asantewaa Owusu:** Writing – original draft, Writing – review & editing.

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.

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